Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Diplopia due to acquired Brown syndrome after COVID-19 infection

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Coronavirus disease 2019 (COVID-19) is a multisystem, inflammatory condition usually presenting with respiratory symptoms, such as fever, shortness of breath, and severe cough. It may also present with ocular, neurological, and musculoskeletal manifestations. However, since the emergence of the disease in 2019, only a few cases with ocular involvement have been reported in the literature. We present a case of acquired Brown syndrome secondary to COVID-19.

Case Report

A 31-year-old woman, an intensive care unit nurse, was admitted to the ophthalmology service, Ankara University, School of Medicine, with a complaint of double vision and pain in up-and-left gaze for 4 days. No history of systemic or ocular disease was noted other than dyschromatopsia. However, 3 weeks prior to admission, she tested positive for COVID-19 on real-time polymerase chain reaction testing. Complete blood count analysis showed a white blood cell count of 3340/mm^3, indicating leukopenia. Laboratory blood analysis revealed that ferritin, fibrinogen, D-dimer, erythrocyte sedimentation rate, and C-reactive protein levels were within normal limits. The patient received combined treatment of favipiravir, enoxaparine, and dexamethasone as a standardized therapy for COVID-19. Prior to admission, however, only acetylsalicylic acid (81 mg/day) had been given to the patient.

On examination, visual acuity was 20/20 in each eye. Biomicroscopic and fundus findings were within normal limits, and intraocular pressure was 15 mm Hg in each eye. The patient was orthophoric in primary position but had a right hypotropia of 5$^\Delta$ in up-and-left gaze and a right hypotropia of 2$^\Delta$-3$^\Delta$ with right head tilt. The patient had limited elevation in adduction (Figure 1A). Standard forced duction testing[1] revealed minimal restriction of elevation in adduction. In addition, the patient experienced pain on palpation over the trochlea. Orbital magnetic resonance imaging (MRI) revealed thickening and contrast enhancement in the distal part of superior oblique muscle–tendon-trochlea complex (Figure 2A–D).

The patient complained of headache and was evaluated by the neurologist at our institution. She underwent laboratory tests and cranial MRI. Rheumatoid factor, antinuclear antibody, antinuclear cytoplasmic antibody, and anti–double stranded DNA were all negative. MRI revealed nonspecific hyperintensities in the periventricular white matter of the parietal lobes, indicating vasculitis (Figure 2E–F). The patient received 1 gm of intravenous methylprednisolone per day for 3 days, followed by 64 mg of oral methylprednisolone per day, tapering 16 mg every 2 days. Because the patient did not have diplopia in primary position, no additional ocular treatment was required. By 2 months' follow-up, diplopia had resolved. There was mild limitation to elevation in adduction (Figure 1B); repeat MRI of the orbits showed resolution of the previously observed hyperintense thickening of the trochlea–superior oblique complex (Figure 2G–H).

Discussion

Ocular motility disorders associated with oculomotor nerve dysfunction in patients with COVID-19 have been reported previously.[2,3] We present a case of myositis/trochleitis that manifested with diplopia and limited elevation in adduction, consistent with a Brown syndrome secondary to COVID-19.

COVID-19 is known to affect muscles, tendons, and cartilage, and cases of myositis involving paraspinal, facial, bulbar, proximal limb, and obturator muscles

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FIG 1. A, Ocular motility examination at day 1 of admission showing limitation of adduction in upgaze in the right eye, 3 weeks after COVID-19 diagnosis. B, Improved elevation deficiency in adduction at 2 months' follow-up.

