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Purtscher-like retinopathy following a bowel movement

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ABSTRACT

Purpose: To describe a case of Purtscher-like retinopathy that developed after a bowel movement.
Observations: A 32-year-old male presented with blurry vision and bilateral temporal paracentral scotomas that developed immediately after standing up from a bowel movement. Fundoscopic examination was notable for bilateral cotton wool spots in the nasal macula. Optical coherence tomography showed bilateral intraretinal fluid, subfoveal fluid, and scattered areas of inner retinal hyperreflectivity and thickening corresponding to the areas of cotton wool spots on examination. No treatment was administered and the patient had significant improvement in symptoms 2 days later with resolution of macular edema.
Conclusions: Here we report a case of Purtscher-like retinopathy after a bowel movement. Although the exact mechanism of Purtscher-like retinopathy is unknown, there are multiple reports of Purtscher-like retinopathy after extreme events involving Valsalva, such as during weightlifting, and we postulate that this presentation is likely of similar pathophysiology.

1. Introduction

Purtscher's and Purtscher-like retinopathy are occlusive microvasculopathies caused by traumatic or non-traumatic causes, respectively. The pathophysiology of Purtscher's and Purtscher-like retinopathy is unknown but is thought to be due to vascular occlusion of precapillary arterioles, which predominantly affects the posterior pole and inner retinal layers. The traumatic causes of Purtscher's retinopathy are classically cranial trauma or thoracic compression.[1] There are many reported etiologies of Purtscher-like retinopathy, including pancreatitis, renal failure, connective tissue disorders, embolism (fat, air, amniotic fluid), thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, and hematologic malignancies.[2] Additionally, there are multiple reports of Purtscher-like retinopathy developing after extreme Valsalva, such as weightlifting, and reports of vision loss after a bowel movement, such as from Valsalva retinopathy or acute retinal venous dilation.[3–6] Here we present a case of Purtscher-like retinopathy following a bowel movement, which to our knowledge has not been previously reported in the literature.

2. Case report

A 32-year-old male with a history of asthma, migraine, gastroesophageal reflux disease, LASIK surgery, and laminectomy for spinal stenosis 3 weeks prior, presented with acute onset of blurry vision in both eyes. The symptoms began immediately upon standing up after passing a bowel movement on the evening of presentation. The bowel movement was not unusual and there was not excessive straining. The patient initially noticed a large white cloud obstructing his vision that resolved after 10 minutes. This was followed by blurry vision and a subjective inability to focus. The patient denied flashes, floaters, headache, lightheadedness, numbness, dizziness, eye pain, or trauma.

On examination the patient's Snellen visual acuity was 20/125 OD and 20/80 OS. Pupillary exam, extraocular movements, color vision, and intraocular pressure was normal. The anterior segment was notable for a flat LASIK flap OU. On dilated fundus examination, there were small juxtapapillary and macular areas of inner retinal whitening consistent with cotton wool spots in both eyes (Fig. 1). The optic nerves were sharp without edema. Automated perimetry was notable for bilateral temporal paracentral scotomas. Optical coherence tomography (OCT) showed bilateral intraretinal macular fluid in the outer plexiform layer, bilateral subfoveal fluid, and bilateral scattered areas of inner retinal hyperreflectivity and thickening corresponding to the areas of retinal whitening seen on fundoscopic examination (Fig. 2). Fluorescein angiography showed early patchy punctate staining with late leakage nasal to the nerve and in the juxtapapillary macula in both eyes (Fig. 3).

The patient's presentation was thought to be due to Purtscher-like retinopathy and the patient was sent for an expedited systemic work-

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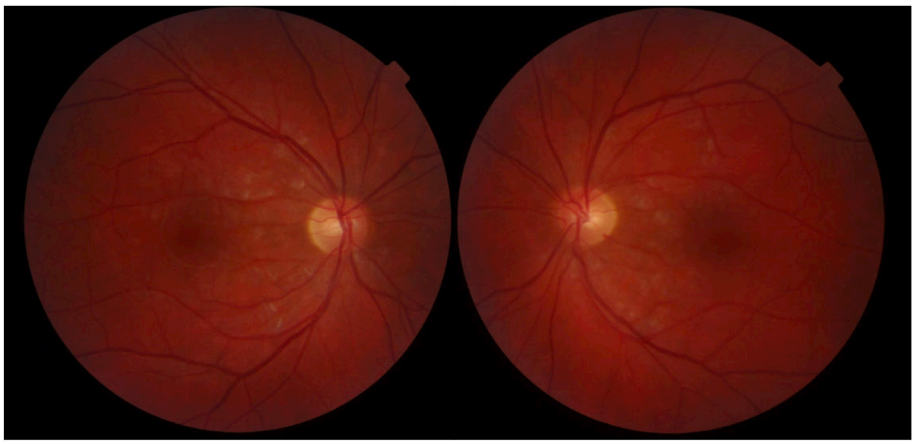

Fig. 1. Findings on presentation. Color fundus photograph of both eyes demonstrating juxtapapillary and macular areas of inner retinal whitening consistent with cotton wool spots.

