Charmine Trajano,

Vodka, Epigastric Pain and Patchy Vision

Abstract
A 46 y/o white female, admitted to the hospital for alcohol related acute pancreatitis, presents to the eye clinic reporting blur OU & scotoma OD. Comprehensive eye exam reveals ophthalmic manifestations consistent with Purtscher’s retinopathy.

I Case Hx
A 46 y/o white female admitted to the hospital for alcohol related acute pancreatitis presented to the eye clinic complaining of increasing blurry vision OU & a central gray scotoma OD for 2 days after admission. Ocular hx was unremarkable. Medical hx is significant for HTN, Barrett’s esophagus, hypothyroidism, DVT, cholecystectomy, anxiety, depression, & alcohol dependence. Medications included: Buspirone, Levothyroxine, Lisinopril, Omeprazole, Thiamine, Folic Acid, & Hydromorphone.

II Findings
Acute abdominal pain with elevated amylase & lipase enzymes confirmed the systemic dx. VA’s sc are 20/20- OU. Pupils, cover test, motility, color vision & confrontation fields are normal OU. Amsler grid revealed scattered scotomas OU. HVF reveals small relative paracentral defects OU without neurologic field cuts. DFE revealed multiple peripapillary & macular cotton wool spots (CWS) OU, polygonal areas of retinal whitening with clear zones adjacent to retinal arterioles & 1 flame heme OS. Spectralis OCT volume scan revealed intra-retinal thickening OU & macular edema OS>OD.

III DDx
Purtscher’s retinopathy, CRAO, hypertensive retinopathy, commotio retinae, radiation retinopathy, interferon retinopathy & HIV retinopathy.

IV Dx/Discussion
Purtscher’s retinopathy is an ischemic retinopathy typically presenting after severe head/chest trauma. It can also be associated with long bone fracture, embolization (fat, air, or amniotic fluid) or a variety of systemic conditions including: acute pancreatitis, pancreatic carcinoma, multiple myeloma, lymphoma, & connective tissue diseases. It is typically bilateral. Clinical features are typically restricted to the posterior pole & include: CWS, Purtscher flecken, retinal hemes, optic disc swelling & RPE mottling. The presence of Purtscher flecken is unique to Purtscher's retinopathy. These are polygonal-shaped focal areas of retinal whitening within the superficial inner retina between arterioles & venules. They are restricted to the peripapillary retina & posterior pole with characteristic clear zones adjacent to the large diameter arterioles. This clear zone corresponds to the capillary free area adjacent to the retinal vessels, suggesting that primary pathology of the flecken involves the pre-capillary arterioles. Purtscher’s retinopathy is proposed to be caused by intermediate-sized emboli occluding the microcirculation. Pancreatic inflammation releases proteases that activate the complement cascade which can cause coagulation & leukoembolization of retinal arterioles & subsequent retinal ischemia.

V Tx/Management
There is no proven tx for Purtscher’s retinopathy. There have been reports of tx with IV steroids, papaverine, & hyperbaric oxygen, but no tx has proven superior to observation alone. Management is based on treating the underlying condition. Case reports have shown that the visual outcome is variable & unpredictable. Resolution is typically seen 1-3 months after initial presentation.

VI Conclusion
Purtscher’s retinopathy is presumed to be caused by microvascular occlusion of small arterioles by microparticles generated by a variety of conditions. The most commonly associated condition is trauma, followed by acute pancreatitis. The pathophysiology involves activation of complement by proteases released by the inflamed pancreas causing leukoembolization of retinal arterioles. Fundus findings of CWS & hemorrhages are nonspecific, but the presence of Purtscher flecken is pathognomonic this type of retinopathy. There is no proven tx & management is based on treating the underlying systemic condition. It
is important for optometrists to recognize the signs of Purtscher’s in patients to address the underlying condition to prevent further vision loss.

Residency Affiliation: New England College of Optometry
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