OBSERVATION

Bilateral Purtscher-Like Retinopathy Associated With COVID-19 Infection

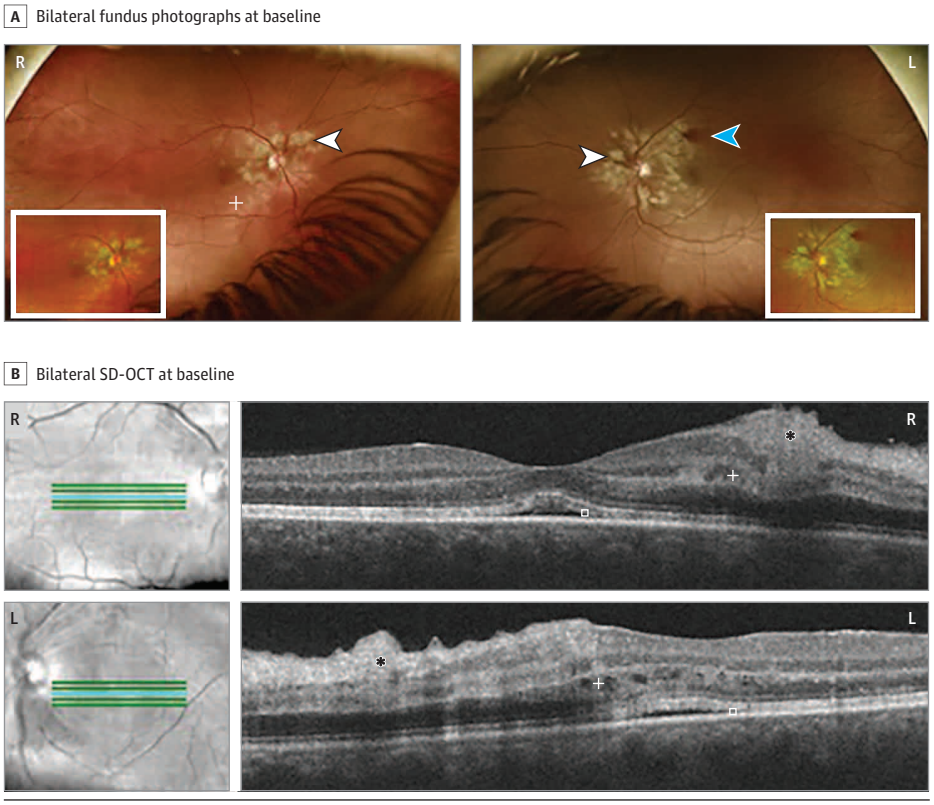
Purtscher retinopathy is an occlusive microvasculopathy related to trauma first described by Otmar Purtscher.[1] When the condition occurs in the absence of trauma it is referred to as *Purtscher-like retinopathy* and has been described in conditions such as acute pancreatitis, fat embolism syndrome, kidney failure, and childbirth.[1] Both conditions present with sudden unilateral or bilateral vision loss and nerve fiber layer infarcts, optic disc swelling, retinal hemorrhages, and pathognomonic *Purtscher flecken* (a term describing discrete areas of retinal whitening between the arterioles and venules) on fundus examination.[1] The retinopathy typically spontaneously resolves within 1 to 3 months.[1]

Report of a Case | Here we describe a case of a 37-year-old female individual who presented with sudden-onset blurred vision in both eyes 4 days after testing positive for COVID-19. She reported mild symptoms of COVID-19 infection that did not require hospitalization, such as malaise, headaches, fever, and loss of taste and smell, but she did not endorse a cough. Infection was confirmed with a series of 3 positive rapid antigen test results for SARS-CoV-2. Prior to infection, she had received 2 doses of the messenger RNA-based Pfizer-BioNTech vaccine. Her ocular history was remarkable for a bilateral YAG peripheral iridotomy in 2019 for high hyperopia.