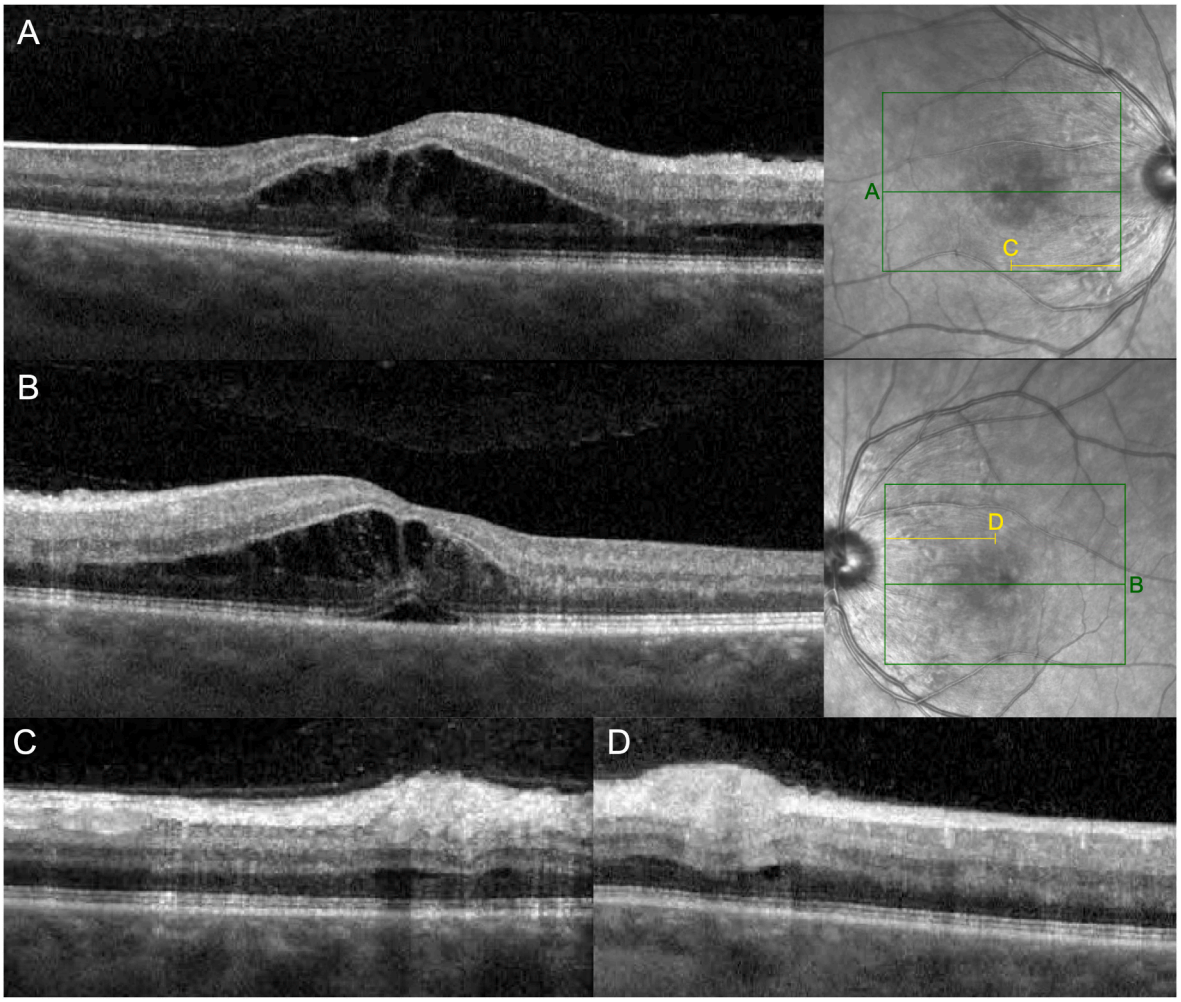

Fig. 2. Findings on presentation. Optical coherence tomography of both eyes showing bilateral intraretinal macular fluid in the outer plexiform layer (A,B), bilateral subfoveal fluid (A,B), and bilateral scattered areas of inner retinal hyperreflectivity and thickening (C,D).

