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The Recognition and Management of Respiratory Dyskinesia in Parkinson Disease: A Case Report and Literature Review

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Case Report

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Abstract

Background: Isolated respiratory dyskinesia is rarely reported rather than other symptoms involving choreiform or dystonic movements of orofacial muscles, neck, limbs and trunk in idiopathic Parkinson Disease (PD). Because of the potential comorbidity of respiratory dysfunction for PD patients, isolated or predominantly respiratory dyskinesia as a consequence of levodopa (L-dopa) therapy may be underrecognized, and extensive investigations are carried out, often repeatedly during recurrent admission, looking for infection, pulmonary emboli, heart failure and anxiety. So, it is important for clinicians to recognize the symptom accurately.

Case presentation: We report a patient of Parkinson disease manifesting as stressfully alternating irregular tachypnea, shortness of breath, stridor and brief periods of apnea after administration of Levodopa. With no features of autonomic insufficiency. Chest CT scans, pulmonary function and related examinations of pulmonary embolism were none revealed. These symptoms disappeared after treatment according to dyskinesia.

Conclusions: Respiratory dyskinesia induced by Levodopa occurred rarely in Parkinson disease, especially isolated respiratory dyskinesia could be the single symptom without any other simultaneous phenomenon such as limbs or trunks dyskinesia. It is very important to recognize it timely, prevent and relieve respiratory obstruction, especially tracheotomy necessarily and adjust the drugs according to the rule of dyskinesia induced by Levodopa. The mechanism of it is not unclear, may be the concurrent of peripheral and central respiratory disorders induced by levodopa pulse stimulation.

Keywords: Dyskinesia; Levodopa; Parkinson Disease; Respiratory Disorder

Abbreviations: BID: Twice a Day; D2: Dopamine D2 Receptor; DBS: Deep Brain Stimulation; GPI: Globus pallidus; LID: Levodopa-Induced Dyskinesia; PD: Parkinson Disease; QID: Quarter a Day; STN: Subthalamic Nucleus; TID: Three Times a Day; UAO: Upper Airway Obstruction

Case Report

A 75-year female was admitted to our hospital with her perplex dyspnea. She had a 10-year history of PD. With the onset of static tremor and bradykinesia of right limb, which gradually spread to the other side two years ago. Her parkinsonism improved well when treated with levodopa/benserazide, which dosages gradually escalated from 50/12.5 mg TID to 100/25mg QID three years ago, accompanied with the recommencement of entacapone 1 tablet QID. She developed worsening parkinsonism and the trouble of motor complications when she was first admitted to our hospital, involving motor fluctuations and dyskinesia, manifesting as dyspnea accompanied with involuntary choreiform movements of upper

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limbs and trunk (Video 1), lasting for 1.5 hours, disappeared after stopping entacapone, nonetheless accompanied with worsening bradykinesia, then relieved by the combination of selegiline. These symptoms emerged once again two years ago (Video 2), dyspnea did not relieve when stopping selegiline and decreasing levodopa/benseride to 50/12.5 mg three times a day, disappeared when stopping all drugs. Meanwhile the motor disorders aggravated to bedridden, admitted to our hospital. Neurological examination was consistent with idiopathic PD, with no features of autonomic insufficiency.

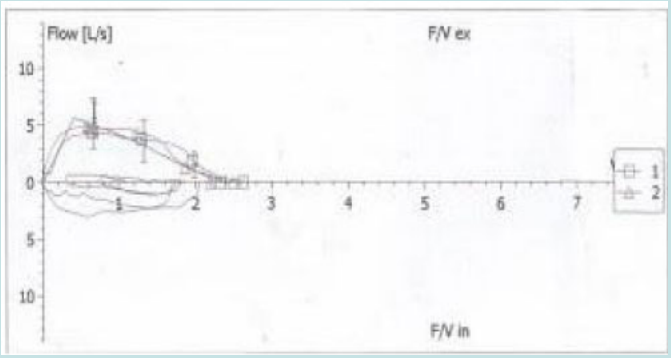
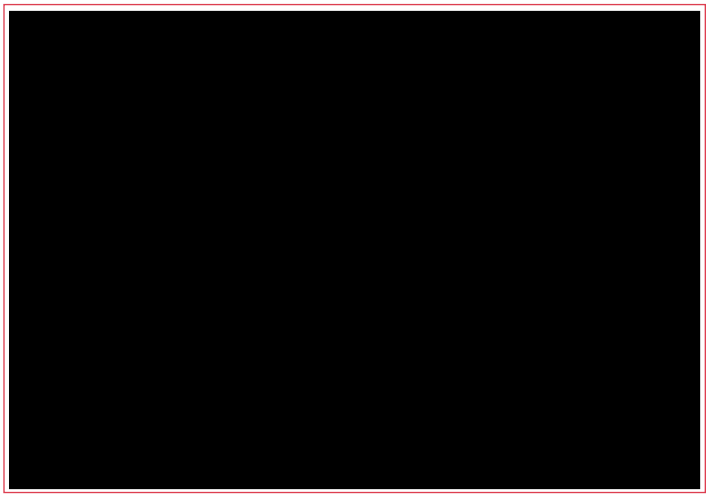


Figure 1: The flow-volume loop of the PD patient. Ordinate: Flow(L/s), Abscissa: Volume (L).

