

AN UNUSUAL CASE OF UNILATERAL PURTSCHERS RETINOPATHY



We saw a rare presentation of vision loss in preeclampsia—but only in one eye.

BY AARON S. CAMPEAS, BSC, AND BOLES LAV KOTLYAR, MD

In 1910, Otmar Purtscher published an article describing a patient with bilateral vision loss after head trauma.¹ On examination, the patient had retinal hemorrhages and whitening in the posterior pole in each eye. This chorio-retinopathy associated with trauma or systemic injury—which has findings of cotton-wool spots, retinal hemorrhage, optic edema, and areas of retinal ischemia—is now called Purtschers retinopathy. Etiologies of this condition include pancreatitis, fat embolism, air embolism, amniotic fluid embolism, HELLP syndrome, trauma, long bone fracture, preeclampsia, and eclampsia.² Despite the many potential causes, Purtschers retinopathy is extremely rare, with one study finding an incidence of only 0.24 cases per million per year in the United Kingdom and Ireland.³

We report the case of a woman with preeclampsia who experienced acute unilateral vision loss secondary to Purtschers retinopathy. Our literature review using PubMed and Scopus to search “preeclampsia Purtscher retinopathy” found no other cases of unilateral visual involvement of Purtschers retinopathy in patients with preeclampsia.

CASE REPORT

A 23-year-old Black woman who was 27 weeks pregnant presented to the ophthalmologist with decreased vision in her right eye for 3 days. Past medical history was significant for anemia, managed with iron and prenatal supplements. On examination, BVCA was 20/250 OD and 20/20 OS. No afferent pupillary defects were present. Anterior segment examination and IOP were unremarkable in each eye. On fundoscopy, the left eye was unremarkable. Fundoscopy of the right eye revealed dilated veins with scattered inferior peripapillary flame-shaped hemorrhages, cotton-wool spots, and Purtscher flecken (Figure 1). OCT showed retinal thickening, subretinal fluid, and nerve fiber layer edema (Figure 2). Fluorescein angiography was deferred due to pregnancy, and her blood pressure was 174/122. Given these findings, a diagnosis of Purtschers retinopathy was made, likely due to preeclampsia.

Due to the presumptive diagnosis of preeclampsia, the

patient was urgently referred to her obstetrician. The patient did not follow up with the obstetrician as recommended but presented to the emergency department 1 week later. She had a blood pressure of 168/94 and 4+ proteinuria. She was diagnosed with preeclampsia, a Cesarean section was performed 2 days later, and two healthy babies were delivered.

Eleven weeks postpartum, the patient’s VA was counting fingers OD. Fundus examination showed inferior optic nerve pallor. There was normalization of the retinal vascular caliber. The subretinal fluid and macular edema had almost resolved. Fluorescein angiography showed normal transit time with hypofluorescence in a wedge-shaped area inferotemporally and peripheral nonperfusion (Figure 3). Fine lacy collateral vessels were seen adjacent to areas of nonperfusion. No late leakage was present centrally. The patient’s findings were consistent with postinflammatory optic atrophy and sequelae of retinal ischemia due to Purtschers retinopathy. As of publication, visual acuity remained counting fingers OD.

DISCUSSION

Preeclampsia is a systemic vascular disorder characterized by hypertension, end organ damage, endothelial dysfunction, and hypercoagulability. Preeclampsia occurs in 3% to 5% of pregnancies, and visual changes can be seen in 25% of severe cases. Common visual complaints include blurred vision, photopsia, visual field defects, and blindness. Vision loss can be caused by serous retinal detachment, focal necrosis of retinal epithelial cells, cortical blindness, central retinal vein occlusions, and Purtschers retinopathy.⁴

Purtschers retinopathy is an arteriolar microvascular disease characterized by occlusion of the 45 μ m-diameter peripapillary arterioles that supply the superficial retinal capillaries. Patients typically present with acute vision loss. Common fundoscopic findings are cotton-wool spots (93% of cases), retinal hemorrhages (65%), and Purtscher flecken (63%). The diagnosis of Purtschers retinopathy should be suspected in cases with any of these three signs.⁵

One study recorded data at the 1- and 6-month marks for patients (24 total eyes) with Purtschers retinopathy. At

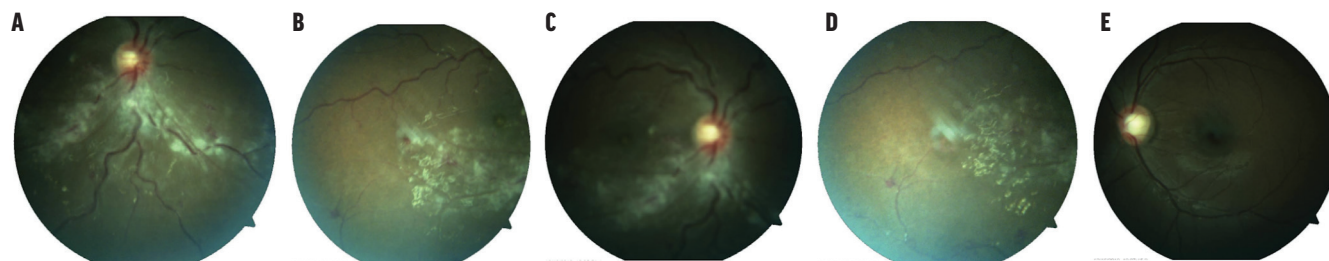


Figure 1. Fundoscopic imaging of the right eye revealed dilated veins (A), cotton-wool spots (B), hemorrhages (C), and Purtscher flecken (D). The left eye was unaffected (E).

