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Tremor and Other Hyperkinetic Movements

New Observations Letter

A Case of Severe Myoclonus due to Carbon Dioxide Retention in the Setting of Chronic Obstructive Pulmonary Disease Exacerbation

Halil Onder

Neurology Clinic, Yozgat City Hospital, Yozgat, Turkey

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*To whom correspondence should be addressed. E-mail: halilnder@yahoo.com

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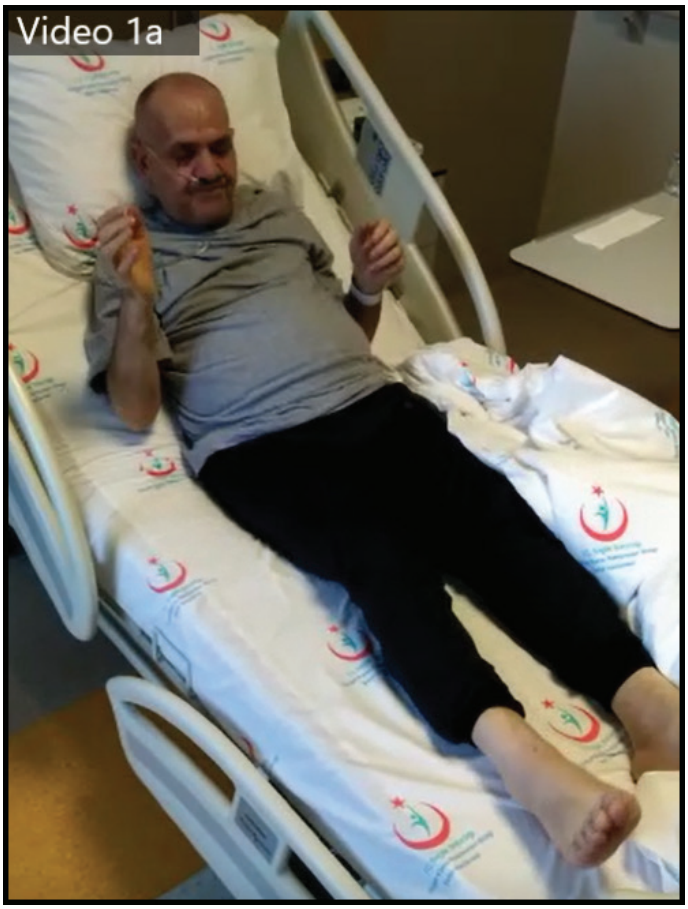
Dear Editor

A-70-year-old male patient with a history of chronic obstructive pulmonary disease (COPD) over the previous 10 years was referred from Physical Therapy Department for etiological investigations on his inability to walk. His medical history revealed that he had been discharged from pulmonology 3 weeks ago after the administration of medications for COPD exacerbation. He was hospitalized for 2 weeks and received antibiotherapy of ceftriaxone (2 g intravenous daily for 1 week) for pneumonia, medication for COPD, as well as bilevel positive airway pressure (BIPAP) therapy. However, after discharge, BIPAP therapy could not be administered due to social problems (his relatives did not show any interest in the patient, and he had no support for the administration of BIPAP therapy at home). After being discharged from hospital, it was noticed that he could not walk or even mobilize due to severe involuntary jerks in his whole body. Therefore, he has applied to physical therapy polyclinic (2 weeks after being discharged), from which he was referred to neurology clinic. At admission to neurology polyclinic, the patient was not taking any medication except for bronchodilator therapy including salbutamol and ipratropium inhaler. He was evaluated as fully orientated and cooperated. However, he had poor mobilization, and he was dependent on a wheelchair. There were severe, sudden, brief jerks in his whole body, which were aggravated during mobilization (Video 1). These jerks were so powerful that he could not move from lying to a sitting position without any support due to severe jerks compatible with myoclonus. Other neurological examinations including motor and sensory examination were within normal ranges. The patient was hospitalized in order to conduct etiological investigations for myoclonus. Vital signs were regular (temperature: 36.2°C, pulse: 90, SPO_2: 90%). Cranial *magnetic resonance imaging* (MRI) was in normal range (Figure 1). Laboratory investigations revealed mild leukocytosis (hemoglobin [Hb]: 12.7/dL, white blood cell counts [WBC]: 16,000/mm^3, plt: 233,000/mm^3), and hypocalcemia (7.9 mg/dL). However, arterial blood gas test showed carbon dioxide retention (pH: 7.33, pCO_2: 76, pO_2: 26.8, HCO_3: 41.2). Taken together, carbon dioxide retention was considered as the cause of myoclonus in the forefront, and the patient was referred to pulmonology department to receive therapy for COPD exacerbation on the same day of hospitalization. Increases in bronchial secretions were apparent, and wheezing was detected on auscultation. Chest X-ray showed chronic signs of COPD. BIPAP treatment was suggested, with initiation of piperacillin/tazobactam (4.5 g thrice daily) at first due to the diagnosis of atypical pneumonia. However, BIPAP therapy did not provide any amelioration in blood gas test results. Hence, the patient was intubated and taken to intensive care unit (on the second day of hospitalization). After the following 3 days, decrease in bronchial secretions was achieved and the need for aspiration was significantly reduced. Remarkably, the myoclonic movements were almost recovered following intubation, soon after normalization of hypercarbia. After final evaluation by pulmonology department, it was decided that the patient was able to protect the airway and maintain sufficient ventilation. Hence, following gradual preparation for pre-extubation, extubation was performed (the patient could easily tolerate intubation into such a level that



Video 1. **Myoclonic Movement Which Was Recorded Soon after Hospitalization at the Same Day of Admission (Concurrently pCO2:76)**. (a) Images recorded while the patient was trying to mobilize from lying position to sitting position. (b) Images recorded while the patient was trying to put his body into standing position.

