Post Infectious Opsoclonus-Myoclonus Syndrome in a Cov-2 Infected Patient

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Abstract

Opsoclonus-Myoclonus syndrome (OMS) is a rare neuroinflammatory disorder characterized by rapid and involuntary eye movements (Opsoclonus), general involuntary muscles jerks (myoclonus) and ataxic gait.

In adults, OMS can be paraneoplastic in origin (lung and breast cancer) and also related to viral infections, metabolic disorders or post streptococcal pharyngitis. However, OMS has been rarely described secondary to acute respiratory diseases. We describe a patient showing an OMS few days after mild respiratory symptoms attributable to CoV-2 pneumonitis. Cranial MRI discharged acute brain injury and other tests including Spine fluid discharged a direct effect on the CNS. Patient improved after one week of corticoids therapy. We propose an OMS post SARS-CoV-2 infection as a possible etiology.

Keywords: Opsoclonus-Myoclonus syndrome (OMS); SARS-CoV-2; MRI

Introduction

OMS typically presents in children, associated to brain tumors (neuroblastomas 50%). It is characterized by opsoclonus (rapid, multidirectional, conjugate eye movements), myoclonic jerks, ataxia and sleep disturbances. The clinical course may be monophasic or chronic relapsing. A similar adult-onset condition also occurs but is associated with different types of cancer, most commonly small-cell lung cancer and adenocarcinoma of the breast. Nevertheless, OMS has been associated to some infectious and post infectious disorders [1,2]. OMS patients use to show a monophasic course and they show a good response to immunological medical strategies (corticoids or Immunoglobulins). To our knowledge, it has not been reported OMS patients associated to the SARS-CoV-2 infection.

Case Report

A 68 year-old man came to our emergency room giving 6-days history of a mild global headache, fatigue, persisting cough and fever (37.5°C) and also, he referred a progressive tremor in his left arm associated to gait difficulties in the last 48 hours.

In his medical record, just only showed depression and High blood pressure.

On presentation, the patient was oriented but drowsy and slow to respond, his temperature was 38.6°C, blood pressure 140/87 mm Hg, pulse rate 90 beats/min and respiratory rate 20 breaths/min.

On neurological examination, he showed Opsoclonus when saccadic eyes movements were explored, predominantly to the left side and no other cranial nerves impairment were observed. He also showed generalized myoclonic movements predominately in his left arm which increased when any voluntary movement was tried and a limb dysmetria in his left arm and an ataxic gait (Video).
A blood count showed a white blood cell count of 8,630/ml (4100 to 10,900/ml), C-reactive protein was 14.3 mg/L (< 10 mg/L), Lymph count 1,65 (1 - 3), Ferritin 1977 (30 - 400 ng/dl), Dimer-D 1111 ng/dl (0 - 500 ng/dl); PCR for CoV-2 was Positive. Cerebrospinal fluid (CSF) tests were normal: (cells count 0 cells, protein 11 mg/dl (1.5 - 45 mg/dl) and glycorraquia 65 mg/dl (glycemia 78 mg/dl) CSF culture was negative for bacteria and PCR for virus such as HSV-1, HSV-2, VZV, CMV HIV and CoV-2.

The Thorax X-Ray showed a bilateral interstitial pneumonia pattern and a normal EKG. The Cerebral MRI was negative for brain injury.

Lopinavir/ritonavir and hydroxychloroquine were prescribed showing stability in regard to the respiratory parameters but not the neurological symptoms, then after iv Methylprednisolone 500 mg daily for 5 days was prescribed in the second week of ingress showing a clear improvement of the OMS.

Lung cancer and other neoplastic disorders were ruled out because CT Abdominal/Thorax were normal and onconeural antibodies were not found (anti-Hu, Yo, Ma2, CRMP-5, amphiphysin and Ri).

Patient was discharged from the hospital at the 5th-week of ingress referring a moderate tremor in his left hand and mild walking difficulties.

Discussion

Although OMS can be paraneoplastic in origin, it can also result from viral infections, post-streptococcal pharyngitis, metabolic disorders, metastases, and intracranial hemorrhage.

Some neurological symptoms have been associated to CoV-2 infected patients during the COVID19-pandemic such as ischemic cerebrovascular disease attributable to the hypercoagulability and a possible cytokine storm reaction [4,5]. Other authors have reported polyneuropathies and even more, a necrotizing encephalomyelitis attributed to a neuro-invasive effect of a SARS-Cov-2 patient [6].

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Our patient showed an opsoclonus-myoclonic syndrome 1-week after mild systemic clinical manifestations attributable to SARS CoV-2 infection, possibly, the CoV-2 pneumonia was the trigger of the neurological symptoms (OMS).

We ruled out a direct effect of any microorganism on the CNS because the CSF was negative for all the microorganism including PCR for CoV-2. We are aware about the limitations of the validity of PCR for CoV-2 in CSF; however, an immune-mediated post infectious cause seems to be the most probable etiology implicated in our case.

The bilateral pneumonia attributable to a SARS CoV-2 infection possibly triggered an immuno-mediated reaction on the dentato-rubral and pallido-Luysian system of the brainstem.

Brainstem ischemic stroke and other brain injuries such as acute disseminated encephalopathy or acute hemorrhagic leukoencephalopathy were also ruled out because the Cerebral MRI and CSF were absolutely normal.

In the case of post infectious CNS diseases, there are no biologic markers and cerebral magnetic resonance imaging is essential to diagnosis, in detecting diffuse or multifocal asymmetrical lesions throughout the white matter on T2 and FLAIR-weighted sequences and high-dose intravenous steroids are accepted as first-line therapy [7,8]. Our patient showed no abnormalities in the Cerebral MRI probably because the early steroids therapy prescription could modulate a stronger immunological reaction over the brainstem.

Conclusion

We show a case report of a patient who developed an opsoclonus-myoclonus syndrome after one week of mild general symptoms associated to SARS-CoV-2 pneumonia. We ruled out a direct effect of the SARs-2 on the central nervous system and relate all the neurological symptoms to a post infectious immunomodulated response to the COVID-19 pneumonia.

A high prevalence of “atypical variants” was found in some series with site-restricted or not visible damage. Probably our case could be considered an “atypical variant” of a post infectious neurological disorder [9].

Bibliography


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