Chest CT scans, pulmonary function (Figure 1) and related examinations of pulmonary embolism were none revealed. With treatment of levodopa/benseride 50/12.5 mg QID, her bradykinesia improved otherwise she cannot ambulate independently (Video 3), occasional dose-peak involuntary limb dyskinesia appeared after combination of pramipexole 0.125mg TID (Video 4), and disappeared with the treatment of amantadine 100mg BID (video 5), then the parkinsonism improved well. The symptoms of dyspnea have no longer occur as followed six months with the treatment of levodopa/benseride 50/12.5 mg QID, pramipexole 0.125mg TID and amantadine 100mg BID.

Video 1

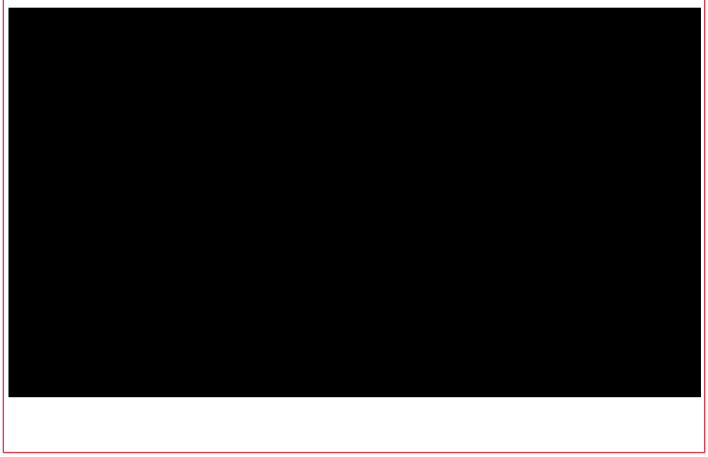


Video 1: The patient manifesting indescribable dyspnea accompanied with involuntary choreiform movements of upper limbs and trunk.

Video 2

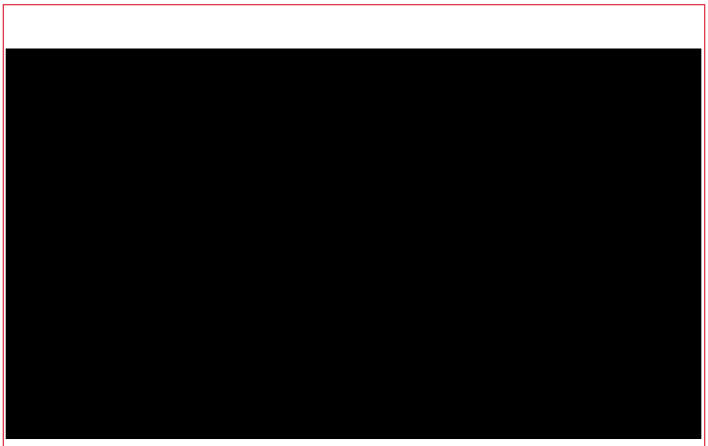
Video 2: The patient manifesting indescribable dyspnea accompanied with dyskinesia of upper limbs and trunk.

Video 3



Video 3: The patient manifesting bradykinesia, she cannot ambulate independently but dyskinesia disappears.

Video 4



Video 4: The patient manifesting slight dyskinesia but no dyspnea.

Video 5

Video 5: The patient manifesting bradykinesia but can ambulate independently and do not appear dyskinesia.

Discussion

Parkinson's Disease (PD) is one of neurological degenerative diseases which is characterized by resting tremor, bradykinesia, rigidity and postural instability. Levodopa is a highly effective pharmacotherapy, which is attributed to the motor dysfunction of PD is strongly linked with nigrostriatal dopaminergic denervation [1-2]. Movement disorders gradually appeared followed long-term taking of Levodopa, the treatment of PD is essentially an equilibrium between adequate relief of motor symptoms and control of motor complications, including motor fluctuations (wearing off, on-off and freezing) and Levodopa-Induced Dyskinesia (LID).

LID emerge as a clinical problem in almost all after a decade of dopaminergic therapy and around half of patients after 5 years [3], including peak-dose dyskinesia, biphasic dyskinesia and dystonia. often involved in the relatively serious hemi-side, characterized by choreograph, athetosis, dystonia and so on. The respiratory dyskinesia had rarely reported.

