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Purtscher-Like Retinopathy After Acute Ischemic Colitis

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Abstract

Purpose: To describe a case of Purtscher retinopathy, a rare microvascular process hypothesized to have an occlusive etiology. **Methods:** A single case was evaluated. **Results:** A 72-year-old White woman presented with acute, painless bilateral vision loss and severe abdominal pain. She was hospitalized for ischemic colitis of unknown origin. Although her symptoms were improving, funduscopic examination findings of retinal hemorrhages, cotton-wool spots, and Purtscher flecken prompted a referral to ophthalmology. The diagnosis of Purtscher-like retinopathy was made, and treatment of her precipitating ischemic colitis resolved her visual symptoms. **Conclusions:** Purtscher-like retinopathy is a rare clinical diagnosis of exclusion. By describing a previously unreported etiology, this case may improve our understanding of the pathogenesis, management, and treatment of this rare retinopathy.

Keywords

ischemic colitis, Purtscher retinopathy, retinal microvascular disease, systemic conditions of the eye

Introduction

Purtscher retinopathy is a rare, occlusive microvasculopathy associated with cranial or thoracic trauma. In atraumatic cases, it is termed *Purtscher-like retinopathy*.[1] Both pathologies, estimated to have an incidence of 0.24 persons per million, are underreported.[2] Understanding their pathophysiology sheds light on the diverse etiologies contributing to them.

Retinal microvascular occlusion from inciting trauma or inflammation is hypothesized to cause complement system activation, leukocyte aggregation, and clot formation.[3] This results in arteriolar precapillary occlusion and retinal nerve fiber layer infarction.[2,4,5] The etiology of Purtscher-like retinopathy therefore includes various inflammatory and vasculopathic stressors. Symptoms typically manifest as painless vision loss 24 to 48 hours after the precipitating event, often accompanied by central, paracentral, or arcuate scotomata.[1,6] The diagnosis is confirmed clinically with cotton-wool spots, retinal hemorrhages, and Purtscher flecken on funduscopy.[1] Here, we present a rare case of Purtscher-like retinopathy after ischemic colitis.

Case Report

A 72-year-old White woman with no significant medical history developed intractable abdominal pain and rectal bleeding. The patient had normal blood pressure at presentation and no significant vascular risk factors. Computed tomography (CT) of the abdomen and pelvis showed circumferential thickening and abnormal enhancement of the splenic flexure and descending colon to the juncture of the sigmoid colon (Figure 1).

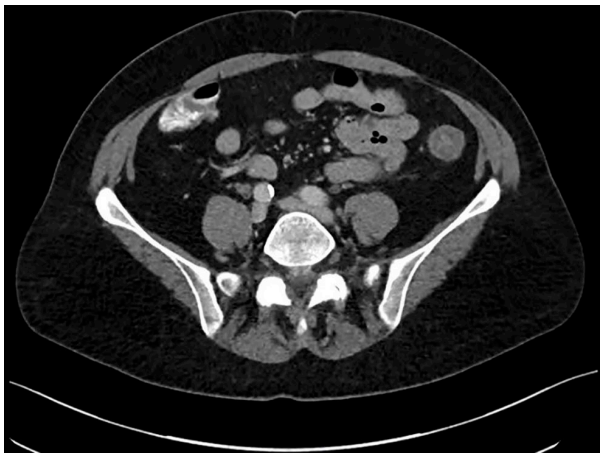

Figure 1. Computed tomography (CT) of the abdomen and pelvis in the axial plane shows circumferential thickening and abnormal enhancement involving the distal splenic flexure extending to the rectosigmoid and not involving the sigmoid colon, with mural thickening up to 0.9 cm.

