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## Introduction & Importance

Oculomotor nerve palsy is an ocular pathology due to injury to the third cranial nerve. Partial oculomotor palsy can present with variable eye duction movements and varying degrees of ptosis and pupillary dysfunction<sup>1</sup>. Patients with complete oculomotor palsy present with complete ptosis, “down and out” eye deviation, a dilated pupil with sluggish reaction, and diplopia<sup>2</sup>. Dysfunction can result from multiple different etiologies including intracranial aneurysms, microvascular disease, trauma, viral infections, and compression from masses<sup>3</sup>. Prognosis is typically good and self-limiting, and occlusion with a patch can be helpful<sup>4</sup>. Reports of COVID-19 infections causing oculomotor nerve palsies have been documented<sup>5</sup>, but the use of corticosteroid has not been fully investigated since very few reports have been identified<sup>6-8</sup>.

## Case Presentation

An otherwise healthy 40-year-old male awoke on August 11<sup>th</sup>, 2022, with moderate diplopia, unable to focus with binocular vision and developed eyelid ptosis two days later. He had a prodrome of sore throat and diarrhea 5 days prior. He had been infected with Omicron on January 5<sup>th</sup>, 2022, with mild rhinitis symptoms; however, a rapid test could not confirm it. No intracranial or vascular pathology were identified on CT head, CT angiogram, or MRI. Repeat COVID-19 PCR test was negative. Neuro-ophthalmology diagnosed him with left partial oculomotor nerve palsy presumed secondary to viral microvascular injury. COVID-19 infection seemed likely given the history but could not be confirmed. He elected treatment with a left eye patch and dexamethasone 8 mg PO TID for 7 days, with a taper over 5 days. By day 7, partial eye mobility began to return. Functional binocular vision was present by day 14. Near complete function was restored on day 20 to the point where he had normal depth perception and no longer needed the eye patch. Full return of function in all extremes of gaze was restored on day 52.

## Discussion & Conclusion

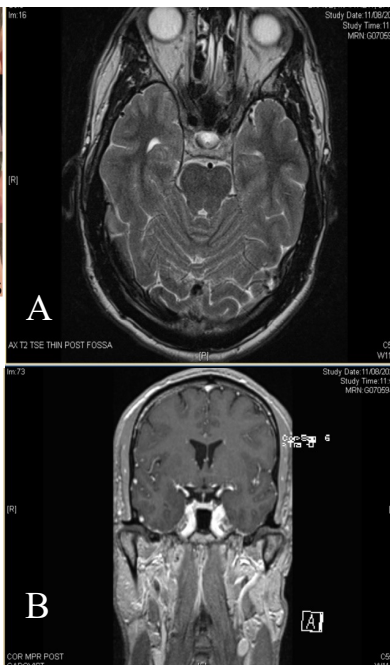
In the setting of viral infections, the underlying pathophysiology of partial oculomotor nerve palsy is thought to be due to microvascular injury. Viral palsies involving the 7<sup>th</sup> or 8<sup>th</sup> cranial nerves are treated with corticosteroids to presumably reduce edema and regain function<sup>9,10</sup>. While the evidence is equivocal, it is often the only tool available. **3 other reports of unilateral oculomotor nerve palsy due to COVID-19 have been treated with corticosteroids:**

1. **Acute onset:** 10-year-old boy infected with COVID-19. Prescribed prednisone 2mg/kg/day for 10 days and complete recovery was achieved in 7 days<sup>6</sup>.
2. **Acute onset:** 46-year-old male infected with COVID-19. Treated with 1 g/day of oral methylprednisolone for 3 days and showed full recovery after two weeks<sup>7</sup>.
3. **Delayed onset:** 60-year-old woman infected with COVID-19. 6 weeks later, she developed diplopia and a fixed, dilated pupil. Treated with oral prednisone 60 mg with a slow taper and eventually showed full recovery after a few weeks<sup>8</sup>.

There are very few cases of COVID-associated oculomotor neuropathies treated with corticosteroids to formulate treatment decisions on given the novelty of the virus. Seeing as corticosteroids are included in the current viral 7<sup>th</sup> and 8<sup>th</sup> cranial neuropathy guidelines<sup>9,10</sup>, they may have played a similar important role in hastening the recovery timeframe. Whether corticosteroids sped the patient’s recovery is unknown. However, seeing as full recovery was attained in all four cases, the role of corticosteroid in reducing local inflammation and restoring significant function in a short time cannot be dismissed.



**Figure 1.** Clinical progression of left oculomotor nerve palsy treated with dexamethasone. NOTE: severe ptosis in the left eyelid and brow, the lack of medial abduction in the affected eye on rightward gaze, and how the affected eye reaches further medially throughout recovery.



**Figure 2.** A) T2-weighted brain MRI in the axial view and B) Gadolinium-enhanced T1-weighted brain MRI in the coronal view. No oculomotor muscular atrophy and is free of any masses, lesions, infarction, hemorrhages, or oculomotor nerve impingement.

## References

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