Purtscher’s-like retinopathy associated with acute pancreatitis

Greggory M. Gahn, Arshad M. Khanani, Majid Khan, Aamir A. Aziz, Fawwaz A. Siddiqui, Nikolas JS. London, Lekha K. Mukkamala, Lawrence S. Morse

1. Report of a case

A 22-year-old man with a history of heavy alcohol use was referred to the ophthalmology clinic due to sudden onset loss of central vision. He was recently discharged from a local hospital following seven days of treatment for acute pancreatitis and alcohol withdrawal. At the time of initial presentation, the patient was only oriented to person and was not able to answer questions appropriately. Initial laboratory testing revealed leukocytosis, thrombocytopenia, elevated AST, and elevated ALT. Abdominal ultrasound showed mildly enlarged pancreas and increased liver echogenicity. Chest X-ray was normal. On the third day of his hospitalization, the patient’s mentation had improved and he began to complain of bilateral visual loss, which he described as “black spots” in his central vision. He denied any improvement in his central vision throughout the remainder of his hospital stay and was discharged following the resolution of his alcohol withdrawal and pancreatitis.

The patient was seen in the ophthalmology clinic eight days following the loss of central vision. In the clinic, he reported intact peripheral vision that he describes as looking “purple with yellow flashes”. He also noted pain in the occipital region of his head and in both eyes when looking to the right or left. Visual acuity was reduced to counting fingers in both eyes. Slit lamp examination and intraocular pressures were normal. Fundus examination revealed diffuse bilateral macular edema and retinal hemorrhages due to macular ischemia. Optical coherence tomography (OCT) showed diffuse bilateral intra-retinal edema with hyper-reflectivity consistent with hemorrhage. Optical coherence tomography angiography (OCTA) was also performed and demonstrated lack of detectable flow in the macula of both eyes.

The findings were consistent with a diagnosis of Purtscher-like retinopathy. The patient received bilateral intravitreal injections of bevacizumab 1.25mg and sub-tenons triamcinolone acetone 40mg with a second injection of triamcinolone acetone 40mg being given three months later. Three months following the initial presentation, the patient’s visual acuity improved to 20/400 bilaterally from counting fingers. Fundoscopic examination at this time revealed prominent, but improved, inner retinal whitening bilaterally. OCTA images demonstrated persistent bilateral macular ischemia.

2. Discussion

The association between acute pancreatitis and Purtscher-like retinopathy was first documented by Inkeles and Walsh in 1975 when they described three cases of the characteristic retinal appearance in patients with acute pancreatitis. The pathogenesis of Purtscher-like retinopathy is believed to be the result of leukoembolization, which results in arterial
Fig. 1. Initial presentation true-color fundus and Fluorescein Angiogram imaging of the right and left eyes. A,B: Pre-treatment true-color fundus photo of the right (A) and left (B) eyes, respectively, showing retinal whitening, cotton-wool spots, pre-retinal and intra-retinal hemorrhages. C,D: Fluorescein Angiogram of the right (C) and left (D) eyes, respectively, showing blocking due to pre-retinal hemorrhages and hypofluorescence due to macular ischemia.

Fig. 2. At presentation enhanced-depth imaging spectral-domain optical coherence tomography (SD-OCT) and OCT-angiography (OCTA) of the right and left eyes. A, B: SD-OCT b-scans of the right (A) and left (B) maculas, respectively, showing intra-retinal edema and pre-retinal hemorrhage. C,D: Macular OCTA imaging of the superficial and deep vascular networks in the right (A) and left (B) eyes, respectively, showing microvascular occlusion.
blockage and subsequent ischemia of the microvascular bed; however, another possible source of emboli is fat embolization following enzymatic digestion in the pancreas, leading to elevations in serum lipase.\textsuperscript{4,5} Once in the precapillary arteriolar vasculature, the fat emboli induce complement activation which promotes leukocytic aggregation that can become large enough to occlude retinal vessels.\textsuperscript{6}

In the presented case, the patient’s altered mentation secondary to alcohol withdrawal makes it difficult to identify the exact onset of visual symptoms; however, studies examining Purtscher-like retinopathy have demonstrated that retinal alterations appear within the first week from the onset of the associated condition. In these studies, spontaneous visual recovery of at least two Snellen lines occurred in half of all cases.\textsuperscript{3}

The best treatment for Purtscher-like retinopathy remains unclear. A systematic review did not find statistically significant differences in the improvement of visual acuity when comparing treatment with steroids to observation.\textsuperscript{7} Despite this, a limited number of case reports have documented mixed results with the use of intravitreal steroids.\textsuperscript{4,8,9} Clinical improvement with high-dose corticosteroids may be attributed to the stabilization of damaged neuronal membranes that allows for partial recovery of fibers that have not experienced irreversible damage. Steroids also provide the additional benefit of inhibiting further complement activation and granulocyte aggregation.\textsuperscript{10} Further investigation with prospective trials are needed in order to establish the efficacy of steroids in the treatment of Purtscher-like retinopathy.

3. Conclusions

Purtscher-like retinopathy is a rare but serious potential complication of acute pancreatitis. Diagnosis is made by ophthalmoscopy with visualization of the characteristic retinal appearance. The eye condition has a guarded prognosis with no evidence-based treatment for this retinopathy.

References

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