At initial presentation, visual acuity with habitual correction measured 20/150 OD and 20/80 OS. Her intraocular pressures were 17 mm Hg OD and 15 mm Hg OS. Anterior segment examination showed a relative afferent pupillary defect on the left. Dilated fundus examination revealed nerve fiber layer infarcts, intraretinal hemorrhages, and Purtscher flecken bilaterally (**Figure 1**A). Spectral-domain optical coherence tomography revealed thickening and hyperreflectivity of the nerve fiber layer and areas of subretinal fluid and intraretinal edema bilaterally (Figure 1B). No additional testing for thrombophilia (such as platelet levels, prothrombin time, or partial thromboplastin time) or complement levels was performed. Based on the clinical findings, the patient was diagnosed with Purtscher-like retinopathy and was counseled on the self-limiting nature of the condition.

One month later, visual acuity with habitual correction improved to 20/30 OD and 20/50 OS and pinhole improvement to 20/30. Fundus examination showed considerable

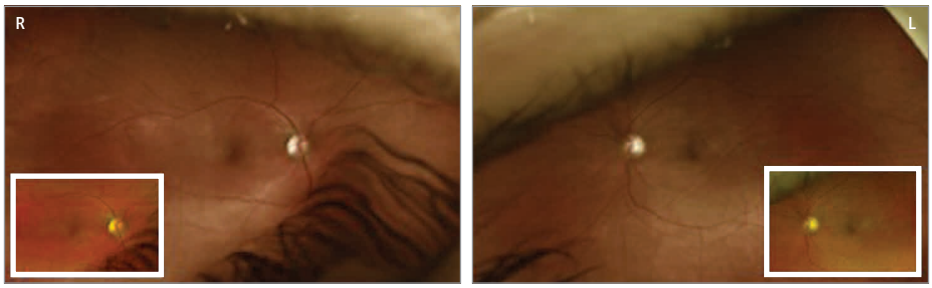
Figure 1. Initial Presentation of Bilateral Purtscher-Like Retinopathy in a 37-Year-Old Female Individual



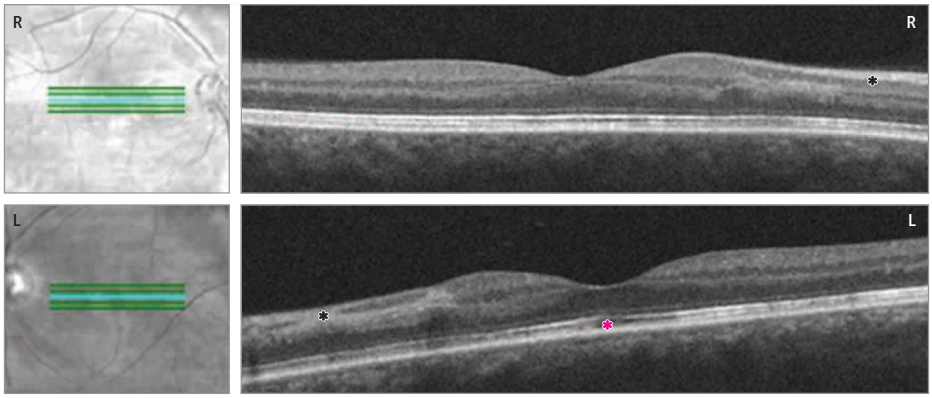
A, Bilateral fundus photographs showing nerve fiber layer infarcts (plus sign), intraretinal hemorrhages (blue arrowhead), and Purtscher flecken (white arrowhead).
B, Spectral-domain optical coherence tomography (SD-OCT) showing thickening and hyperreflectivity of the nerve fiber layer (asterisk), areas of subretinal fluid (square), and intraretinal edema (plus sign).

