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A Case of Parkinson plus Syndrome Associated with Paranoid Schizophrenia

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Abstract

Background: Schizophrenia is a serious psychiatric illness, whose antipsychotic therapy used in their treatment may induce the Parkinson disease. In this case, after discarding the iatrogenic etiology, it was considered an association of paranoid schizophrenia with symptoms and signs of Parkinsonism, identified as Parkinson plus or atypical Parkinson syndrome, being impossible to differentiate between a MSA or PSP, based on scintigraphy with IBZM.

Case Report: This case is about a 70 years old man with paranoid schizophrenia diagnosed when he was 48 years and treated with antipsychotic drugs, antidepressants and benzodiazepines. When he was 68 years old, emerged the first symptoms associated with the Parkinson's disease and were made several therapeutic adjustments that resulted unfruitful. Neurological examinations were realized to confirm Parkinson´s disease diagnosis (positron emission tomography with computed tomography, polysomnography and magnetic resonance) and made new therapeutic adjustments to both pathologies.

Conclusion: Parkinson plus syndrome, is an unusual case, where are affected several systems and whose therapeutic approach requires a meticulous control of physiological and behavioral functions**.** The association of psychiatric and neurological pathologies translates into an individualized approach and highly strict with respect to the drugs association.

Keywords

schizophrenia; antipsychotics; paranoid schizophrenia; Parkinson plus; multi-systemic atrophy

Introduction

Schizophrenia is a severe mental illness, characterized by positive and negative symptoms and cognitive deficits that affect almost all aspects of metal activity, including the perceiving, attention, memory and emotion [1]. Epidemiologically, the incidence and prevalence of schizophrenia are highly variable, affecting 1 % of the world population [4]. This variability is related to the contribution of genetic and environmental factors to the risk of the disease [2,3]. More specifically, the diagnosis and classification of mental illnesses such as schizophrenia, is mediated by diagnostic manuals like DSM-5 or ICD-10, with concise and explicit criteria [5]. In the case of paranoid schizophrenia, the symptoms include delusions of persecution and frequent auditory hallucinations, as well as mistrust continues, anxiety, anger, tendency to quarrel with little or no impairment of cognitive functions [6,7].

Since 1950, ten years after introduction of first generation antipsychotics, that the therapeutic

approach of schizophrenia has suffered valuable pharmacological advances [8,9].

Case Report

It is a case about a 70 years old Caucasian man, with clinical history of paranoid schizophrenia, diagnosed for over 20 years. He is referenced for follow-up in neurology for suspicion of Parkinson's disease. The patient is subjected to neurological examinations in order to differentiate what type of atypical Parkinsonism syndrome he had (drug-induced Parkinsonism, multiple system atrophy – MSA or progressive supranuclear palsy – PSP). During the twenty years of schizophrenia treatment, the patient was medicated with some antipsychotic drugs of first and second generation. Was stabilized with the following drugs: risperidone 1mg once/day, amitriptyline + perphenazine 25mg + 2 mg twice/day, flupentixol 100 mg monthly injectable and zolpidem 10 mg at bedtime.

At the end of 20 years with the abovementioned medication (68 years), the patient shows lack of facial expression, tremor especially in his right arm, muscle rigidity, unstable gait, orthostatic hypotension, frequent falls and urinary sphincter dysfunction. In the diagram 1 is made a sequence of pharmacological interventions as well as of the symptoms that arise in the various stages of treatment. In the first neurological approach, proceeded to clinical confirmation of Parkinson's disease, in which has been withdrawn the amitriptyline+perfenazina, being replaced by sertraline 50 mg once/day and incorporated carbidopa+levodopa 25mg+100mg with slow start to up a daily dose of four tablets.

The magnetic resonance indicated a mild cerebral atrophy of the frontal lobes. After a period of one month, treatment with carbidopa and levodopa reaches the daily dose of 4 tablets, but the patient does not respond adequately to treatment, appearing orthostatic hypotension and falls. Again reassessed neurologically, the patient begins to take carbidopa + levodopa (25mg+100mg) three times a day and is introduced selegiline 5 mg twice a day. The patient didn´t tolerate medication, suffering behavioral disorders as aggressiveness and suicide attempt. After this incidence, the patient was subjected to a battery of tests such as: positron emission tomography with computed tomography (PET-CT), polysomnography, assessment of nervous functions (upper battery of Lisbon for the evaluation of dementia, Garcia et al.1984) and clinical analysis.It was introduced the ropinirol slowly until to reach the 8 mg daily, reducing the selegiline for a daily dose. The patient becomes highly aggressive, makes more two suicide attempts, suffers from persecution delusions and hallucinations. The hypotension was out of control, as well as falls, being hospitalized for 15 days, where were done several clinical analyses, a screening for heart disease and hypovolemic shock, having all normal clinical parameters, suggesting that hypotension is result of Parkinson's medication.

