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Case Study

A Case Report on Risperidone Induced Parkinsonism

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Drug induced Parkinsonism is a common and potentially reversible adverse effect associated with dopamine receptor blocking agents, particularly antipsychotics. Elderly patients are at higher risk due to age related neurodegenerative changes and polypharmacy. This report presents a 63 year old female with a history of type 2 diabetes Mellitus, systemic hypertension, hypothyroidism and psychiatric illness, who developed Parkinsonism features during treatment with risperidone. She presented with fatigue, reduced oral intake and progressive difficulty in walking. Neurological evaluation and imaging studies supported the diagnosis of drug induced parkinsonism. MRI brain revealed generalized neuroparenchymal atrophy with hypertensive microangiopathic changes and small vessel ischemic disease. MRI spine showed multilevel degenerative disc changes. A multidisciplinary approach was adopted in the management of the patient including the modification of Psychiatric medications, discontinuation of the potential contributing agents and the initiation of supportive measures. The patient showed improvement following these interventions. The case highlights the importance of early identification of extrapyramidal symptoms and careful use of antipsychotics in elderly patients with multiple risk factors.

Keywords: Parkinsonism, Antipsychotics, Neuroparenchymal, Microangiopathic.

INTRODUCTION

Drug induced parkinsonism is one of the most frequently encountered forms of secondary parkinsonism. It arises primarily due to blockade of dopamine D2 receptors in the nigrostriatal pathway. Antipsychotic medications remain the leading cause with both typical and atypical agents implicated. Although atypical antipsychotics are considered safer, certain agents such as risperidone retain a significant potential to induce extrapyramidal side effects. The clinical presentation of drug induced parkinsonism closely resembles idiopathic parkinsons disease making diagnosis challenging. However unlike primary parkinsons disease, DIP is often reversible if recognized early and managed appropriately. Among the various classes of medications associated with this condition antipsychotic drugs are the most commonly implicated. These agents exert their therapeutic effects largely through antagonism of dopamine D2 receptors in the brain. However, this same mechanism is responsible for the development of extrapyramidal side effects. Second generation or atypical antipsychotics were developed with the intentions of reducing such adverse effects by incorporating serotonergic activity along with dopaminergic modulation. Despite this certain atypical agent particularly risperidone continue to demonstrate a relatively higher propensity to induce extrapyramidal symptoms compared to others in the same class. This attributed to its strong binding affinity for D2 receptors especially at moderate to high doses which can significantly reduce dopaminergic signaling in motor control pathways. In addition to antipsychotics other psychotropic medications can also influence the risk of developing parkinsonian symptoms. Lithium a

widely used mood stabilizer in the management of bipolar and other effective disorders has been associated with a range of neurological adverse effects. Although lithium does not directly block dopamine receptors in the same manner as antipsychotics, it alters intracellular signaling pathways and neurotransmitter balance including dopaminergic transmission. Prolonged use or elevated serum levels of lithium may lead to neurotoxic effects manifesting as tremors, muscle rigidity and impaired coordination. When used concurrently with antipsychotic medications, lithium may enhance susceptibility to extrapyramidal symptoms by potentiating neurochemical imbalances or reducing the brains ability to compensate for dopaminergic blockade. The combined use of risperidone and lithium is not uncommon in clinical practice particularly in patients with complex psychiatric conditions requiring both mood stabilization and antipsychotic therapy. However such combinations necessitate careful monitoring due to the potential for additive or synergistic adverse effects on the central nervous system. The interactions between these agents may not always be purely pharmacokinetic but can involve pharmacodynamic mechanisms that increase the likelihood of neurological complications including drug induced parkinsonism. Elderly individuals are particularly susceptible due to physiological decline in dopaminergic neurons, presence of comorbidities and concurrent use of multiple medications. Age related decline in dopaminergic neuron density altered drug metabolism and increased sensitivity to central to central nervous system active medications contribute to heightened risk of adverse effects. Furthermore, the presence of comorbid conditions such as hypertension, diabetes Mellitus and structural brain changes like microangiopathy or cerebral atrophy can further compromise neuronal function. Polypharmacy which is common in this age group adds another layer of complexity by increasing the potential for drug interactions and cumulative toxicity. Another important clinical challenge lies in differentiating drug induced parkinsonism from idiopathic parkinsons disease. The overlap in symptomatology often complicates diagnosis particularly in older individuals where both conditions may coexist or mimic each other. However, a key distinguishing feature of drug induced parkinsonism is its potential reversibility following dose reduction or discontinuation of the offending agent. Early identification is therefore critical to prevent unnecessary longterm disability and to optimize patient outcomes. This case report illustrates the development of parkinsonian symptoms in an elderly female patient receiving risperidone in combination with lithium and other medications. It highlights the need for vigilant monitoring, judicious use of psychotropic drugs and timely intervention to mitigate adverse neurological effects especially in high risk patient population.