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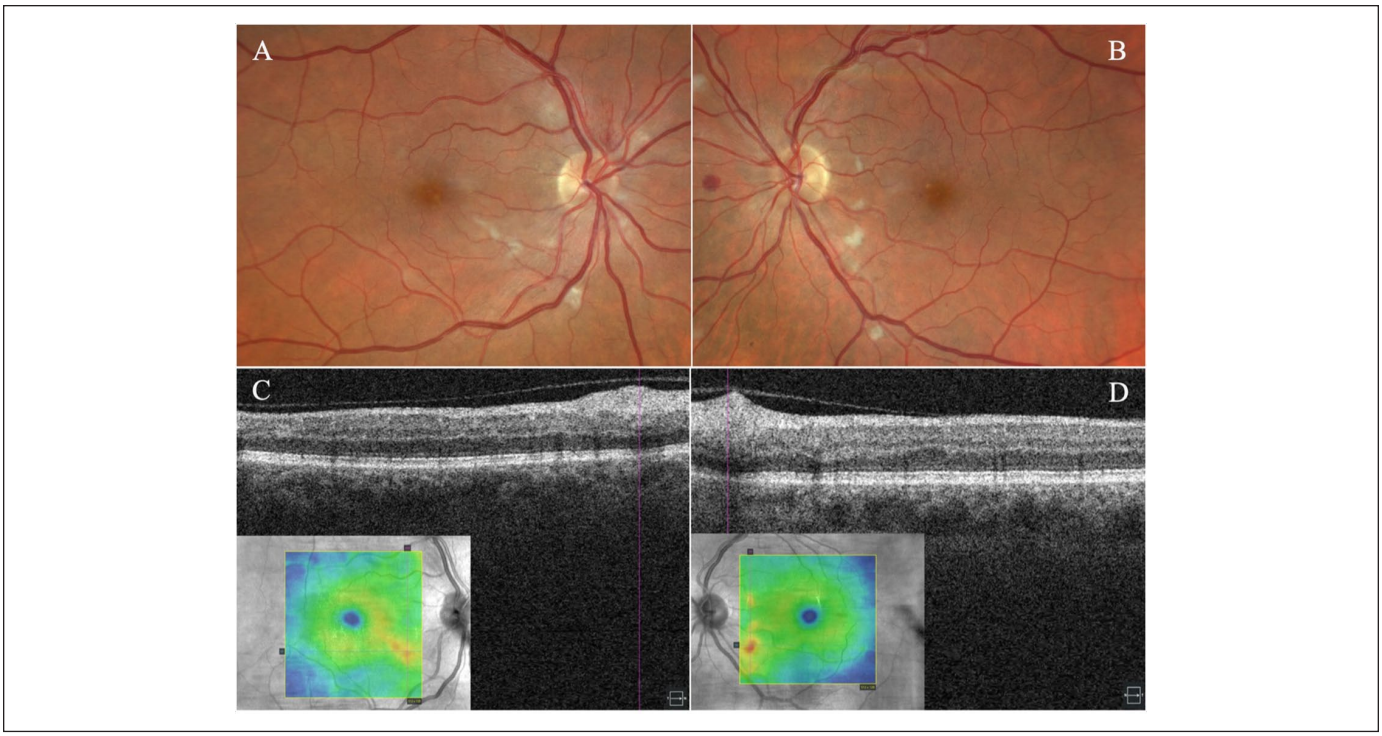

Figure 2. Diagnostic testing performed at presentation. (A) Color fundus photography of the right eye shows a Purtscher flecken along the inferior arcade, superior disc hemorrhage, and scattered peripapillary cotton-wool spots. (B) Color fundus photography of the left eye shows a Purtscher flecken along the inferior arcade, scattered peripapillary cotton-wool spots, and a nasal intraretinal hemorrhage. (C) Optical coherence tomography (OCT) of the macula of the right eye shows thickening of the peripapillary inner retinal layers associated with cotton-wool spots. (D) OCT of the macula of the left eye shows thickening of the peripapillary inner retinal layers associated with cotton-wool spots.

A colonoscopy showed diffusely friable, inflamed, and ulcerated mucosal tissue with contact bleeding from the sigmoid colon to the splenic flexure, with biopsy confirming ischemic colitis as the most likely etiology. The patient was treated with short courses of oral metronidazole, ciprofloxacin, and acetaminophen. At approximately day 1 of hospitalization, she developed bilateral blurry vision and floaters and was referred to outpatient ophthalmology.

