



A Rare Case of Purtscher Like Retinopathy Associated with Pancreatitis

Authors

Dr G Pavan, Dr Priya M. Sinhasan, Dr Vijay N

Corresponding Author

Dr Priya Mallikarjun Sinhasan

Department of Ophthalmology, Room No 128, Vijayanagar Institute of Medical Sciences Ballari

Email: sinhasan.priya@gmail.com, 9986326822

Abstract

A case of 35-year-old patient with acute alcoholic pancreatitis who developed sudden loss of visual acuity is described. The ophthalmoscopic examination revealed diffuse retinal whitening of the posterior pole with confluent cotton-wool spots. Fluorescein angiography showed retinal arteriolar occlusion. The findings were compatible with Purtscher-like retinopathy. The case presented here provides an illustration of a rarely recognized complication of acute pancreatitis that is well documented in the ophthalmological literature but not commonly recognized by physicians of other specialties. Purtscher-like retinopathy with bilateral loss of vision is a rare and severe complication that may follow acute pancreatitis, outcome depends upon resolution of the pancreatic disease. Steroids help in early visual recovery.

Keywords; Purtscher like retinopathy, Pancreatitis, Retina, Flecken, Cotton wool spots, Steroids.

Introduction

Purtscher's retinopathy is an occlusive microvasculopathy associated with trauma. It was first described in 1910 by Otmar Purtscher¹. When there is a non-traumatic etiology, the correct designation is Purtscher-like retinopathy. Frequent causes of Purtscher-like retinopathy include acute pancreatitis, renal failure, and autoimmune disease. An association between Purtscher's retinopathy and acute pancreatitis was first reported by Inkeles and Walsh in 1975. Incidence is 0.24 persons per million per year (including Purtscher and Purtscher-like retinopathies)². Purtscher-like retinopathy in acute pancreatitis is

an indicator of multiorgan failure and is often associated with a fatal outcome³. Diagnosis of Purtscher's and Purtscher-like retinopathies is clinical, with a presentation that usually includes sudden vision loss of variable severity, hours to days after the causal pathology. Fundoscopic signs include cotton-wool spots and intraretinal-hemorrhages, described in 83–92% of a series of cases. Purtscherflecken are considered to be pathognomonic⁴, but occur in only 50% of cases. Treatment is variable with some physicians observing expectantly and others primarily administering corticosteroids.

Case presentation

A 35-year-old male with a history of alcohol abuse presented with complaints of epigastric pain that radiated to the back and flanks. On physical examination, he was conscious, alert, and hemodynamically stable. During the initial 4 hours of hospitalization, he suffered sudden visual impairment. Laboratory blood tests revealed total count 12,600 cells/cumm, mean corpuscular volume (MCV) 99 femtoliters (fl), mean corpuscular hemoglobin (MCH) 33.4 picograms (pg), platelets 1.42 Lakhs cells/cumm. Other remarkable values were serum amylase 1536 IU/l, serum lipase 1126 IU/l, alkaline phosphatase 118 IU/l, and γ -glutamyltransferase 34 IU/l, SGOT 168 IU/l, SGPT 134 IU/l. Chest x-ray was normal. Laboratory investigations were favourable for diagnosis of acute pancreatitis



The patient's best-corrected visual acuity was in counting fingers in both eyes. Slit-lamp examination and intraocular pressures were

normal. Ophthalmoscopic examination of the fundus revealed diffuse retinal whitening of the posterior pole with cotton-wool spots. There were a few superficial haemorrhages. Fluorescein angiography showed evidence of leakage from vessels in the areas corresponding to the cotton-wool spots seen via fundoscopy. The patient was treated with IV methyl prednisolone 1g x 3 days followed by oral steroids. Pancreatitis was treated by IV fluids, antibiotics. Patient's visual acuity improved to 6/60.

Discussion

Purtscher-like retinopathy with bilateral loss of vision is a rare and severe complication that may follow acute pancreatitis. Purtscher-like retinopathy is seen in diverse conditions, including acute pancreatitis; fat embolization; amniotic fluid embolization; preeclampsia; hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome; and vasculitic diseases, such as lupus. Pathophysiology being leukoembolization that causes arterial occlusion and infarction of the microvascular bed. possible sources of emboli include fat emboli in cases of long bone fractures and perhaps pancreatitis from enzymatic digestion of omental fat, amniotic fluid embolization during childbirth and postpartum, air emboli from traumatic chest compression, and granulocyte aggregation resulting from complement activation. Aggregation, which is induced by complement C5a. The most common retinal findings in Purtscher like retinopathy are cotton-wool spots, Purtscherflecken around the optic nerve, and intraretinal hemorrhages. It is widely assumed that the flecken are the result of occlusion of the precapillary arterioles. The characteristic finding is intraretinal whitening but with a clear zone (within 50 μ m) on either side of the retinal arterioles, venules, and precapillary arterioles. This is in contrast to cotton-wool spots, which have ill-defined edges⁴, and are located superficially over vessels. Less common reported findings include serous detachment of the macula, preretinal hemorrhages, dilated vessels, and optic

disc edema. Fluorescein angiography studies demonstrate capillary leakage and staining of the retinal arteries, nonperfusion of the small arterioles that surround the central macula, Perivenous staining, Venous dilation and leakage.

Five diagnostic Purtschner like retinopathy criteria ^{4, 5, 6}

1. Purtscherflecken
2. Retinal hemorrhages, low-to-moderate number (1–10)
3. Cotton-wool spots (typically restricted to posterior pole)
4. Probable or plausible explanatory etiology
5. Complementary investigation compatible with diagnosis

Treatment is variable with some physicians observing expectantly and others primarily administering corticosteroids. some case reports showed hastened visual recovery after corticosteroids use⁷, further information is needed to determine if corticosteroids can or cannot change the natural history of a patient with Purtscher's retinopathy. Patients with retinopathy due to systemic vasculitis, steroid therapy is theoretically beneficial⁸. Control of the underlying disease with other medications may be indicated.

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