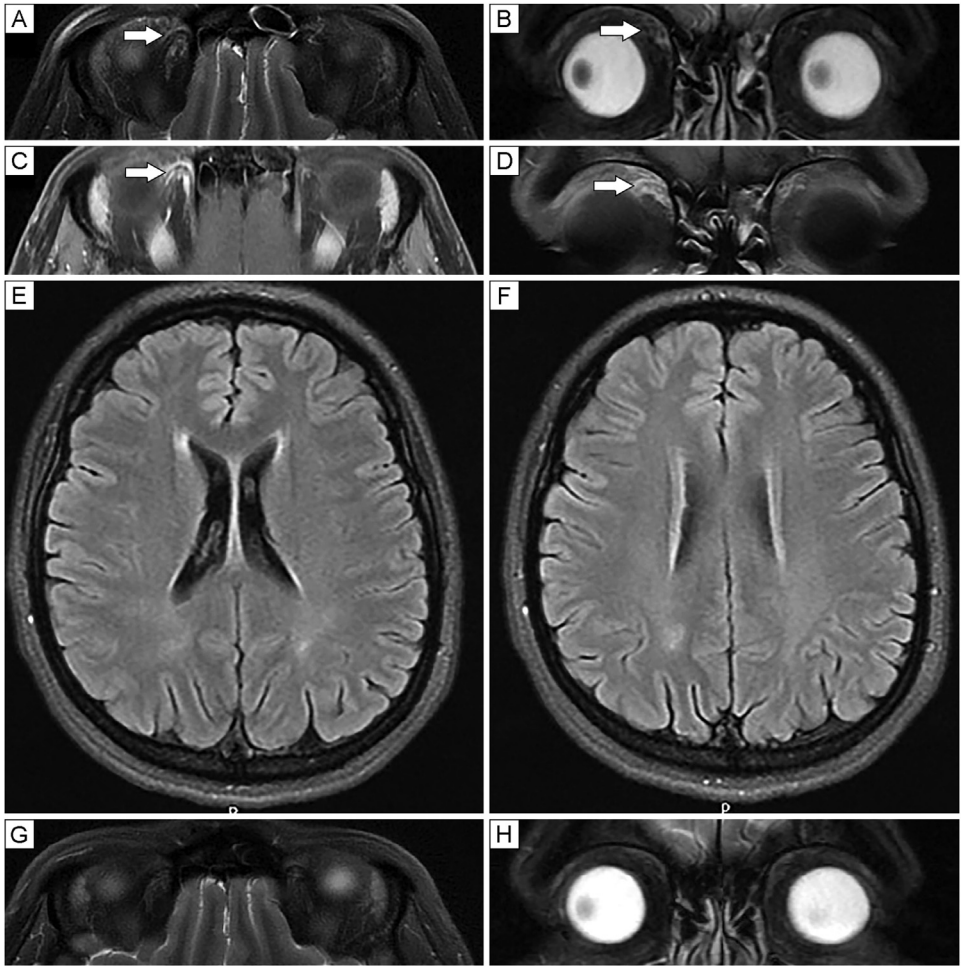

FIG 2. Cranio-orbital magnetic resonance imaging at day 1 of admission (A-F) and at 2 months' follow-up (G-H). Axial (A) and coronal (B) T2-weighted images demonstrate hyperintensity and thickening at the trochlea and anterior part of the superior oblique muscle, with edema in the right orbit. Axial (C) and coronal (D) post-contrast T1-weighted images with fat suppression showing abnormal contrast enhancement at the trochlea and anterior part of the superior oblique muscle in the right orbit (arrow). Axial flair images (E-F) of the brain demonstrate nonspecific hyperintensities in the periventricular white matter of the parietal lobes. Axial T2-weighted (G), and coronal T2-weighted (H) images show complete resolution of the previous high-signal intensity and thickening of the trochlea and anterior part of the superior oblique muscle in the right orbit.

caused by SARS- CoV-2 have been reported.[4-6] In addition, arthritis, enthesitis, and dactylitis associated with SARS-CoV-2 infection have also been reported.[7,8] The exact mechanism of musculoskeletal system involvement in patients with COVID-19 remains unclear, however. Various viral pathogens, such as hepatitis C, human immunodeficiency virus, Middle East respiratory syndrome coronavirus, and influenza A and B are known to cause myositis. Furthermore, SARS-CoV-2 has a high affinity for angiotensin-converting enzyme 2 (ACE2), which skeletal muscles express.[9] Thus, direct viral invasion of skeletal muscles by SARS-CoV-2 is possible.

Another mechanism of musculoskeletal system involvement may be the immune-mediated pathway. Viral pathogens are known to cause immune-mediated myositis by triggering or exacerbating autoimmunity.[10] In addition, many inflammatory cells are stimulated, and cytokines are released in patients with COVID-19. This inflammatory response may trigger immune-mediated pathways or may be myotoxic.

In the present case, acquired Brown syndrome was associated with trochlear tendon complex involvement because orbital MRI revealed thickening and enhancement in the distal part of the superior oblique muscle–tendon-trochlea complex. In addition, the onset of ocular findings 3 weeks after COVID-19 infection suggests that this condition may be a reactive response rather than a direct viral invasion.

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Noncongenital juvenile-onset bilateral lamellar cataract in 1p36 deletion syndrome

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We report the case of a 16-year-old girl with 1p36 deletion syndrome, who experienced visual loss in both eyes for 2 months because of lamellar cataracts. Mutations on some 1p36 genes in both experimental models and humans may be associated with cataract. This is the first detailed description of acquired juvenile-onset bilateral cataract with 1p36 deletion.

Monosomy 1p36, affecting 1 in 5,000 newborns, represents the most common terminal deletion syndrome.[1] Deletions, 95% of which are de novo, occur equally in males and females and in all ethnic groups. It is difficult to identify the genes responsible for the phenotypic manifestations, because the distal end of the short arm of the chromosome 1 is very rich in genes.[2] There is no common breakpoint or deletion size in 1p36 monosomy.[1-3] Affected individuals are phenotypically heterogeneous, because the size of the deletion varies widely. The features are not specific for this syndrome, and the cytogenetic identification of the deletion is often difficult. Therefore, some individuals may be misdiagnosed.[3] The systemic findings are summarized in Table 1.[1,2,4] Ocular malformations or functional visual problems are present in more than half the cases. They include strabismus (35%-67%), hyperopia (41%-67%), myopia (17%-40%), astigmatism (23%), nystagmus (13%-23%), unilateral cataract (5.9%), retinal albinism (5.9%), and unilateral optic nerve coloboma (2.9%).[1-4]

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