Rapid Progression of Parkinsonian Symptoms in Lewy-Body Dementia after Administration of Antipsychotics: Case Report

Ali Mahmood Khan1*, Shanila Shagufta1, Sukaina Rizvi1, Shajeda Borhan1, Auria Meher Chowdhury1 and Abdul Mohit2

1Kings County Hospital Center, USA
2Department of Behavioral Health, Kings County Hospital Center, USA

Submission: August 22, 2017; Published: September 06, 2017

*Corresponding author: Ali Mahmood Khan, Kings County Hospital Center, New York, USA, Email: ali.mahmood_khan@hotmail.com

Abstract

This case is of a 60 year old female patient who lives in an apartment and has no prior psychiatric history. Recently the patient had been getting more and more agitated, paranoid, increasingly withdrawn and did not maintain her ADLs. Patient mentioned being tired, not being able to sleep as people living upstairs play loud music. Upon investigation, there was no loud music being played by the patient’s neighbors at night or at any time. Patient also complained of the people living upstairs did not like her and disturbs her which in fact was not true. Patient lives alone in the apartment and used to be active and all household chores were performed by the patient herself but recently patient has been really lazy and keeps lying on the couch and does not perform her chores. Patient has also started threatening people including her daughter. Patient worked in the city before and has been married twice but both resulting in separations and divorce. As per daughter, patient is easily forgetful, repeats things again and again. Patient denies using any drugs or illicit substances and has no significant medical problem. However according to the daughter patient has a past history of IV heroin abuse (20 years ago) and alcohol abuse.

Introduction

The research is being conducted to find out that if treating lewy body dementia with anti-psychotics rapidly increases Parkinsonian symptoms. According previous researches, anti-psychotics have shown to cause drug induced Parkinsonism. This research is based upon whether or not there is any relationship in between the two.

Case Presentation

This is a case of a 60y/o Female, domiciled in an apartment, unemployed, no prior psychiatric history, brought in by EMS activated by daughter because patient was increasingly withdrawn, getting more and more agitated, paranoid and not maintaining her ADLs. On questioning, patient mentions that she feels tired, did not sleep for many days and does not want to give enough information but reported that, “lady upstairs in my apartment is against me, does not like me, playing loud music in the middle of the night and disturbed me, and I can’t sleep.”

As per daughter, patient was living alone in an apartment, doing everything on her own, but since the past few months patient is gradually becoming more and more isolated and withdrawn, not taking care of herself. As per daughter, her mother was always active, cooked on her own; always cleaned the apartment, good hygiene, always did her nail, but since the past few months patient is not cooking, the apartment is a mess, not clean at all, they found no food in the refrigerator, and losing weight. As per other family members, the patient is also increasingly paranoid, irritable and thinks that lady upstairs is against her and disturbs her by playing loud music. Patient’s daughter spoke with the lady upstairs on her mom’s persistence, who told the patient’s daughter that she is not playing any loud music and in fact goes to work and sleeps early. Patient additionally has been threatening people including her daughter lately. As per daughter; patient has become more forgetful, with perseveration. According to her daughter, patient had past history of IV heroin abuse (20 years ago) and alcohol abuse,
patient denied recent substance abuse and on testing urine toxicology was negative. No recent history of trauma.

The patient was admitted to treat the symptoms of paranoid delusions. Primary pathology at the time of admission most likely was Major depressive disorder with psychotic features with possible dementia as patient reported anhedonia, poor concentration, reduced sleep, and poor attention along with her paranoid delusions. She also has recent stressors including recent divorce, unemployment, and poor family support.

On testing performed CT scan showed frontal lobe atrophy, ventricular dilatation, and micro-vascular ischemia. Labs: TSH, B12, CMP, LFTS were all normal and within limits. Patient’s strengths were her previous employment history, no family psychiatry history, no past psychiatric history and higher education. Risks included her poor insight into her disease, unemployment, heroin/alcohol abuse history, delusions, and paranoid behavior.

Patient worked in the city for many years. Patient has married twice, last marriage seven years ago and they were separated in April, 2012 and after few months eventually divorced. Since then patient has been single. No significant medical problem.

Patient was started on low dose typical antipsychotic, Haldol 0.5mg per-oral q12h, with gradually increasing the dose to reduce psychosis. As the patient was started on the medication, her delusions significantly improved, on the contrary, patient developed severe parkinsonian symptoms such as akinesia, bradykinesia and impaired swallowing as the dose of Haldol was increased up till 2mg BID. This episode significantly increased the suspicion of Lewy Body Dementia. This suspicion with CT scan changes made the suspicion more concrete. On CT scan there was severe dilatation of the ventricles and sulci, particularly in frontal lobes, consistent with atrophy. Additionally there were mild peri-ventricular white matter hypo-densities with preservation of the distinction between grey and white matter. After witnessing the changes seen on CT scan, neurology was consulted, who performed the MOCA, on which the patient scored 16/30. They recommended discontinuation of Haldol and initiation of Seroquel 25mg QHS. After initiation of Seroquel, the patient improved considerably, as his parkinsonian symptoms resolved, and his swallowing greatly improved. Over the course of stay in the hospital, patient’s Seroquel was increased up till 125mg QHS. Patient was discharged from the hospital on this dose, clinically stable.