The results of PET-CT(figure 1) indicates that the availability of postsynaptic dopamine D2 is decreased throughout the striatum, suggesting Parkinson plus syndrome, not being possible to differentiate between PSP and MSA based on scintigraphy IBZM. The polysomnography reveals that the patient suffers from sleep apnea and the neuropsychological assessment shows little changes in higher nervous function. Analytically, the patient has all the normal parameters.

Currently, the patient takes sertraline 50 mg once a day, carbidopa+levodopa (25mg+100mg) three times a day, selegiline 5mg twice a day, ropinirol 2mg once a day (having regard a slow reduction since the 8 mg up to 2 mg), risperidone 1 mg once a day, zolpidem 10 mg at bedtime and the flupentixol

100 mg monthly injectable was replaced by psychiatrist for paliperidone 75mg monthly injectable. After the confirmatory diagnosis of Parkinson plus syndrome and a new therapeutic adjustment, the patient stopped to have aggressive behavior and suicidal thoughts, he was able to redo tasks which previously was unable to do alone such as bathing, dressing, eating, walking, doing exercise, etc. The rigidity has improved, as well as the facial expressions. The orthostatic hypotension remitted, the dysfunction of the urinary sphincter disappeared and the movements, despite the slowness are accurate, performing various daily tasks with consistency.

Discussion

Antipsychotic drugs have a wide range of side effects, the scope of which includes severe and unavoidable effects. The Parkinson plus syndrome [11] is a real “nightmare" for the health professionals and family members. The conjugation of antipsychotic and anti-Parkinson drugs requires a thorough evaluation of the patient clinical history as well as a close monitoring of the caregiver to provide all behavioral and physiological records for the best therapeutic approaches. The need for new generations of antipsychotic and antiparkinsonian drugs with less side effects it becomes imperative.