Figure 2. 1-Month Follow-up of Bilateral Purtscher-Like Retinopathy in a 37-Year-Old Female Individual

A Bilateral fundus photographs at 1-mo follow-up



B Bilateral SD-OCT at 1-mo follow-up



A, Fundus photographs showing considerable improvement to the nerve fiber layer infarcts, intraretinal hemorrhages, and Purtscher flecken bilaterally. B, Spectral-domain optical coherence tomography (SD-OCT) showing improvement of the nerve fiber layer thickening (black asterisk) and intraretinal edema, with only a small amount of subretinal fluid (pink asterisk) in the left eye.

improvement to the nerve fiber layer infarcts, intraretinal hemorrhages, and Purtscher flecken bilaterally (**Figure 2**A). Spectral-domain optical coherence tomography revealed considerable improvement to the nerve fiber layer thickening and intraretinal edema, with a persistent small amount of subretinal fluid (Figure 2B).

Discussion | The pathogenesis of Purtscher and Purtscher-like retinopathy remains debated and largely unknown. The most widely recognized theory is that the condition results from embolic occlusion of the precapillary arterioles. Alternatively, a complement-mediated hypothesis is that complement activation induces the formation of leukocyte aggregates that lead to transient arteriolar occlusion.[1] It has been shown that COVID-19 infection is associated with thromboinflammation, endothelial injury, and complement activation, all likely contributing to the current presentation.[2] Similar to the debate on pathophysiology, the approach to treatment is controversial. Although the condition is generally managed with observation alone, there have been cases of treatment with high-dose steroids, papaverine hydrochloride, and hyperbaric oxygen, although there is no evidence to show superiority of these modalities over observation.[1]

To our knowledge, this is one of only a few cases reported of Purtscher-like retinopathy secondary to COVID-19 infection. Similar presentations have been described in patients with severe COVID-19 infections requiring hospitalization and intubation[3,4] and 1 case[5] has been reported with mild symptoms like this patient. Although the temporal association of this finding with the COVID-19 infection suggests a cause-and-effect relationship, one cannot determine with certainty that the findings were due to COVID-19, or some other confounding event associated with the infection.

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Conflict of Interest Disclosures: None reported.

1. Agrawal A, McKibbin MA. Purtscher's and Purtscher-like retinopathies: a review. *Surv Ophthalmol*. 2006;51(2):129-136. doi:10.1016/j.survophthal.2005.12.003

2. Wang X, Sahu KK, Cerny J. Coagulopathy, endothelial dysfunction, thrombotic microangiopathy and complement activation: potential role of complement system inhibition in COVID-19. *J Thromb Thrombolysis*. 2021;51(3):657-662. doi:10.1007/s11239-020-02297-z

3. Bottini AR, Steinmetz S, Blinder KJ, Shah GK. Purtscher-like retinopathy in a patient with COVID-19. *Case Rep Ophthalmol Med*. 2021;2021:6661541. doi:10.1155/2021/6661541

4. Rahman EZ, Shah P, Ong JE, Goldberg M, Ong SS. Purtscher-like retinopathy in a patient with COVID-19 and disseminated intravascular coagulation. *Am J Ophthalmol Case Rep*. 2021;24:101229. doi:10.1016/j.ajoc.2021.101229

5. Matilde R, Alberto P, Fabio G, et al. Multitarget microangiopathy in a young healthy man with COVID-19 disease: a case report. *Indian J Ophthalmol*. 2022; 70(2):673-676. doi:10.4103/ijo.IJO_1422_21

CORRECTION

Error in Spelling of Nonauthor Collaborator Name: The Original Investigation titled "Changes in Retinal Sensitivity Associated With Cotoretigene Toliparvovec in X-Linked Retinitis Pigmentosa With *RPGR* Gene Variations,"[1] published online February 9, 2023, was corrected to fix the spelling of the first nonauthor collaborator's name on PubMed. This article was corrected online.

1. von Krusenstiern L, Liu J, Liao E, et al; XIRIUS Part 1 Study Group; XOLARIS Study Group. Change in retinal sensitivity associated with cotoretigene toliparvovec in x-linked retinitis pigmentosa with *RPGR* gene variations. *JAMA Ophthalmol*. Published online February 9, 2023. doi:10.1001/jamaophthalmol.2022.6254