CASE REPORT

The reports investigate a case of Risperidone Induced Parkinsonism. A 63-year-old female was admitted with complaints of generalized fatigue and reduced food intake. The symptoms had gradually intensified over time, significantly affecting her ability to perform daily activities independently. The patient had a known medical history of multiple chronic conditions including type 2 diabetes Mellitus, systemic hypertension, hypothyroidism and long-standing psychiatric illness. She was on multiple medications for the management of these conditions including oral hypoglycemic agents, antihypertensive drugs, thyroid hormone replacement therapy and psychotropic medications. Her medication regimen included metformin, glimepiride, a combination of vildagliptin and metformin, dapagliflozin, telmisartan, chlorthalidone, levothyroxine, lithium and risperidone. On clinical examination her vital sign were found to be stable. However, she appears visibly fatigued and exhibited reduced mobility. Notably she had difficulty in walking which raised concerns regarding a possible neurological impairment. Initial management included administration of intravenous fluids, empirical antibiotic therapy, continuation of antihypertensive medications and supportive care measures. Laboratory investigations revealed certain abnormalities including a reduced lymphocyte count, hyponatremia and an elevated erythrocyte sedimentation rate indicating an underlying inflammatory or systemic condition. Radiological evaluation was performed to further investigate her neurological symptoms. Magnetic resonance imaging of the brain demonstrated generalized neuroparenchymal atrophy along with features

suggestive of hypertensive microangiopathy and chronic small vessel ischemic changes. Imaging of the spine revealed multilevel degenerative disc disease including disc desiccation, posterior and anterior disc bulges and structural changes in the cervical, dorsal and Lumbosacral regions. A detailed neurological assessment suggested that presence of parkinsonian features. Based on these findings further evaluation and interdisciplinary consultations were undertaken. Psychiatric review lead to the discontinuation of risperidone and lithium and initiation of lorazepam for symptomatic management. Risperidone was identified as the most probable causative agent for extrapyramidal symptoms observed in the patient. Subsequently appropriate modifications were made to her medication regimen including withdrawal or adjustment of the suspected offending agent. The patient managed with supportive care and aimed at improving mobility and functional status. Gradual clinical improvement was observed following these interventions supporting the diagnosis of drug induced parkinsonism.

DISCUSSION

Drug induced parkinsonism occurs due to dopamine receptor blockade in the basal ganglia. Risperidone although classified as an atypical antipsychotic exhibit strong D2 receptor antagonism, which contributes to its potential to cause extrapyramidal symptoms. The increase in patient susceptibility is due to certain factors such as advanced age, female gender, multiple comorbidities, polypharmacy and chronic exposure to antipsychotic medication. Additionally, underlying brain changes such as microangiopathy and atrophy may reduce the brains compensatory capacity making it more vulnerable to dopamine blockade. According to shin et al, DIP is among the most common cause of secondary parkinsonism and is primarily mediated through dopamine D2 receptor blockade in the nigrostriatal pathway. This mechanism explains the development of motor symptoms such as bradykinesia, rigidity and gait disturbances as observed in the present case. Although second generation antipsychotics are generally associated with a lower incidence of extrapyramidal side effects, Leucht et al have reported that certain agents including risperidone demonstrate a comparatively higher risk among atypical antipsychotics. This is attributed to its relatively strong affinity for dopamine D2 receptors at therapeutic doses. Age is significant determinant in the development of DIP. Jeste et al emphasized that elderly patients are particularly vulnerable due to age related degeneration of dopaminergic neurons and reduced physiological reserve. Polypharmacy is another important risk factor. Haddad et al noted that concurrent use of multiple psychotropic medications increases the likelihood of adverse neurological effects. In the present case the patient was receiving lithium along with risperidone. Gitlin et al reported that lithium influences neuronal signaling and may exacerbate neurotoxicity when used in combination with antipsychotics. The discontinuation of lithium in this patient reflects evidence based clinical practices. Laboratory abnormalities also plays a supportive role in understanding the patients condition. Adrogue et al reported that hyponatremia can lead to neurological manifestations such as weakness, confusion and gait instability. Management of DIP primarily involves withdrawal or dose reduction of the offending drug. Tarsy et al emphasized that early identification and discontinuation of causative agents often result in symptom improvement.