up. The patient underwent brain MRI without contrast and MRA head and neck without contrast that revealed no acute intracranial abnormality and no significant vessel stenosis. Extensive laboratory testing was notable for normal serum chemistries, normal creatinine, normal complete blood count, mildly elevated alkaline phosphatase of 133 U/L, mildly elevated alanine aminotransferase (ALT) of 71 U/L, normal aspartate aminotransferase (AST), normal lipase, normal erythrocyte sedimentation rate (ESR), and an elevated C-reactive protein (CRP) of 37.5 mg/L. Coagulation studies, autoimmune testing (antinuclear antibody, anti-double-stranded DNA, ANCA, lysozyme), and an infectious work-up (QuantiFERON, RPR, FTA-Abs, Lyme antibodies, Chest X-ray) were normal. Transthoracic echocardiogram was notable for normal

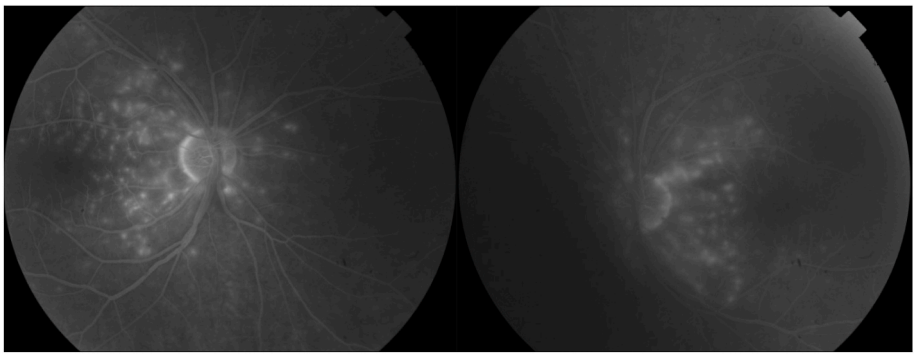

Fig. 3. Findings on presentation. Late-phase fluorescein angiography of both eyes showing patchy staining with leakage in the nasal and juxtapapillary macula in both eyes.

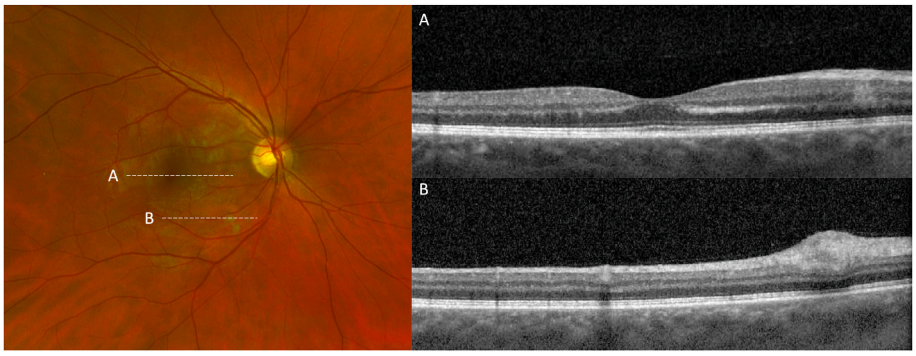

Fig. 4. Findings on follow-up 2 days after presentation. Pseudocolor fundus photograph of right eye continues to demonstrate juxtapapillary and macular cotton wool spots. Corresponding optical coherence tomography lines show resolution of intraretinal and subretinal fluid (A) and persistent foci of inner retinal hyperreflectivity and thickening (B). Similar findings were present in the left eye.

systolic function, normal valvular function, and a likely small patent foramen ovale (PFO). Rheumatology was consulted and concluded that the presentation was not concerning for a systemic inflammatory disorder. Neurology and Cardiology were also consulted and there was low suspicion for an embolic cause to his presentation despite his small PFO.

