Ocular Manifestations Of COVID-19

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ABSTRACT
The severe acute respiratory syndrome coronavirus 2 pandemic has swamped the entire world. It can target any organ of the body, and symptoms can vary from none or mild to even dreadful life-threatening respiratory distress or multisystem organ failure. A conglomeration of ocular symptoms and signs keeps outpouring from various parts of the world, ranging from conjunctivitis, and uveitis to sight-threatening retinal vascular occlusions and calamitous rhino-orbital mucormycosis. Ocular manifestations can be attributed to either direct invasion by the virus, immune-mediated insult of the ocular tissues, or triggering of coagulation cascade inducing prothrombotic state. This article aims to briefly delineate the ocular manifestation of COVID-19, which will be instrumental in early diagnosis, management, and timely vision restoration.

KEYWORDS: Pandemic, Conjunctivitis, Vascular Occlusion.

The COVID-19 pandemic has deluged the world. This outbreak commenced in December 2019 in Wuhan city of Hubei Province, and on March 11, 2020, it was declared a global pandemic. The most prominent whistle-blower of COVID-19, Li Wenliang, was himself an ophthalmologist who contracted COVID-19 from an asymptomatic patient suffering from acute angle-closure glaucoma. However, Li, unfortunately, succumbed to this dreadful virus. The worldwide research on vaccines and treatment continues targeting SARS-CoV-2 structural proteins; however, the mutations in genes for these proteins remain a challenge.

The virus can target any body organ with symptoms ranging from asymptomatic and mild to life-threatening respiratory distress. The ocular manifestations prevalence ranges from 2-32%. The virus binds to the angiotensin-converting enzyme 2 receptors in the respiratory mucosa, gastrointestinal tract, and kidney. These receptors are also present on the cornea, limbus, conjunctiva, endothelial cells of vessels, immune cells, and neurons. The binding is mediated by the lactoferrin present in tears which prevents attachment of the virus to heparan sulfate proteoglycans which helps in its subsequent binding to the ACE-2 receptor. It then undergoes cleavage by protein transmembrane serine protease 2 (TMPRSS2) [1]. It induces virulence by direct cellular invasion and activation of various inflammatory mediators, including cytokines. The ocular involvement is suggested either through direct inoculation from respiratory droplets or aerosolized viral particles, migration from the nasopharynx via the nasolacrimal duct, or even hematogenous spread through the lacrimal gland [1,2].

The prevalence of eyelid, ocular surface, and anterior segment manifestations range from 0.81% to 34.5%. These include follicular conjunctivitis, viral keratoconjunctivitis, hemorrhagic, pseudomembranous conjunctivitis, episcleritis, and blepharitis. The ocular manifestations were associated with higher white blood cell (WBC) count, acute phase reactants, C-reactive protein (CRP), procalcitonin, lactate dehydrogenase (LDH) levels, and increased neutrophils/lymphocyte ratio.

Posterior segment manifestations include central retinal artery and vein occlusion, which is attributed to a pro-coagulant state with increased D-dimer, prothrombin time, activated partial thromboplastin time, fibrinogen, and cytokines. There may be vitritis, acute retinal necrosis, and reactivation of serpiginous choroiditis due to autoimmune activation by SARS-CoV-2 [1-4].

The neuro-ophthalmological manifestations of optic neuritis, cranial nerve palsies, late-onset myasthenia gravis, and neurogenic ptosis are attributed due to misdirected immune system. Miller Fisher Syndrome (MFS), consisting of ophthalmoplegia, ataxia, and loss of tendon reflexes, has also been reported to occur. Patients with MFS, neurogenic ptosis, and ocular myasthenia improved with intravenous immunoglobulins, suggesting immune-mediated pathogenesis in all these entities. There was also an isolated case report on a patient with Adie's tonic pupil, which responded well to oral steroids suggesting a role of autoimmunity. There may be cerebrovascular insults with vision loss or gaze palsies due to infarcts caused by the pro-coagulant state in these patients.

The orbital manifestations include acute dacryoadenitis, which is immunological inflammation with the virus spreading through lacrimal ducts or direct hematogenous, superior ophthalmic vein thrombosis, orbital cellulitis, and rhino-

orbital-cerebral mucormycosis in patients predisposed by uncontrolled diabetes mellitus, immunosuppression, iron overload, elevated deferoxamine, hematological malignancies, neutropenia. The fungal orbital invasion is through thin lamina papyracea of the ethmoid bone, infratemporal fossa, inferior orbital fissure, or orbital apex. The intracranial extension is through the cribriform plate of the ethmoid, supraorbital fissure, and perineural invasion. The lethal outcomes include cavernous sinus thrombosis, sagittal sinus thrombosis, carotid occlusion, cerebral infarction, intracranial aneurysm/hemorrhage, and cerebral abscesses [5].

The management of patients with these manifestations requires thorough history and examination with details about the onset and duration of symptoms, pain, redness, diminution of vision or sudden loss of vision, restriction of ocular movements, and protrusion of the eyeball. The detailed ocular examination must involve an assessment of visual acuity, pupillary reactions, intraocular pressure, extraocular movement in 9 gazes, color vision, and visual field. Anterior segment evaluation with slit lamp examination and thorough posterior segment evaluation after pupillary dilatation with indirect ophthalmoscopy is imperative.

CONCLUSION

This pandemic is likely to remain longer despite widespread research as innumerable mutations in SARS-CoV-2 pose a grim concern. Therefore, all due precautions must be followed by everyone. The ophthalmologists and eye care providers must be acquainted with various ocular manifestations of COVID-19 for early diagnosis and treatment required to restore vision.

CONFLICT OF INTEREST

None.

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