Diagram

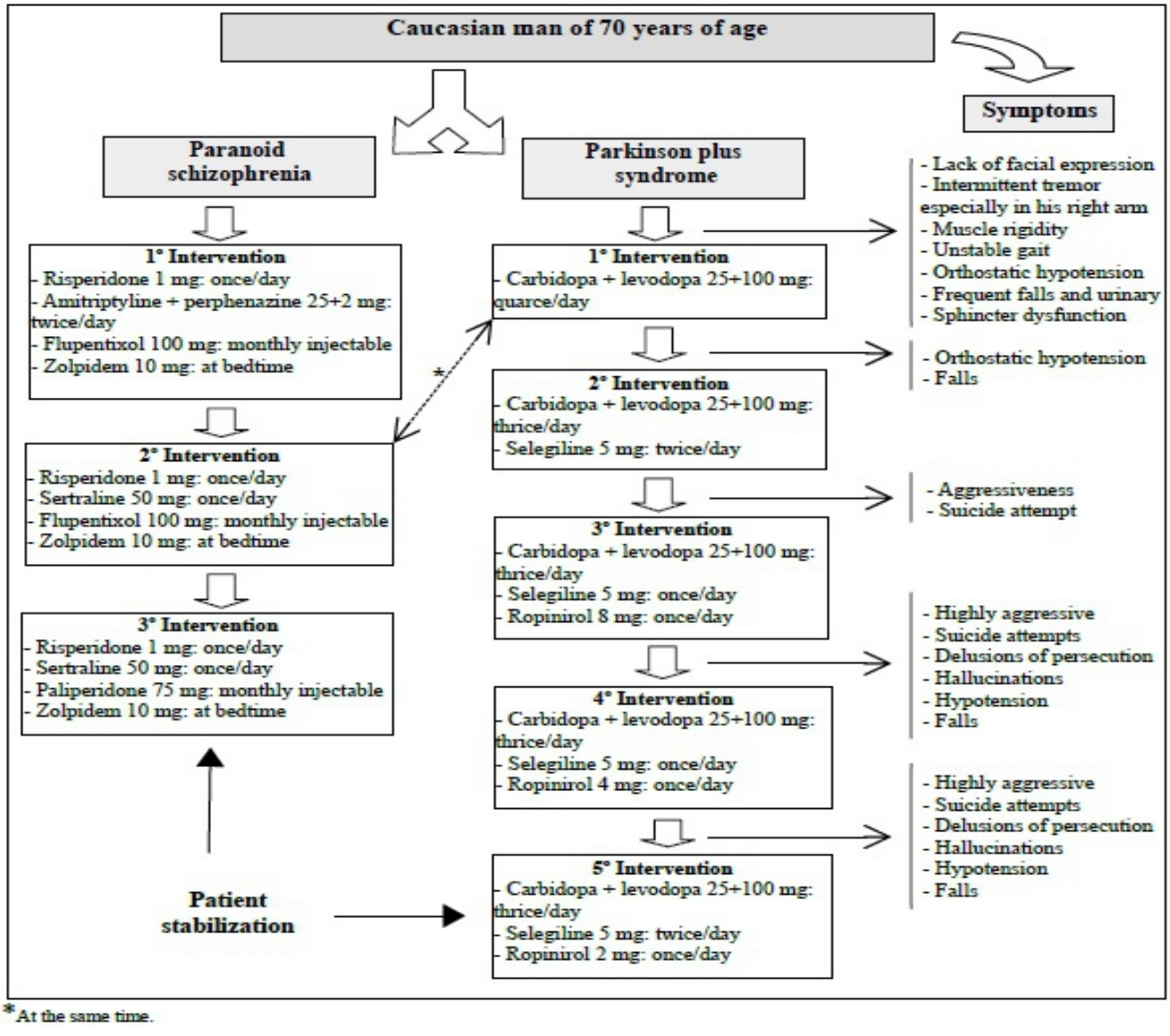

Diagram 1: schematic sequence of the patient's history

Conclusion

As described, one of the possible side effects of antipsychotic drugs is the induction of Parkinson's disease. Initially, this hypothesis was supported due to long term use of neuroleptic drugs, as well as poor response that the patient presented to the Parkinson's disease treatment. An approach focused solely on the typical Parkinson's disease, this responds effectively to anti-Parkinson therapy [10,12]. But in atypical cases such as this, the stable schizophrenic patient develops a framework of atypical Parkinsonism, whose response to anti-Parkinson drugs is very limited and the therapeutic interactions are reflected in a complex world of disturbances in both pathologies, with a minimum threshold of stability in pharmacological adjustment. Due to this difficulty, is inevitable a constant updating, a therapeutic individually adjusted as well as the investigation of new therapeutic approaches that reduce the impact developed by current pharmacological options. More neurological exams are being made in order to justifying the Parkinson plus syndrome and the differentiation between MSA or PSP, the deficient activity of the caudate nucleus and the putamen on both hemispheres, as well as increasing the background activity revealed in the study of dopaminergic function IBZM and SPECT.

Figure

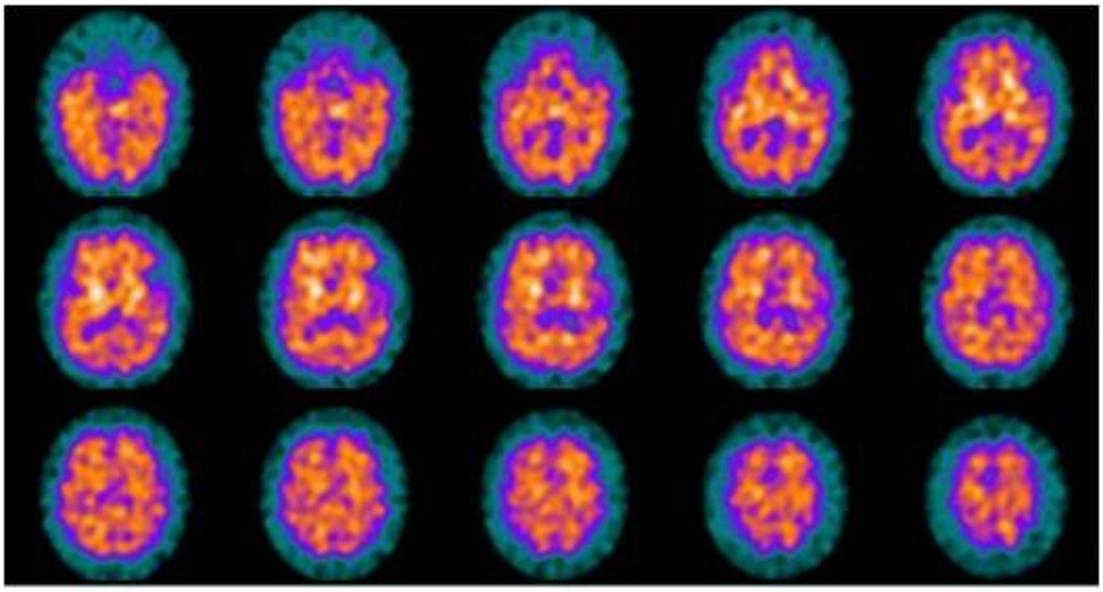

Figure 1: PET-CT images: D2 receptor occupation in the striatum by antipsychotics using ^{123}I IBZM scans and SPET.

PET images at the level of the striatum revealed reduction in striatal dopamine D2 receptor. The qualitative analysis demonstrates decreased uptake of the radiopharmaceutical in basal ganglia, with poor definition of the caudate nucleus and the putamen of both hemispheres. Additionally, there is an increase of background activity.

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