The patient was observed without treatment and follow-up 2 days later revealed improvement in visual acuity to 20/32 OD and 20/40 OS with resolution of macular edema (Fig. 4A). The patient continued to have scotomas temporal to fixation OU that corresponded to areas of cotton wool spots in the nasal macula OU (Fig. 4B). Observation was continued and 1 month later the patient endorsed 90% improvement in his visual symptoms and had decreases in cotton wool spots on examination.

3. Discussion

Here we present a case of Purtscher-like retinopathy that developed after a bowel movement. Although the exact mechanism of Purtscher-like retinopathy is unknown, it is thought to be due to occlusion of precapillary retinal arterioles.[2] Hypothesized causes include embolic phenomena (fat, air, leukocytes, fibrin, platelets, complement activation), vasculitis induced by free fatty acids, and raised intrathoracic pressure leading to venous dilation and impaired venous return.[7] There are reports of Purtscher-like retinopathy developing after extreme Valsalva, such as during weightlifting or when catching a heavy falling object, but none after relatively mild straining, such as during a bowel movement as in this case.[3,4] We suspect that during a bowel movement there is increased intrathoracic pressure which leads to decreased venous return and increased pressure in the retinal vasculature. This transient increased venous pressure may directly alter retinal hemodynamics and to lead retinal ischemia or cause endothelial injury leading to microangiopathy via thrombosis, complement activation, or leukocyte aggregation.[3,7] It is also possible that vessel engorgement or endothelial injury leads to vasospasm that results in retinal arteriolar ischemia. The possibility of fat embolism from the patient's recent spinal laminectomy as an etiology was also entertained; however, this was considered less likely given that the surgery was 3 weeks prior to presentation, symptom onset was immediately after a bowel movement, and a detailed systemic work-up was overall unremarkable. The significance of the patient's PFO was also considered in the pathogenesis. When a PFO is present, Valsalva leads to increased right to left cardiac shunting which could allow microemboli to pass into the arterial circulation and lead to Purtscher-like retinopathy from a distant embolic cause.[8] Neurology and cardiology were consulted during this patient's work-up and felt that this was less likely as there was no known embolic source, no other sequelae from embolic disease, and the PFO was small. In cases where the cause or inciting factor of Purtscher-like retinopathy is unclear, a broad systemic work-up is warranted given that various life-threatening pathologies can be the cause, such as pancreatitis, pancreatic adenocarcinoma, embolism, blood dyscrasias, and renal failure.[2]

Clinically, Purtscher-like retinopathy usually presents with bilateral decreased vision that can range from mild loss to hand motion acuity. Patients may also have visual field deficits including central, paracentral, or arcuate scotomata, but peripheral vision is usually preserved.[7] The most common clinical findings in Purtscher-like retinopathy are cotton wool spots, retinal hemorrhages, and Purtscher flecken, which are pathognomonic for the disease and are discrete polygonal areas of inner retinal whitening between arterioles and venules that usually have an area of clearing adjacent to arterioles. In the early phases of the disease, OCT findings include macular edema, inner retinal hyperreflectivity that correspond to cotton wool spots, and

deeper areas of hyperreflectivity that correspond to Purtscher flecken. Fluorescein angiography can show areas of retinal capillary non-perfusion, retinal ischemia, slower filling of retinal vessels, late leakage from retinal vessels, and peripapillary staining.[2]

There are no proven treatments for Purtscher's or Purtscher-like retinopathy beyond supportive care and the management of any underlying systemic causes. Corticosteroids have been used for the condition with variable results; a 2013 review of thirty-five eyes treated with corticosteroids versus no corticosteroid showed no difference in visual acuity at 1, 3, or 6 months.[2] Anti-vascular endothelial growth factor agents have been shown to be effective for associated macular edema.[9] Other attempted treatments include indomethacin, eculizumab, and hyperbaric oxygen, which have shown some success in various case reports.[3,10,11] The visual prognosis of Purtscher's and Purtscher-like retinopathy is variable and depends on the severity of disease at presentation, with approximately 50% of cases having spontaneous visual improvement of greater than 2 Snellen lines.[12]

4. Conclusions

This is a case of Purtscher-like retinopathy following a bowel movement. Although the exact mechanism of Purtscher-like retinopathy is unknown, there are multiple reports of Purtscher-like retinopathy after extreme events of Valsalva and we postulate that this presentation is likely of similar pathophysiology. In cases where the cause or inciting factor of Purtscher-like retinopathy is unclear, like in this case, a broad systemic work-up is warranted as life threating pathologies can be present.

Patient consent

Consent to publish this report was not obtained as this report contains no personal identifying information.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

None.

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