CONCLUSION

This case report highlights the development of drug induced parkinsonism in an elderly patient receiving risperidone therapy in the presence of multiple comorbid conditions and concurrent medications. The coexistence of underlying cerebral atrophy and microvascular changes along with degenerative spinal abnormalities contributed to the complexity of clinical presentation and may have increased the patient vulnerability to neurological adverse effects. The case underscores the critical Importance of early recognition of extra pyramidal symptoms in patients receiving antipsychotic medications. Prompt identification and timely modification of the treatment regimen played a key role in the effective management of the condition and in preventing further functional deterioration. It also demonstrates drug induced parkinsonism is potentially reversible when appropriate measures are taken at early satge. Healthcare professionals must exercise heightened caution while prescribing antipsychotics particularly in elderly patient who are at increased risk due to

physiological changes, comorbidities and polypharmacy. Regular monitoring, individualized treatment planing and patient specific risk assessment are essential to minimize adverse effects and optimize therapeutic outcomes. Ultimately a multidisciplinary approach is crucial in ensuring safe and effective patient care in such complex clinical scenarios.

REFERENCES

1. Shin HW, Chung SJ. Drug-induced parkinsonism. J Clin Neurol. 2012;8(1):15–21.
2. Fahn S, Jankovic J, Hallett M. Principles and practice of movement disorders. 2nd ed. Philadelphia: Elsevier; 2011.
3. Tarsy D, Baldessarini RJ. Movement disorders induced by antipsychotic drugs. N Engl J Med. 2006;354(12):1243–52.
4. Leucht S, Cipriani A, Spineli L, et al. Comparative efficacy and tolerability of antipsychotics. Lancet. 2013;382(9896):951–62.
5. Jeste DV, Blazer D, Casey D, et al. ACNP White Paper: Update on geriatric psychopharmacology. Neuropsychopharmacology. 2008;33(5):957–70.
6. van Harten PN, Hoek HW, Kahn RS. Acute dystonia induced by antipsychotic drugs. Eur Neuropsychopharmacol. 1999;9(6):453–7.
7. Haddad PM, Sharma SG. Adverse effects of atypical antipsychotics. CNS Drugs. 2007;21(11):911–36.
8. Friedman JH. Drug-induced parkinsonism. Curr Treat Options Neurol. 2014;16(4):281.
9. Factor SA, Molho ES. Movement disorders: Neurologic principles and practice. McGraw Hill; 2008.
10. Meltzer HY. Update on typical and atypical antipsychotic drugs. Annu Rev Med. 2013; 64:393–406.
11. Casey DE. Extrapyramidal syndromes and tardive dyskinesia. Schizophr Res. 1991;4(2):109–20.
12. Miller DD. Review and management of antipsychotic adverse effects. J Clin Psychiatry. 2000;61(Suppl 8):14–7.
13. Aquino CC, Lang AE. Tardive dyskinesia syndromes. Parkinsonism Relat Disord. 2014;20(Suppl 1): S113–7.
14. Lieberman JA, Stroup TS, McEvoy JP, et al. Effectiveness of antipsychotic drugs. N Engl J Med. 2005;353(12):1209–23.
15. Gitlin M. Lithium side effects and toxicity. Int J Bipolar Disord. 2016; 4:27.
16. Stahl SM. Essential psychopharmacology. 4th ed. Cambridge: Cambridge University Press; 2013.
17. Rang HP, Dale MM, Ritter JM, Flower RJ. Rang and Dale's pharmacology. 8th ed. Elsevier; 2016.
18. Katzung BG, Trevor AJ. Basic and clinical pharmacology. 14th ed. McGraw Hill; 2018.
19. Olanow CW, Stern MB, Sethi K. The scientific and clinical basis for Parkinson disease. Neurology. 2009;72(Suppl 4): S1–136.
20. Lang AE, Lozano AM. Parkinson's disease. N Engl J Med. 1998;339(15):1044–53.
21. Debette S, Markus HS. Clinical significance of white matter hyperintensities. BMJ. 2010;341:c3666.
22. Fazekas F, Chawluk JB, Alavi A, et al. MR signal abnormalities in aging brain. AJR Am J Roentgenol. 1987;149(2):351–6.
23. Raz N, Rodrigue KM. Differential aging of the brain. Neurosci Biobehav Rev. 2006;30(6):730–48.
24. Borenstein DG. Epidemiology, etiology, diagnostic evaluation of low back pain. Curr Opin Rheumatol. 2001;13(2):128–34.
25. Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med. 2000;342(21):1581–9.
26. Suchowersky O. Parkinson disease overview. Neurol Clin. 2002;20(3):645–60.
27. Chou KL. Diagnosis and differential diagnosis of Parkinson disease. UpToDate; 2020.
28. Brigo F, Erro R, Marangi A, et al. Differentiating drug-induced parkinsonism. Parkinsonism Relat Disord. 2014;20(1):12–6.
29. Koller WC. Drug-induced movement disorders. Neurol Clin. 1984;2(3):699–713.
30. Marsden CD. Extrapyramidal disorders. J Neurol Neurosurg Psychiatry. 1982;45(12):1087–99.
31. Keepers GA, Fochtmann LJ, Anzia JM, et al. APA schizophrenia guideline. Am J Psychiatry. 2020;177(9):868–72.
32. National Institute for Health and Care Excellence. Psychosis and schizophrenia guideline. London: NICE; 2014.
33. World Health Organization. Safety monitoring of medicinal products. Geneva: WHO; 2002.
34. Burn DJ, Tröster AI. Neuropsychiatric complications of Parkinson's disease. Lancet Neurol. 2004;3(9):547–56.

