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Case Report

Idiopathic intracranial hypertension leading to bilateral optic atrophy in a patient with recent COVID-19 infection: a case report

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ABSTRACT

Neurologic complications are common in patients hospitalised with COVID-19 infection. Most common complications are myalgias, headaches, encephalopathy and dizziness. Uncommon complications are stroke, motor and sensory deficits, seizures, ataxia and movement disorders. Multiple neuro-ophthalmological manifestations have also been reported in association with COVID-19. These complications may be the result of a range of pathophysiological mechanisms like hypoxic neuronal injury during active COVID-19 infection, RAS dysfunction, immune dysfunction and direct injury by the virus etc throughout the course of the disease. Here we reported a case of neuro-ophthalmic complication of Idiopathic intracranial hypertension (IIH) followed by bilateral optic atrophy in a middle-aged man with recent COVID-19 infection. He presented to the emergency with complaints of headache, dizziness and sudden painless bilateral diminution of vision for 3 days. His fundus examination was suggestive of bilateral papilledema, his MRI brain was normal and opening pressure of CSF was raised on lumbar puncture. His MRV was normal, there was no evidence of CSVT. He was started on steroids and acetazolamide. His headache improved but there was no improvement in visual acuity. Repeat fundus showed pale disc and MRI orbit was suggestive of bilateral optic atrophy.

Keywords: Idiopathic intracranial hypertension, Optic atrophy, Papilledema

INTRODUCTION

Since December 2019, Coronavirus disease 2019 (COVID-19) has become a global pandemic caused by the highly transmissible Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[1]

A wide variety of neuro-ophthalmologic manifestations have also been found in association with COVID-19, mostly related to demyelinating disease. While the mechanism of these manifestations is unknown, hypotheses include direct neuronal invasion, endothelial cell dysfunction leading to ischemia and coagulopathy, or a widespread inflammatory 'cytokine storm' induced by the virus.[2] Optic neuritis has developed in several infected patients, presenting with neuromyelitis Optica spectrum disorder and anti-Myelin oligodendrocyte glycoprotein (anti-MOG) antibodies.[3]

Patients presented with subacute vision loss, a relative afferent pupillary defect, pain with eye movements, optic disc oedema, and radiographic findings of acute optic neuritis following a COVID-19 infection.[4]

CASE REPORT

A 42-years-old male presented to the emergency department with history of headache and blurry vision for 3 days. He had history of COVID-19 infection 22 days ago for which he was home isolated as he had mild respiratory symptoms. There was no past history of diabetes mellitus, obstructive sleep apnoea, migraine, gastritis, obesity.

There was no significant drug intake history. He complained of headache, not unilateral, continuous, sometimes pulsatile, associated with nausea but no vomiting.

This headache was followed by blurring of vision that worsened gradually over a period of 3 days. It was painless, acute in onset and progressive. Vitals were normal; pulse=78/min, blood pressure of 130/80 mmHg, oxygen saturation of 99%, and respiratory rate of 17 breaths per min.

CNS examination showed no neurodeficit, no cranial nerve palsies. Ophthalmology examination showed bilateral mild papilledema. Keeping in view a possibility of idiopathic intracranial hypertension patient was subjected to an urgent MRI brain and MR venogram. It showed no intracranial abnormality, no evidence of sinus venous thrombosis, no apparent bilateral venous sinus narrowing/stenosis.

Lumbar puncture (LP) revealed an opening pressure of 40 cm H_2O, and 30 ml clear and colourless CSF was drained at this time. CSF analysis was within normal limits. With evidence of increased intracranial pressure by LP findings, acetazolamide 250 mg TID was initiated.

The patient reported improvement in his headache but his visual acuity remained the same. Over the next three days there was no improvement in his vision. Keeping a possibility of post viral/post infectious optic neuritis patient was started on injection dexamethasone 6 mg IV TID.

LP was repeated which revealed CSF opening pressure of 25 cm H_2O. Further imaging of bilateral orbit MRI revealed bilateral optic atrophy. There was no evidence of optic neuritis and no peri-optic enhancement thereby settling the suspicion of anti-MOG syndrome or NMOSD in correlation with his recent COVID-19 infection. Patient has been on follow up for 2 months. There has been no significant improvement in his visual acuity.

He was discharged on acetazolamide and topiramate and supportive drugs for headache prophylaxis; with regular follow-up in our OPD. He showed improvement in his headache but no visual improvement or deterioration.

