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CASE REPORT | PANCREAS

Blind After a Binge: Purtscher-like Retinopathy in Acute Alcoholic Pancreatitis

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ABSTRACT

Purtscher-like retinopathy is an occlusive microvasculopathy that causes sudden onset vision loss of varying severity. The condition is a rare complication of acute pancreatitis and is scarcely documented in the literature. In addition, it is vision-threatening, and there are no evidence-based therapies available. We report a 37-year-old woman presenting with abdominal pain and nausea in the setting of heavy alcohol use. She was diagnosed with acute pancreatitis. During hospitalization, she developed acute visual disturbance and was found to have significantly diminished visual acuity. A dilated funduscopic examination demonstrated multiple retinal cotton wool spots bilaterally and macular edema. The patient was diagnosed with Purtscher-like retinopathy in the setting of acute pancreatitis. Although the prognosis for her vision was guarded, given the ischemic nature of the insult, she had subjective improvement in visual acuity during the remainder of the hospital course with symptomatic management.

INTRODUCTION

Purtscher retinopathy is an occlusive microvasculopathy, often associated with trauma that causes sudden onset vision loss of varying severity. It is labeled as Purtscher-like when caused by nontraumatic conditions. These conditions may include acute pancreatitis, renal failure, connective tissue disorders, childbirth, bone marrow transplant, and fat embolism syndrome.[1] Both Purtscher and Purtscher-like retinopathy are extremely rare, with a combined estimated incidence of 0.24 cases per million.[2] Although prognosis is variable, and some patients experience spontaneous resolution, this is a vision-threatening condition and thus is important to recognize. This is particularly important in acute pancreatitis, where it is associated with an overall poorer prognosis.[3] Given the rarity of the condition, there are no evidence-based therapies available. Therefore, increased recognition may allow for different therapies to be investigated. We present a case of Purtscher-like retinopathy in the setting of acute alcoholic pancreatitis.

CASE REPORT

A 37-year-old woman presented to the emergency department with a chief concern of abdominal pain. The pain had been ongoing for approximately one week and was reported to be a dull epigastric discomfort, rated 7/10 in severity, with radiation to the back. She also complained of associated nausea, with multiple episodes of bilious emesis. The patient reported similar symptoms intermittently over the past year, with this episode being the most severe. The episodes seemed to correlate with her alcohol use because over the same period she had begun drinking up to one pint of liquor daily. Aside from heavy alcohol use, her medical history was only significant for gastroesophageal reflux disease.

The initial laboratory results were notable for a lipase level of 161 IU/L (upper normal limit 51 IU/L), and an abdominal ultrasound was negative for cholelithiasis or biliary duct dilatation. The patient was diagnosed with acute alcoholic pancreatitis and admitted for supportive care, which included fluid resuscitation, analgesia, and antiemetics. She had no evidence of shock or organ failure.

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On the first day of hospitalization, the patient complained of visual hallucinations and diminished visual acuity. She was also noted to be tachycardic, hypertensive, and tremulous. With appropriate treatment of withdrawal, her vital signs normalized and tremors resolved; however, her visual disturbance persisted. Specifically, she complained of "blurred vision," "problems with near vision" (she could not read anything on the screen of her cellular phone), and "seeing colors in places where they don't belong."

Physical examination demonstrated equal, round, and reactive pupils, as well as intact extraocular movements. Visual acuity testing revealed 20/100 right-sided acuity and 20/200 left-sided acuity to both near and far visions, respectively. Ophthalmology was consulted, and a dilated funduscopic examination of both the right and left eye demonstrated multiple retinal cotton wool spots and macular edema (Figure 1). The patient was diagnosed with Purtscher-like retinopathy in the setting of acute pancreatitis.

Initially, it was thought that her visual disturbance was likely a symptom related to her alcohol withdrawal because she had many other withdrawal symptoms. However, the vision changes persisted despite the treatment of withdrawal. An important differential, in this case, included methanol poisoning, particularly given her recent alcohol abuse, although there was no reported methanol consumption and investigations did not reveal acute renal insufficiency or an anion gap acidosis.

The patient did not receive any specific treatment of Purtscher-like retinopathy because there are no evidence-based therapies available for this condition. She received supportive care for acute alcoholic pancreatitis, the inciting etiology.