At the eye clinic 1 week later, the patient's gastrointestinal symptoms had resolved and visual symptoms were improving. The visual acuity (VA) was 20/30 (20/50 pinhole) OD and 20/20 (20/40 pinhole) OS. Confrontational visual fields and extraocular movements were full. Anterior segment examinations of both eyes were unremarkable and without anterior chamber or vitreous cells. A dilated fundus examination of the right eye showed sharp optic disc margins, a superior disc hemorrhage, scattered peripapillary cotton-wool spots and Purtscher flecken, and few macular drusen. The examination of the left eye found sharp optic disc margins, scattered peripapillary cotton-wool spots and Purtscher flecken, a nasal intraretinal hemorrhage, and few macular drusen (Figure 2, A and B). Optical coherence tomography (OCT) of the maculas showed bilateral scattered areas of focal nerve fiber layer thickening corresponding to cotton-wool spots, with no intraretinal or subretinal fluid (Figure 2, C and D). OCT of the nerve fiber layer showed subtle inferonasal and nasal thickening of the optic nerve heads (average thickness, 120 μm OD; 110 μm OS). The complete blood count, comprehensive metabolic panel, erythrocyte sedimentation rate, C-reactive protein, and CT of the head were unremarkable.

At the patient's 2-week follow-up, visual symptoms continued to improve. The VA was 20/25 OD and 20/25 OS. Dilated fundus examinations showed resolution of a superior disc hemorrhage in the right eye and an improved nasal intraretinal hemorrhage in the left eye. However, a new inferotemporal intraretinal hemorrhage was seen in the left eye that was previously masked by a cotton-wool spot. Retinal ischemic lesions were found in both eyes (Figure 3, A and B). Fluorescein angiography (FA) of the right eye revealed no gross abnormalities; focal hypofluorescence inferotemporally consistent with known intraretinal hemorrhage blocking was seen in the left eye (Figure 3, C and D). The patient was lost to follow-up after the 2-week visit.

Conclusions

Purtscher-like retinopathy is a rare retinal vasculopathy and is associated with trauma or systemic disease secondary to the occlusion of retinal arterioles from leukoembolization, endothelial damage, and complement activation. Commonly associated

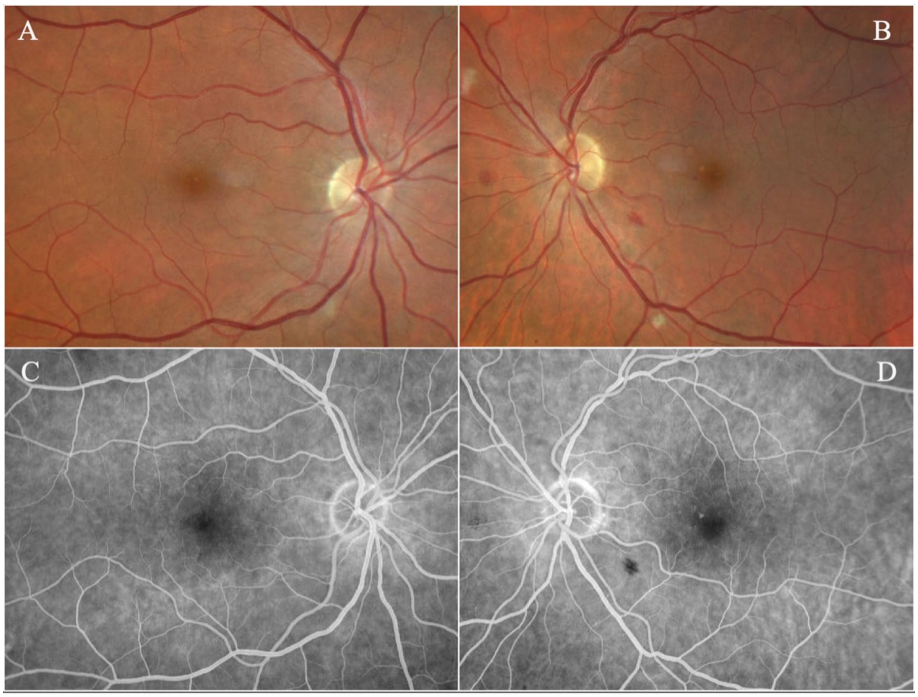

Figure 3. Diagnostic testing performed at the 2-week follow-up. (A) Color fundus photography of the right eye shows interval improvement in Purtscher flecken along the inferior arcade and near total resolution of superior disc hemorrhage and scattered peripapillary cotton-wool spots. (B) Color fundus photography of the left eye shows interval improvement in the Purtscher flecken along the inferior arcade, the resolution of scattered peripapillary cotton-wool spots with the appearance of previously masked intraretinal hemorrhage inferotemporal to the optic disc, and an improvement in the nasal intraretinal hemorrhage. (C) Fluorescein angiography (FA) of the right eye shows no gross abnormalities. (D) FA of the left eye shows a focal area of hypofluorescence inferotemporally, consistent with blocking from the previously described intraretinal hemorrhage in an otherwise unremarkable study.