Our patient's symptoms of headache, optic disk oedema, and high opening pressure on LP made the diagnosis of Idiopathic intracranial hypertension. His MRI brain and MRV were unremarkable. LP showed high opening pressure (40 cm H_2O) and normal CSF analysis, with headache that improved with therapeutic LP, acetazolamide and steroids.

He fulfilled the modified Dandy's criteria for IIH. However as there was no improvement in visual acuity, MRI orbit was done which was suggestive of bilateral optic atrophy.

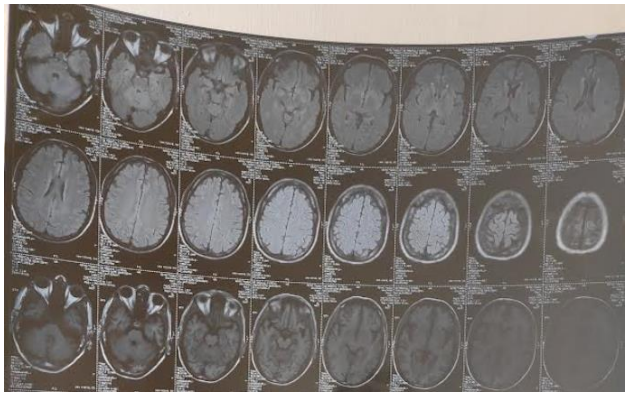

Figure 1: MRI brain normal study.

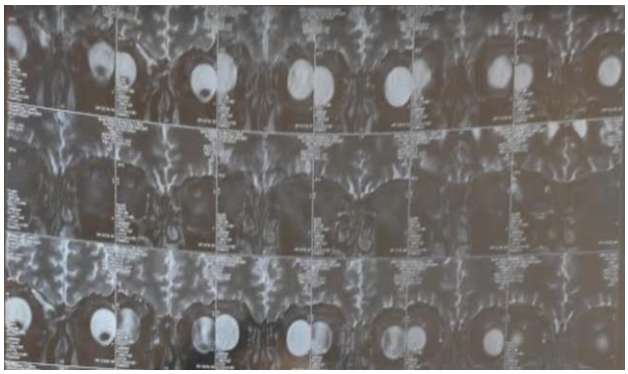

Figure 2: MRI orbit showing bilateral optic nerve atrophy.

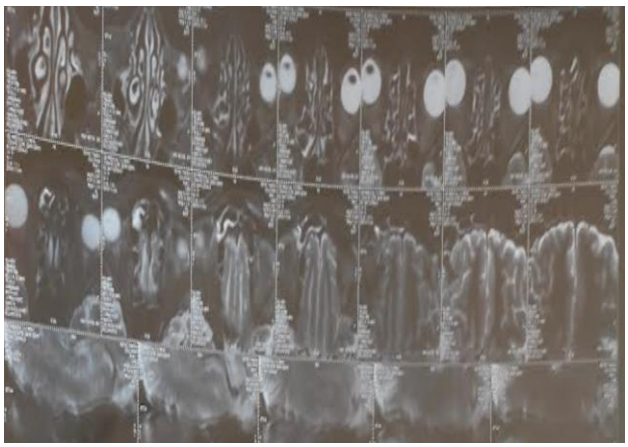

Figure 3: MRI brain showing bilateral optic nerve atrophy.

DISCUSSION

COVID-19 infection caused a horrific pandemic worldwide. Some people experienced flu-like symptoms, while many died due to pulmonary complications. During the first phase of the pandemic, pulmonary symptoms were in the limelight but later other signs and symptoms were also reported. There haven't been many studies which analyse the ophthalmic and neuro-ophthalmic

complications post COVID-19 infection. It is mostly in the form of individual case reports for now.

During the COVID-19 pandemic, 35% of the patients experienced some neurological disorders such as: headache, anosmia, hypogeusia, dizziness, ischaemic stroke, visual deterioration, and Guillain-Barré syndrome. In the background of neurological manifestations, the neurotropism of the COVID virus is suspected. Some of the reported neuro-ophthalmologic manifestations are papillophlebitis, optic neuritis, Adie's tonic pupil, Miller Fisher syndrome, neurogenic ptosis and cerebrovascular accident.