Discussion

Lewy body dementia is a type of progressive brain disorder in which lewy bodies (protein alpha synuclein) build up in areas of the brain that regulate behavior, cognition and movement. This condition impairs functions such as memory retention, thinking, executive functions and the ability to understand visual information [1]. Patients may have fluctuations in attention or alertness, problems with movement including tremors, stiffness, difficulty walking, hallucinations and alterations in sleep and behavior. It is also said to be probably the second most common cause of degenerative dementia in older people, only Alzheimer’s disease is more common [2].

Parkinsonism is a general term used to describe neurological or brain disorders that cause symptoms similar to those seen in Parkinson’s disease [3]. These symptoms include an ongoing loss of motor control system which causes resting tremors, stiffness, postural instability and slow movement [4,5]. It also causes a wide range of non motor symptoms which include depression, loss of olfactory function, and cognitive changes. The symptoms mentioned are also similar to those observed in patients suffering from lewy body dementia therefore it is difficult to distinguish lewy body dementia from Parkinson’s disease [6].

Anti psychotics are drugs used to treat symptoms of psychosis such as delusions, hallucinations, paranoia or confused thoughts. It is used in diseases such as schizophrenia, severe depression and severe anxiety [7]. These drugs are also useful at stabilizing episodes of mania in patients suffering from bipolar disorder. The main action of antipsychotic drugs is that they act on dopamine receptors reducing the levels of excessive dopamine. They may also affect levels of other neurotransmitters namely acetylcholine, nor-adrenaline and serotonin [8]. Older antipsychotics are called typical or first generation antipsychotics. First generation antipsychotics are now rarely used and are only used when second generation antipsychotics are not effective. Atypical antipsychotics are less likely to produce drug induced disorders such as Parkinsonian symptoms, restlessness, rigidity, tardive dyskinesia, tremors and other unwanted movements [5]. These are more effective at treating the negative symptoms of schizophrenia such as lack of motivation and social withdrawal and are also more effective in treatment resistant patients. Clozapine was the first atypical antipsychotic to be approved by the US food and drug administration [3]. All antipsychotics are related to adverse effects which include increased risk of sedation, sexual dysfunction, postural hypotension, cardiac arrhythmias and sudden cardiac death.

According to researches, Parkinsonian symptoms are worsened or aggravated when treated with antipsychotics. The extra pyramidal symptoms caused by antipsychotics include Parkinsonism (pseudo Parkinsonism), akathisia, acute dystonia and tardive dyskinesia [9]. The Parkinsonian symptoms caused by antipsychotics are reversible which can be treated by reducing the dosage or adding oral anti cholinergic agents but keeping in mind that these drugs can cause their own side effects. The Parkinsonian symptoms caused by antipsychotics include quivering of the hands and arms as well as rigidity in the shoulders and arms. It also causes bradykinesia, akinesia,
hyper salivation and small shuffling gait. The cause of drug induced Parkinsonian symptoms is related to drug induced changes in the basal ganglia which occur after the inhibition of the dopaminergic receptors by the antipsychotic drugs [10].

Conclusion

After research it can be stated that there is some relation between Parkinsonian symptoms in lewy body dementia and antipsychotics. Patients taking antipsychotic drugs for Parkinsonian symptoms in lewy body dementia were found to have even worse symptoms than patients who were not using antipsychotics. Therefore it can be stated that the symptoms are aggravated and there is also rapid progression of Parkinsonian symptoms in lewy body dementia after the administration of antipsychotics.

Reference

1. Shin HW, Chung SJ (2012) Drug-Induced Parkinsonism. J Clin Neurol 8(1): 15-21.
2. McKeith I (2004) Dementia with lewy bodies. Dialogues Clin Neurosci 6(3): 333-341.
3. Frank C (2003) Dementia with Lewy bodies. Review of diagnosis and pharmacologic management. Can Fam Physician 49: 1304-1311.
4. Gomperts SN (2016) Lewy Body Dementias: Dementia With Lewy Bodies and Parkinson Disease Dementia. Continuum (Minneap Minn): 435-463.
5. López SJL, Mena MA, de Yébenes JG (2012) Drug-induced parkinsonism in the elderly: incidence, management and prevention. Drugs Agin 29(2): 105-118.
6. Ballard C, Corbett A, Howard R (2014) Prescription of antipsychotics in people with dementia. The British Journal of Psychiatry 205(1): 4-5.
7. McKeith IG, Galasko D, Wilcock GK, Byrne EJ (1995) Lewy body dementia-diagnosis and treatment. The British Journal of Psychiatry 167(6): 709-717.
8. John PT, Michael JF, Jiaobao H, Nicola B, Sarah P, et al. (2012) Visual cortex in dementia with Lewy bodies: magnetic resonance imaging study. Br J Psychiatry 200(6): 491-498.
9. Rianne MVDL, Tom D, Blossom CMS, Matthew PA, Elizabeth E, et al. (2016) Longitudinal course of behavioral and psychological symptoms of dementia: systematic review. The British Journal of Psychiatry 209(5): 366-377.
10. Livingston G, Kelly L, Lewis-HE, Baio G, Morris S, et al. (2014) Non-pharmacological interventions for agitation in dementia: systematic review of randomized controlled trials. Br J Psychiatry 205(6): 436-442.