Although the prognosis for her vision was guarded, given the ischemic nature of the insult, she had a subjective improvement in visual acuity during the remainder of the hospital course. Per recent patient records, it appears as though her vision has returned to baseline, although she has had multiple readmissions for acute alcoholic pancreatitis.

DISCUSSION

Purtscher retinopathy is an occlusive microvasculopathy that causes sudden onset vision loss. It was first described in 1910 when Otmar Purtscher noted areas of retinal whitening and hemorrhage in the posterior pole of both eyes of a patient after a traumatic head injury.[4] Although the exact mechanism of this pathology is unknown, the most accepted theory is microembolization with distal arteriolar precapillary occlusion and infarct of the retinal nerve fiber layer.[5,6] When this occurs in nontraumatic conditions, it is designated as Purtscher-like retinopathy.

A recent systematic review of several databases (Medline, EMBASE, ISI, EBSCO, Science Direct, and Google Scholar) from 1980 to 2010 identified 68 total cases of Purtscher and Purtscher-like retinopathies.[1] Of those, 13 occurred in the setting of acute pancreatitis, second only to trauma as a leading cause.[1] In acute pancreatitis, the suspected mechanism of ischemic insult to the retina involves pancreatic proteases entering the systemic circulation and causing widespread activation of the complement and coagulation cascades by complement-derived mediators.[7,8] This leads to embolization of leukocyte and fibrin aggregates to the retinal arterioles.[7,8] When seen in acute pancreatitis, limited data suggest that Purtscher-like retinopathy is associated with multiorgan failure and increased mortality.[3]

The diagnosis of Purtscher retinopathy is clinical, with varying loss of vision occurring usually hours to days after the causal pathology.[6] Diagnostic criteria have been proposed and generally include either an associated illness (eg, pancreatitis) or event (eg, trauma) that may be associated with Purtscher retinopathy and lesions on funduscopic examination.[1,2] The lesions can include cotton-wool spots (confined to the posterior pole), retinal hemorrhages, or Purtscher flecken, which are considered to be pathognomonic and are found in approximately 50% of cases.[1,2]

The prognosis for visual recovery in Purtscher retinopathy is variable, and whereas some patients experience spontaneous resolution, this is a vision-threatening condition.[1,2]

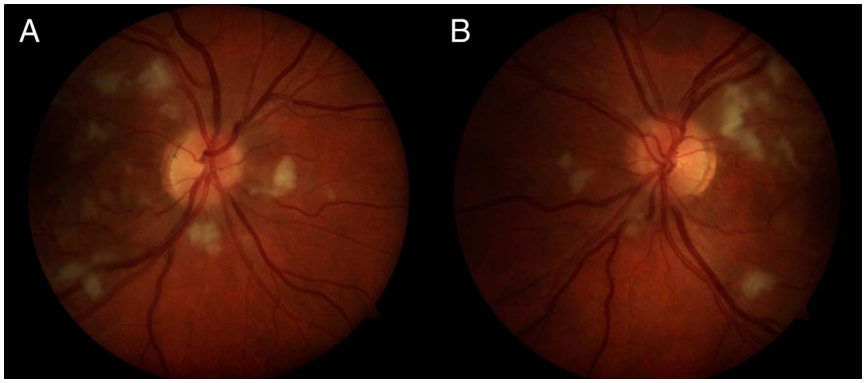

Figure 1. Multiple retinal cotton-wool spots and macular edema were seen in the (A) right and (B) left eye.

Furthermore, there are no well-established therapies or treatment guidelines available, given the paucity of data on this condition. High-dose corticosteroids have been suggested as a potential therapy, working by inhibiting complement-associated granulocyte aggregation and stabilizing damaged neuronal membranes.[6] Although there have been cases of improvement in vision after corticosteroid administration, they have not been found to show statistically significant improvement in outcomes and therefore remain controversial in this setting.[1,2,5,6,9] Other treatments with benefit seen in case reports include nonsteroidal anti-inflammatory drugs and hyperbaric oxygen.[10,11] Given the lack of validated beneficial therapies, observing expectantly and aggressive treatment of the underlying etiology may be the most reasonable course of action.[1]

DISCLOSURES

Author contributions: T. Tariq and M. Reaume drafted the manuscript. D. Hammar, A. Shallal, and M. Schauer edited and revised the manuscript. T. Tariq is the article guarantor.

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