Montalvan in May 2020 published a systematic review explaining the possible neuropathogenicity of the virus. It proposed that there is likely invasion of cerebral circulation endothelium via hematogenous spread of SARS-CoV-2 from systemic circulation and potential viral propagation through the cribriform plate and olfactory bulb.[5]

COVID-19-associated demyelination is also hypothesized to be attributed to the cytokine storm due to IL-1, IL-6, and TNF-α, which then activates the glial cells and thereby causes demyelination.[6] Another possible hypothesis is ascribed to SARS-CoV-2-triggered production of antiglial cell antibodies in the Para infectious or postinfectious state, thereby leading to demyelinating pathologies such as Acute or subacute disseminated encephalomyelitis (ADEM), acute haemorrhagic leukoencephalitis with MRI features of concentric demyelination pattern, acute transverse myelitis, and neuromyelitis optica.

Neuro-ophthalmic manifestations are not common and at present, isolated case reports are all that we have to build a foundation. In March 2021, Sen et al published review of ophthalmic manifestations of COVID-19. The mean age of the patients with neuro-ophthalmic manifestations in the study was 42.3±16.2 (median 43, 6-71) years. Of the 19 cases reported, 13 were males while only seven had systemic comorbidity in the form of hypertension and diabetes. One patient had SLE with advanced Chronic kidney disease (CKD), Chronic obstructive pulmonary disease (COPD). Patients presented with ophthalmic complaints either concurrently or within a few days of onset of systemic symptoms of COVID-19. The median gap from COVID-19 to development of ophthalmic symptoms was 5 (mean 11.3±13.3, 0-42) days.[7]

There have also been cases reported of secondary intracranial hypertension with concurrent COVID-19 infection and MIS-C in paediatric population.[8] Owing to autoantibody production and thrombophilic disorders in COVID-19, physicians must have low threshold to investigate secondary IIH and demyelinating disorders in patients with headache and decreased vision following recent COVID-19 infection.[9] Idiopathic intracranial hypertension (IIH) constitutes a constellation of signs and symptoms of raised intracranial pressure with fulfilment of modified Dandy's criteria.

Physicians must ask leading questions about double vision, decreased vision, pain with eye movements, gait abnormalities, or other neurological conditions while screening patients with COVID-19 symptoms. In patients presenting with these complaints, COVID-19 testing may be prudent while doing the tests to determine aetiology. Treating doctors should also do a quick assessment of visual acuity, pupillary response, ocular motility, ptosis, optic disc, and reflexes since majority of these conditions occur in the early phase of the disease. Neuroimaging with angiography with attention to cranial nerves for any abnormal enhancement or cerebral infarcts can be advised based on the assessment.

CONCLUSION

The pandemic caused by SARS-CoV-2 has had health implications of unparalleled magnitude. The infection can range from asymptomatic to mild to life threatening respiratory distress. It can affect almost every organ of the body. Direct effect due to virus, immune mediated tissue damage, activation of the coagulation cascade and prothrombotic state induced by the viral infection, the associated comorbidities and drugs used in the management are responsible for the findings in the eye. Ophthalmic manifestations may be the presenting feature of COVID-19 infection or they may develop several weeks after recovery. Ophthalmologists and physicians should be aware of the possible associations of ocular diseases with SARS-CoV-2 in order to ask relevant history, look for specific signs, advise appropriate tests and thereby mitigate the spread of infection as well as diagnose and initiate early treatment for life and vision threatening complications. In the COVID-19 pandemic, health systems struggled to prioritize care for affected patients; however, physicians also attempted to maintain care for other less-threatening medical conditions that could have led to permanent disabilities if untreated. IIH is a relatively common condition affecting young females that could lead to permanent blindness if not properly treated. Diagnosis and follow-up of papilledema due to IIH during and after the COVID-19 pandemic can be facilitated by nonmydriatic fundus photography and optical coherence tomography. COVID-19 may mimic IIH by presenting as cerebral venous sinus thrombosis, papillophlebitis, or meningoencephalitis, so a high index of suspicion is required in these cases. When surgical treatment is indicated, optic nerve sheath fenestration is the primary procedure of choice. IIH is a serious vision-threatening condition that could lead to permanent blindness and disability at a relatively young age if left untreated. It could be the first presentation of a COVID-19 infection. Certain precautions if taken during the diagnosis and management of this condition, may allow appropriate care to be delivered to these patients while minimizing the risk of COVID-19 infection.

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