Initially respiratory disorder is supposed to correlated with anxiety, bradykinesia and cardiopulmonary dysfunctions, and may due to the front torso tilts and thoracic deformation which accompanied with disease progressing.

The dyspnea which we describing is diagnosed dose-peak dyskinesia as follows:

(1) Dyspnea always happened almost 1 hour after taking levodopa, which conforming to dose peak.

(2) Dyspnea accompanied with classical symptoms of dyskinesia, involuntary torso twist.

(3) Dyspnea disappeared when treating according to the principle of dyskinesia.

(4) Objective examinations of respiratory has no abnormal.

It has been reported that symptoms of dyspnea such as irregular respiratory rate and depth occurred 30-120 minutes after taking Levodopa, coinciding with dose peak effect [4-7]. The clinical manifestations mainly include rapid and severe abnormal breathing, especially apnea and stridor, respiratory frequency speed up and respiratory depth shallowed. These symptoms always appear when the therapy of Levodopa or the dose of Levodopa accelerating, accompanied with involuntary movements, and disappears when adjusting therapy treatment according to dose-peak dyskinesia. Which is identical to this case.

Symptomatic respiratory dyskinesia in idiopathic PD has been more underrecognized in clinical practice rather than dyspnea with shallow or rapid respiratory as an off-period phenomenon, Levodopa-induced diaphragmatic dyskinesias may lead to distressful dyspnea [8], especially peak-dose, which is associated with the administration of levodopa and ergot-derived dopamine agonists [9]. It is prone to emerge when the discontinuation of anti-Parkinson medications and stopped levodopa. On account of the extensive evaluation of cardiac and pulmonary disorders always emergence when shortness of breathing happened in the older population of PD patients, the adverse effect of LID should be considered early in differential diagnosis, which would subside or disappear after reducing or stopping dopaminergic administration.

The explanations of peak-dose LID may be as follows:

(1) The chorea of respiratory muscles results in paradoxical chest and abdominal movements when including breathing, rapid shallow breathing and a decline in pulmonary function have been detected by pulmonary function tests and inductive plethysmography [10].

(2) Respiratory is controlled through a complex system including motor and premotor cortex, brainstem, and cervical phrenic nucleus and nerve, sensors and autonomic system. Dopamine medicate the central control of ventilatory rhythm at multiple levels. Dopamine involved in both peripheral chemoreceptor and brainstem respiratory center, the destruction of the substantia nigra and loss of dopaminergic neurons in PD patients can reduce peripheral chemosensitivity, resulting in the denervation hypersensitivity of peripheral chemoreceptor [11], which always produce tachypnea. Otherwise, dopamine-containing neurons in brainstem control breathing [12], the sensitivity of dopaminergic receptor may play's an important role in the LID.

Upper Airway Obstruction (UAO) and restrictive respiratory dysfunction is the main accepted etiology during various respiratory abnormalities in PD patients [13]. It is well known that symptomatic UAO is not common in PD. Little is known as mentioned stridor, which is the lethal form of UAO. Cheah et al [14] has demonstrated that the three etiologies of stridor in PD as follows: bilateral vocal cord paralysis, laryngeal spasm, and dystonia of the pharyngeal, jaw, and neck muscles. UAO is associated with the high degree of dorsal column arthrosis which due to restriction in movement affected by thoraco-abdominal movements [13].

Bilateral vocal cord paralysis is the most common reason of stridor, which can be detected with the laryngoscopy, it is the predictor that high-pitched sounds occasionally when agitated in normal life for PD to exposed to stridor [13]. The pathogenesis of vocal cord paralysis may involve in the degeneration of the nucleus ambiguous, which has been demonstrated in patients with multiple system atrophy [15].

Laryngeal spasm is the second reason for stridor, striated muscles of the upper airway are invariably involved in the

involuntary movement characteristic of PD, moreover, copious oral secretion, can trigger laryngospasm [16].

The rare cause of stridor is the dystonia of the pharyngeal, jaw, and neck muscles. In our case, the stridor associated with dyskinesia of limb mostly due to the above reasons and abnormal thoraco-abdominal movements.

When the emergence of respiratory dyskinesia, especially severe stridor in acute setting, the priority management is secure airway quickly and safely, intubation and tracheostomy are often indicated [15,17]. Secondly, respiratory dyskinesia can be disappeared with titrated haloperidol which is a butyrophenone neuroleptic that acts via competitive blockade of D2 family receptors on basal ganglia post-synaptic neurons [18]. Thirdly, respiratory dyskinesia can be resolved with reduction or minimization of levodopa and administration of dopamine agonist [19]. Lastly, respiratory dyskinesia can be also treated with surgery operation, such as GPI-DBS [20] and STN-DBS [8], Surprisingly, respiratory dyskinesia can happen post-operation of DBS [21].

In conclusion, the mechanism of respiratory dyskinesia of PD is not unclear, we will insist on studying this item on the future days.

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