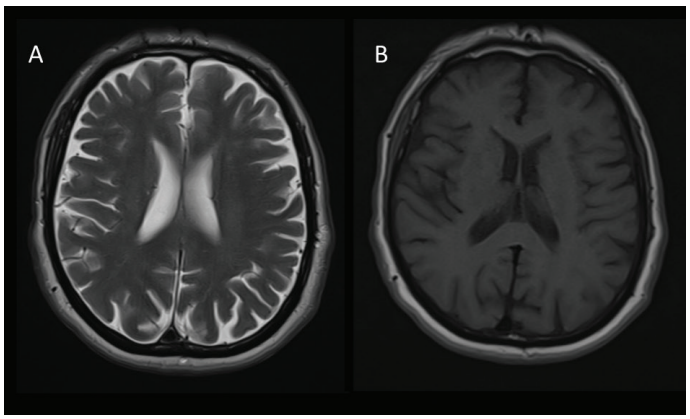
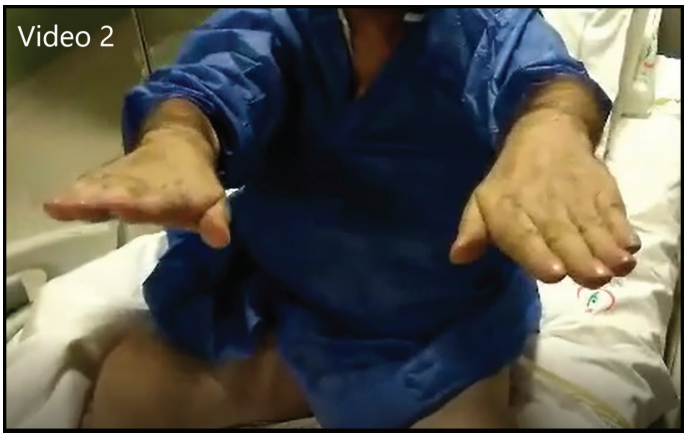


Figure 1. **Cranial MRI.** Cranial MRI (T2 and T1 Sequences) of the Patient, Showing Normal Findings.

no sedation and analgesia were required during the pre- and post-intubation periods). On neurological examination, it was observed that myoclonic jerks were significantly reduced (Video 2). Concurrently performed arterial blood gas test revealed significant improvement of hypercarbia (pCO_2: 42.5, ph: 7.49, pO_2: 79.7, HCO_3: 33). After 1 week of hospitalization, piperacillin/tazobactam was stopped, and the patient was discharged with the recommendation of BIPAP therapy.

Discussion

Myoclonus is characterized by sudden, brief, shock-like involuntary movements, associated with bursts of muscular activity or silencing of muscular activity. There are many classifications of myoclonus, including clinical, neuroanatomical, and etiological aspects. Neurodegenerative diseases, stroke, and ictal events can be postulated as the forefront etiological causes of myoclonus.[1] On the other hand,



Video 2. **Post-treatment.** Video, Recorded on the 6th Day of Hospitalization, Showing Recovery of Myoclonic Jerks after Treatment of Carbon Dioxide Retention (pCO_2:42.5).

metabolic encephalopathies are also considered as a common cause of myoclonus, which are typically seen in the intensive care unit.[1] Hyponatremia, hepatic encephalopathy, uremia, hypophosphatemia, hypo or hyperglycemia, hyperthyroidism, and pulmonary encephalopathy (carbon dioxide retention) have been reported as the major causes of myoclonus associated with metabolic encephalopathies.[1] Among those, carbon dioxide retention has been acknowledged as an inducer of myoclonus for quite a long time.[1,2] However, to our knowledge, there is no proper demonstration of a case with myoclonus induced by carbon dioxide retention in the literature. Therefore, we think that the presentation of this patient, in whom a significant amelioration of myoclonus was achieved after COPD therapy, may provide a substantial perspective for clinicians. Through the presentation of this patient, first, we attract attention to the fact that carbon dioxide retention is a crucial cause of myoclonus among metabolic agents. In addition, remarkably, the patient did not suffer from dyspnea. Besides, on neurological examination, he was evaluated as orientated and cooperated (findings not compatible with encephalopathy). We think that the recognition of this point may be crucial for timely etiological diagnosis such that it can even be the only manifestation of COPD exacerbation, as in our case.

References

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