35. Tolosa E, Wenning G, Poewe W. The diagnosis of Parkinson's disease. Lancet Neurol. 2006;5(1):75–86.
36. van Harten PN, Matroos GE, Hoek HW, et al. Incidence of EPS with atypical antipsychotics. J Clin Psychiatry. 1998;59(Suppl 12):13–8.
37. Peluso MJ, Lewis SW, Barnes TR, et al. Extrapyramidal motor side-effects of antipsychotic drugs. Br J Psychiatry. 2012;200(5):387–92.
38. Remington G. Understanding antipsychotic "atypicality". J Psychiatry Neurosci. 2003;28(4):275–84.
39. Seeman P. Dopamine receptors and antipsychotic drugs. Synapse. 2002;45(2):59–89.
40. Kapur S, Mamo D. Half a century of antipsychotics. Am J Psychiatry. 2003;160(12):2143–54.
41. Mena MA, García de Yébenes J. Drug-induced Parkinsonism. Expert Opin Drug Saf. 2006;5(6):759–71.
42. López-Sendón J, Mena MA, de Yébenes JG. Drug-induced movement disorders. Drug Saf. 2013;36(5):295–314.
43. Factor SA. Current status of symptomatic medical therapy in Parkinson's disease. Neurotherapeutics. 2008;5(2):164–80.
44. Friedman JH, Factor SA. Atypical antipsychotics in Parkinson disease. Mov Disord. 2000;15(2):201–11.
45. Jankovic J. Parkinson's disease: clinical features and diagnosis. J Neurol Neurosurg Psychiatry. 2008;79(4):368–76.
46. Aquino CC, Fox SH. Clinical spectrum of levodopa-induced complications. Mov Disord. 2015;30(1):80–9.
47. Rizek P, Kumar N, Jog MS. An update on drug-induced movement disorders. Curr Neurol Neurosci Rep. 2016;16(6):54.
48. Tinazzi M, Antonini A, Bovi T, et al. Clinical and imaging studies in DIP. Mov Disord. 2009;24(12):1715–24.
49. Tolosa E, Garrido A, Scholz SW, et al. Challenges in diagnosis of Parkinson's disease. Lancet Neurol. 2021;20(5):385–97.
50. Fusar-Poli P, Smieskova R, Serafini G, et al. Antipsychotic safety and tolerability. CNS Drugs. 2014;28(5):383–99.

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