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DRUG-RESISTANT INFESTATION DELUSION IN PARKINSON'S DISEASE

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INTRODUCTION

Infestation delusion (ID) is a rare psychiatric condition in which the patient has the conviction of being infested without any medical/microbiological evidence (Davis et al. 2016, Flann et al. 2010, De Berardis et al. 2013, Fowler et al. 2019).

In ID, misinterpretation of normal cutaneous sensetions usually occurs, for which the patient develops delusional interpretations (Davis et al. 2016). ID can be differentiated in primary, arising spontaneously, or secondary to medical or psychiatric disorders (Fowler et al. 2019). Despite the lack of descriptions, the association of psychosis in Parkinson's disease (PD) is not uncommon, as it can arise spontaneously, in elderly patients, in dementia or related with antiparkinsonian medications, especially at high doses (Davis et al. 2016, Flann et al. 2010).

In primary ID, antipsychotics are the baseline of treatment, while in secondary ID, management requires treatment of the underlying condition, and commonly adjunctive use of antipsychotics (Flann et al. 2010). The treatment dilemma in drug-induced ID is that withdrawal of antiparkinsonian medication relieves the psychotic side-effects but worsens the movement disorder, and antipsychotics have the potential to block striatal dopamine receptors. Eliminating other triggering factors should be the first step, and then discontinuation/ reduction of antiparkinsonian medication is required, choosing a drug with a low psychosis-inducing profile and the greatest antiparkinsonian activity. If insufficient or if motor symptoms get worse, an atypical antipsychotic is recommended (Davis et al. 2016, Flann et al. 2010).

Clozapine is the only recommended, however, quetiapine is often tried first, due to better tolerability. Both have the least extrapyramidal side effects (Davis et al. 2016, Kölle et al. 2010).

We intend to acknowledge readers to psychotic symptoms in PD and to emphasize the management strategies in patients refractory to first-line therapy.

CASE REPORT

We report the case of a 63-year-old patient with history of depression and idiopathic PD, 4 years since the diagnosis, and treated with levodopa 300 mg/day. One year after PD diagnosis, and right after the introduction of pramipexole, she developed symptoms of ID. Pramipexole was than suspended and olanzapine 7.5 mg was started. Three months later, due to maintenance of symptoms, a switch to quetiapine was attempted, with marginal improvement and full relapse thereafter. Two years after, with symptoms persistence, she started feeling snake movements in her abdomen, accompanied by anhedonia and clinophilia.

She was admitted to a psychiatric ward. On examination, she had mild bradykinesia, hypomimia, decreesed blinking, predominantly left-sided resting tremor and rigidity. Depressive mood, hypophonic speech, slow thinking, delusional ideas of infestation with mystical ideas; perceptual distortion of normal skin pigmentations; delusional interpretations and somatic/tactile hallucinations, generalized pain and sting with significant emotional impact, and passive thoughts of death and partial insight. Laboratory evaluation and brain computed tomography showed no abnormalities.

Firstly, quetiapine was titrated up to 650 mg id and due to reported cases of success, therapy with lorazepam was also associated. Rivastigmine was introduced as well and titrated to 13.3 mg/24h. Due to treatment resistance, a switch was conducted from quetiapine to 300 mg/day clozapine. Without any symptom remission, levodopa reduction was tried, unsuccessfully, and with worsening motor function. Given the pharmacological resistance, she started Electroconvulsive Therapy (ECT). After 9 treatments, there was almost complete remission of psychotic and affective symptoms. There were improvements at the motor level and the patient herself recognized a return to premorbid functioning. She maintained an outpatient follow-up schedule of ECT and the remaining therapeutics already in place.

DISCUSSION

The pathophysiology of ID is unknown. A decreesed striatal dopamine transporter functioning leading to increased extracellular levels and hyperactivity of dopamine systems, may be its neurobiological background. It has been reported arising during treatment with dopamine agonists or substances influencing

dopamine transporter. In situations of dopamine dysregulation like PD, normal stimuli may be perceived with aberrant intensity.