systemic causes of Purtscher-like retinopathy include acute pancreatitis, pancreatic adenocarcinoma, renal failure, lymphoproliferative disorders, and complications related to childbirth, among others.[7–9] To our knowledge, this is the first case report of Purtscher-like retinopathy secondary to ischemic colitis.

In the setting of ischemic colitis of unknown origin, the development of Purtscher-like retinopathy in this patient was most likely caused by a leukoembolic process from an inflammatory insult. However, cholesterol, fibrin-platelet, calcium, leukocytic aggregates, air, fat, and amniotic fluid emboli are all thought to be plausible sources of micro-occlusive events in this condition. Purtscher-like retinopathy is a diagnosis of exclusion; thus, other etiologies of cotton-wool spots and intraretinal hemorrhages (eg, ischemic, inflammatory, infectious, embolic, neoplastic, pharmacologic) must be investigated and ruled out.[7,10] Our patient's medical history and ocular history before presentation were unremarkable, her blood pressure was normotensive, and a further laboratory workup and neuroimaging were unremarkable.

Purtscher-like retinopathy manifests with sudden onset of a painless decrease in vision. Dilated fundus examination findings, typically observed in the posterior pole and immediate nasal peripapillary retina, include cotton-wool spots (93%), retinal hemorrhages (65%), and Purtscher flecken (63%),[1] which are pathognomonic of the disease and described as polygonal areas of whitening in the inner retina with a characteristic clear zone between the ischemic retina and arteriole. These lesions are differentiated from cotton-wool spots, which are ill-defined lesions located superficial to the retinal vessels, potentially obscuring larger vasculature.[4,11] In this case, color fundus photographs showed both Purtscher flecken and cotton-wool spots. Several lesions along the superior and inferior arcades had sharp borders adjacent to the vasculature, representing Purtscher flecken (Figure 2, A and B).

To accurately diagnose Purtscher-like retinopathy, a systematic review in 2013 by Miguel et al[1] proposed that 3 of the 5 following criteria should be present: Purtscher flecken, low-to-moderate number (1-10) of retinal hemorrhages, cotton-wool spots (typically restricted to the posterior pole), a probable or plausible explanatory etiology, and a complementary investigation compatible with the diagnosis. OCT of the macula may show the inner retinal layers' hyperreflectivity, macular edema, outer retinal atrophy, and photoreceptor loss.[12,13] FA features are variable but include choroidal hypofluorescence, macular ischemia, capillary nonperfusion, or optic disc leakage.[13] In this patient, OCT of the maculas showed scattered hyperreflectivity

of the inner retinal layers, while FA was grossly unremarkable (Figure 2, C and D).

At present, there is no accepted treatment for Purtscher-like retinopathy because the condition is thought to improve with removal of the precipitating event within 1 to 3 months after onset.[14] Xia et al[15] found no difference in vision improvement to glucocorticoid therapy in Purtscher-like retinopathy. The visual prognosis varies by the etiology of disease, but tends to improve within the first 2 months in most cases.[1] In our patient, no ophthalmic treatment was necessary because her condition improved with the resolution of ischemic colitis on oral antibiotics alone.

Purtscher-like retinopathy is a rare micro-occlusive vasculopathy presenting with sudden-onset vision loss after traumatic or inflammatory events. It is diagnosed clinically based on characteristic features once other etiologies have been excluded. Ischemic colitis may be considered a probable or plausible explanatory etiology in the known list of precipitating events for this poorly understood condition.

Ethical Approval

This study adhered to the tenets of the Declaration of Helsinki. Ethical approval was not required.

Statement of Informed Consent

Verbal informed consent including permission for publication of all photographs and images included herein was obtained from legally authorized representatives before the study.

Declaration of Conflicting Interests

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