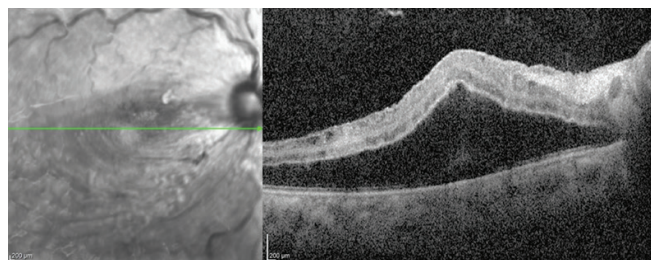


Figure 2. OCT showed retinal thickening consistent with macular edema, subretinal fluid, and nerve fiber layer edema.

1 month, 74% of eyes still had acute retinopathy findings of hemorrhages, cotton-wool spots, or Purtscher flecken. At 6 months, 100% of acute retinal findings had disappeared. In addition, 50% of eyes had VA improvements by 2 lines and 23% by 4 lines, with a mean improvement of 2.7 lines. Final VA remained 20/200 or less in 11 of 24 eyes.³ Another review found that 10 of 25 patients had a final VA of 20/200 or less in at least one eye at their final vision assessment.⁶

Treatment for Purtscher's retinopathy is typically limited to the treatment of the underlying condition (in our case, the condition of preeclamptic pregnancy) and waiting for symptom resolution. Isolated case reports have shown visual improvements after administering a 3-day course of 250 mg intravenous methylprednisolone four times a day. A proposed mechanism of improved outcomes with high-dose corticosteroids is the stabilization of damaged neuronal membranes to allow for healing.² One case report detailed a patient with Purtscher's retinopathy who improved from 20/800 to 20/50 at 1 week post methylprednisolone.⁷

Another report detailed two eyes with Purtscher's retinopathy treated with methylprednisolone. Only one improved by 2 or more lines, and neither eye showed improvement greater than 4 lines.³ Given the small quantity of data and varying results, corticosteroids are not recommended.

The unilateral involvement of our patient is perhaps a result of anatomic arteriolar vascular differences between the right and left eyes. Flow studies suggest Purtscher's retinopathy may be the result of the interplay between the angle of bifurcation, flow volume, capillary wall stress and levels of endothelin peptide, prostacyclin, nitric oxide, and local autoregulation.⁸ In our case, the increased flow rate due to preeclampsia-induced hypertension may have led to changes in wall stress. Differences in the angles of bifurcation



Figure 3. Fluorescein angiography displays hypofluorescence in a wedge-shaped area

in the microvasculature between the left and right eyes may have led to different degrees of wall stress, thus resulting in unilateral involvement.

CONCLUSION

Delayed diagnosis and intervention with preeclampsia can result in permanent damage to end organs, including the eyes. Our case emphasizes the need to aggressively treat these patients, often by expedited delivery. In the absence of effective treatments for acute occlusive retinal vascular disease, prevention through vigilance and managing systemic risk factors are our best available approaches to preventing vision loss. ■

1. Purtscher O. Noch unbekannte befunde nach schadeltrauma. *Ber Dtsch Ophthalmol Ges.* 1910;36:294-301.
2. Tripathy K, Patel BC. Purtscher Retinopathy. In: StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2020. Updated August 11, 2020.
3. Agrawal A, McKibbin M. Purtscher's retinopathy: epidemiology, clinical features and outcome. *Br J Ophthalmol.* 2007;91(11):1456-1459.
4. Abu Samra K. The eye and visual system in the preeclampsia/eclampsia syndrome: what to expect? *Saudi J Ophthalmol.* 2013;27(1):51-53.
5. Miguel AIM, Henriques F, Azevedo LFR, Loureiro AJR, Maberly DAL. Systematic review of Purtscher's and Purtscher-like retinopathies. *Eye (Lond).* 2013;27(1):1-13.
6. Agrawal A, McKibbin MA. Purtscher's and Purtscher-like retinopathies: a review. *Surv Ophthalmol.* 2006;51:129-36.
7. Atabay C, Kansu T, Nurlu G. Late visual recovery after intravenous methylprednisolone treatment of Purtscher's retinopathy. *Annals Ophthalmol.* 1993;25:330-333.
8. Harrison TJ, Abbasi CO, Khraishi TA. Purtscher retinopathy: an alternative etiology supported by computer fluid dynamic simulations. *Invest Ophthalmol Vis Sci.* 2011;52(11):8102-8107.

AARON S. CAMPEAS, BSC

- Student, Touro College of Osteopathic Medicine, New York, New York
- acampeas@student.touro.edu
- Financial disclosure: None

BOLES LAV KOTLYAR, MD

- Retinal Surgeon, Vitreous Retina Macula Specialist of New Jersey, Millburn, New Jersey
- Retinal Surgeon, Eyecare Partners, Millburn, New Jersey
- boleslav.kotlyar@gmail.com
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