Reported cases of ID in PD, occurred secondarily to pergolide, pramipexole, trihexyphenidyl, armodafinil and introduction/titration of ropinirole (Flann et al. 2010). In most cases, complete resolution is obtained with discontinuation of such drug and dose reduction is ineffective (Flann et al. 2010). For persistent ID, treatment include benzodiazepine trial (Davis et al. 2016) and even cholinesterase inhibitors, that have mild effect on psychosis features of PD (Zahodne & Fernandez 2008, Panchal & Ondo 2018).

In this case, the onset of psychotic symptoms occurred during the clinical phase of PD, after exposure to pramipexole. The patient presented with delusional ideas of infestation, delusional interpretations and tactile-somatic hallucinations, with significant emotional impact. After pramipexole withdrawal, there was maintenance and posterior worsening of symptoms. She was diagnosed with ID in the context of an organic psychosis related to PD, and a secondary major depressive disorder relapse.

Given the pharmacological resistance and symptom severity the patient started treatment with ECT. As far as we know ECT has never been described in patients with ID associated with PD, however good results were achieved in each of these conditions alone.

In PD psychosis treated with ECT, patients showed improvement in motor and psychotic symptoms without important adverse effects (Zahodne & Fernandez 2008, Calderón-Fajardo et al. 2015, Nishioka et al. 2014), such as our patient.

CONCLUSION

Treatment of ID in the context of PD usually includes the elimination of triggering factors, discontinuation/reduction of antiparkinsonian medication and the use of atypical antipsychotics. With this work, we intend to reinforce the idea that in refractory cases of ID in the context of PD, ECT seems to be an effective option, with no major adverse effects, and with good results in association with clozapine (Factor et al. 1995).

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Rita Diniz Gomes: literature searches and analyses; conception of the project; manuscript writing of the first draft and review.

Elisa Silva: literature searches and analyses; manuscript writing of the first draft and review.

Cátia Fernandes Santos: literature searches and analyses, manuscript review and critique.

Filipa Senos Moutinho: literature searches and analyses, conception of the project, manuscript review and critique.

References

1. *Calderón-Fajardo H, Cervantes-Arriaga A, Llorens-Arenas R, Ramírez-Bermudez J, Ruiz-Chow Á, Rodríguez-Violante M: Electroconvulsive therapy in Parkinson's disease. Arq Neuropsiquiatr 2015; 73:856-60*
2. *Davis J, Kurek J, Sethi K, Morgan J: Delusional Infestation in Parkinson's Disease. Mov Disord Clin Pract 2016; 4:111-115*
3. *De Berardis D, Serroni N, Marini S, Rapini G, Valchera A, Fornaro M et al.: Successful ziprasidone monotherapy in a case of delusional parasitosis: a one-year followup. Case Rep Psychiatry 2013; 2013. doi:10.1155/2013/913248*
4. *Factor SA, Molho ES, Brown DL: Combined clozapine and electroconvulsive therapy for the treatment of drug-induced psychosis in Parkinson's disease. J Neuropsychiatry Clin Neurosci 1995; 7:304-7*
5. *Flann S, Shotbolt J, Kessel B, Vekaria D, Taylor R, Bewley A et al.: Three cases of delusional parasitosis caused by dopamine agonists. Clin Exp Dermatol 2010; 35:740-742*
6. *Fowler E, Maderal A, Yosipovitch G.: Treatment-induced Delusions of Infestation Associated with Increased Brain Dopamine Levels. Acta Derm Venereol 2019; 99:327-328*
7. *Kölle M, Lepping P, Kassubek J, Schönfeldt-Lecuona C, Freudenmann RW: Delusional infestation induced by piribedil add-on in Parkinson's disease. Pharmacopsychiatry 2010; 43:240-242*
8. *Nishioka K, Tanaka R, Shimura H, Hirano K, Hatano T, Miyakawa K et al.: Quantitative evaluation of electroconvulsive therapy for Parkinson's disease with refractory psychiatric symptoms. J Neural Transm 2014; 121:1405-10*
9. *Panchal SC & Ondo WG: Treating Hallucinations and Delusions Associated With Parkinson's Disease Psychosis. Curr Psychiatry Rep 2018; 27. doi:10.1007/s11920-018-0869-z*
10. *Zahodne LB & Fernandez HH: Pathophysiology and treatment of psychosis in Parkinson's disease: a review. Drugs Aging 2008; 25:665-82*

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