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Levodopa Induced Dyskinesia With Rhabdomyolysis In Parkinson's Disease - Case Report

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Abstract

Parkinson's disease is a neuro-degenerative disorder characterized by loss of dopaminergic neurons in the substantia nigra pars compacta. Levodopa is the gold standard in the treatment of the disease. Levodopa induced dyskinesia is a treatment limiting complication. Rhabdomyolysis due to levodopa induced dyskinesia is a rare and life threatening condition. There are only a few case reports in literature of levodopa induced dyskinesia with rhabdomyolysis. Herein we report a case of Rhabdomyolysis due to levodopa induced dyskinesia in a patient with Parkinson's disease.

Conclusion

In any patient with advanced Parkinson's disease presenting with dyskinesia rhabdomyolysis- levodopa induced dyskinesia should be considered. Early recognition and prompt treatment results in favorable outcomes.

Keywords: Parkinson's disease, Rhabdomyolysis, Levodopa induced dyskinesia

Introduction

Parkinson's disease is a neuro-degenerative disorder characterized by loss of dopaminergic neurons in the substantia nigra pars compacta. This results in progressive motor symptoms, including tremors, bradykinesia, rigidity, and impaired posture [1]. The mainstay of treatment is levodopa which along with carbidopa, a peripheral decarboxylase inhibitor is the gold standard for symptomatic treatment of Parkinson's disease. [2]

During the early stages of the disease treatment with levodopa is very effective as the spared dopaminergic neurons are able to store exogenous dopamine and regulate its release so that normal physiological DA receptor stimulation within the stratum is maintained. However levodopa induced dyskinesia is a common complication of therapy in advanced stages of the disease and often becomes treatment limiting. Prevalence of Levodopa-induced dyskinesia is approximately 30% and 50% after 5-year and 10-year of initiating levodopa therapy, respectively [3].

Risk factors for developing Levodopa-induced dyskinesia are younger age of onset of Parkinson's, female gender, low body weight and North American ethnicity [4]. Rhabdomyolysis associated with

levodopa induced dyskinesia is extremely rare and life threatening condition. Complications include acute kidney injury, electrolyte imbalances, cardiac arrhythmias and death. Early diagnosis and prompt treatment is the key.

There are only a few case reports in literature of levodopa induced dyskinesia with rhabdomyolysis. Here we report a case of levodopa induced dyskinesia complicated by rhabdomyolysis in a patient with Parkinson's disease.

Case Report

A 62 year old lady, known case of Parkinson's disease since 12 years and type 2 diabetes mellitus since 13 years presented with generalized dyskinesia since 2 days. She was prescribed tab syndopa plus (levodopa 100mg+carbidopa 10mg) 5 times a day, tab amantadine 129 mg once daily tab selegeline 5 mg once daily tab pramipexole 0.25 twice daily, tab pacitane 2 mg thrice daily and tab glimiperide and metformin combination (1mg,500mg) for diabetes mellitus. Her daughter gives history of intake of tab syndopa plus (levodopa 100mg+carbidopa 10mg) multiple times a day by the patient to control her symptoms, more than the prescribed dose. At the time of admission her vital signs were - pulse rate 104/min, blood pressure 110/70 mm Hg, respiratory rate of 24/min, SpO2 88% in room air, increased to 99% with oxygen via facemask at 4L/min, temperature 101^0F and GCS was E3V3M5. There were no chest signs. She had tongue bite leading to oral bleeding and generalized dyskinesia involving all the limbs and neck. She was electively intubated and ventilated to secure her airway. Blood investigations revealed elevated leucocyte count ($17,570/mm^3$), slightly increased serum creatinine (1.1 mg/dL) and hyperkalemia (serum potassium 6.4mEq/L). Creatine phosphokinase was markedly elevated (15,000U/L). Urinalysis showed presence of myoglobinuria. CT brain and CSF study was normal. Working diagnosis was Levodopa-induced dyskinesia with rhabdomyolysis and aspiration pneumonia after ruling out other causes of rhabdomyolysis. All anti Parkinson medications were stopped. She was treated with intravenous fluids, antihyperkalemic measures, forced alkaline diuresis and intravenous antibiotics. She had worsening of creatine levels (4.4 mg/dL), oliguria and persistent hyperkalemia for which she underwent 4 cycles of hemodialysis. Her dyskinesia gradually improved and syndopa plus (levodopa +carbidopa) was restarted at a lower dose and gradually increased over 1 week to achieve maximum dose of four times a day. Her creatine kinase levels showed a decreasing trend and by day ten her creatine phosphokinase levels reduced to 350U/L, her potassium level normalized, urine output normalized and serum creatinine levels stabilised (1.1mg/dL). Her GCS improved to 15. She was extubated on day ten. At present she is asymptomatic and she is being planned for discharge.

Discussion

Levodopa is the gold standard drug in treatment of motor symptoms of Parkinsons disease. It is the metabolic precursor of dopamine and is converted to dopamine by dopa decarboxylase. Carbidopa is combined with levodopa to prevent its peripheral conversion to dopamine and thereby reduce side effects like nausea and vomiting. It also helps in lowering the dose of levodopa required for treatment. The use of levodopa is limited by the development of Levodopa-induced dyskinesia and is related to disease progression [5]. It can present as chorea, athetosis, dystonia, ballismus or myoclonus [6]. The most common type is chorea. The three most common types are peak-dose dyskinesia, diphasic dyskinesia, and early-morning dystonia. It usually starts on the side first affected by Parkinson's. The pulsatile stimulation of dopamine receptor due to short half-life of levodopa is the main mechanism of Levodopa-induced dyskinesia which increases as the disease progresses [7]. Treatment options include adjustment of dosing of levodopa and amantadine - an NMDA receptor antagonist. The most common causes of rhabdomyolysis in adults are drugs and toxins, trauma, excessive muscular activity, temperature extremes, muscle ischemia, prolonged immobilization, infection, electrolyte and endocrine abnormalities, genetic disorders, connective tissue disorders and unknown conditions [8]. In Parkinson's disease, Levodopa-induced dyskinesia leading to rhabdomyolysis is very rare complication of treatment. The exact mechanism is unknown. Loss of dopaminergic neurons due to disease progression may play a role in its development. Other causes include up-titration of dopaminergic medications, switching dopaminergic medication from immediate-release to extended-release formulation, infection, trauma, dehydration, and living in high

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environmental temperature. In our case excessive intake of tab syndopa plus (levodopa 100mg+carbidopa 10mg) was the cause of dyskinesia which led to rhabdomyolysis. The key to successful management in patients with Levodopa-induced dyskinesia with rhabdomyolysis include early recognition, prompt reduction in dopaminergic medications, treating possible precipitating factors, and proper supportive treatment, such as rehydration and hemodialysis. In our case early diagnosis was aided by a proper history from the bystanders. With the timely implementation of appropriate interventions the patient recovered.

Conclusion

In any patient with advanced Parkinson's disease presenting with dyskinesia clinicians should have a high index of suspicion and levodopa induced dyskinesia should be considered as a differential diagnosis. Rhabdomyolysis should be considered as a possible complication in patients presenting with levodopa induced dyskinesia. Early recognition of the condition and prompt treatment lead to excellent outcomes.

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