CASE REPORT

Bilateral severe visual loss in a patient with acute pancreatitis: Purtscher-like Retinopathy

Perda visual severa bilateral após pancreatite: retinopatia de Purstcher

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ABSTRACT

Purtscher retinopathy is a unilateral or bilateral visual loss following acute injury to the thorax or head. It is characterized by large cotton-wool spots, hemorrhages, and retinal edema. Vision loss may be permanent due to isquemia of the retina, and optic atrophy. Is thought to be a result of injury-induced complement ativation causing granulocyte aggregation and leukoembolization. Other conditions may active complement and may produce similar fundus appearance including acute pancreatitis, collagen-vascular disease, childbirth, and amniotic fluid embolism. Herein, we describe a patient with bilateral permanent visual loss following diagnosis of acute pancreatitis.

Keywords: Retina. Pancreatitis. Optic atrophy. Complement activation. Optic nerve.

RESUMO

Retinopatia de Purscher é caracterizada por perda visual uni ou bilateral seguida de trauma de tórax ou crânio. A retinopatia apresenta-se com manchas algodonosas, hemorragias e edema da retina. A perda da visão pode ser permanente devido a isquemia da retina e atrofia óptica. A sua causa é provavelmente devido a ativação do complemento causando agregação dos granulocitos e a leucoembolização. Outras condições podem ativar o complemento produzindo quadro semelhante no fundo de olho, tais como pancreatite aguda, doenças de colágeno, parto e embolia de fluido amniótico. Relatamos o caso de uma paciente com perda de visão súbita e permanente após diagnostico de pancreatite aguda.

Palavras-chave: Retina. Pancreatite. Atrofia óptica. Ativação do complemento. Nervo óptico.

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INTRODUCTION

Purtscher Retinopathy is a condition first described in 1910 by Otmar Purtscher, in a patient with severe head trauma. It is characterized by sudden severe vision loss after acute compression injuries to the thorax or head, long-bone fracture, and crush injury. It is associated with presence of multiple white retinal patches, unilateral or bilateral, usually accompanied by retinal hemorrhages, large cotton-wool spots and occasionally disc edema.[1,2] Diverse conditions can cause a Purtscher-like retinopathy such as acute pancreatitis, fat and amniotic fluid embolization, preeclampsia, hemolysis, vasculitic diseases and lupus, and it has been also described in HELLP (hemolysis, eleveted liver enzymes, low platelets) syndrome, pancreatic adenocarcinoma, and renal failure.[3,4] This condition is thought to be a result of injure-induced complement activation, which can lead to granulocyte aggregation and leukoembolization.[2] The treatment is based on the underlying cause and the prognosis depends on the affected area. Some evidences support, as a routinely treatment, use of systemic steroids that can improve visual outcome in some patients, however, there is little evidence to back such treatment.[5]

CASE REPORT

30 years-old, female, began with repeated vomiting and abdominal pain. After 3 days showed low sudden visual acuity in both eyes. Laboratory tests revealed white blood count of 11.420/mm^3, hemoglobin of 13,6 g/dl, platelet count of 176.800/mm^3, amilase of 3.306 U/L (normal, 23 to 125U/L), lipase 3.944 U/L (normal, 23 to 60U/L), total bilirrubine of 1,60mg/dL. She was diagnosed with biliary pancreatitis and underwent laparoscopic cholecystectomy on the 26th day of hospitalization, but persisted with decreased visual acuity. In her history, no hypertension, diabetes mellitus, glaucoma, trauma or eye surgery. Ophthalmic examination found visual acuities of count finger at 2 meters, and sluggish pupillary light responses in both eyes. The anterior segment and intraocular pression were unremarkable. On examination of fundus revealed large peripapillary yellow-white patches at the level of the inner retina in both eyes, superficial retinal hemorrhage were observerd as well (Figure 1). Fluorescein angiography showed blockage of the choroidal fluorescence in the early stages, which was associated with the yellow-white patches and hemorrhage (Figure 2). The initial hypothesis was confirmed and treatment was initiated with oral prednisone 40mg/day. Despite the use of corticosteroids the patient did not improve the visual acuity. Five months after the diagnosis the visual acuity remained count finger in the OD, and fall to hand moviments in the OS, fundoscopy showed optic nerve atrophy in both eyes (Figure 3).

DISCUSSION

Purtscher Retinopathy is a rare disease, characterized by a severe sudden unilateral or bilateral visual loss associated with thorax or head trauma, however, also can occur in other situations, such as acute pancreatitis, collagen-vascular diseases, as systemic lupus erythematosus, and also has been seen in childbirth, fat embolism, and amniotic fluid.[6] In these cases the term Purtscher-like retinopathy is used. This visual disorder is a very rare systemic manifestation of acute pancreatitis which was not correlated to the severity of the disease. In general, the prognosis is good with improvement of visual acuity, but this may remain with permanent visual loss when it envolves the optic disc or the macula. Large cotton-wool spots, hemorrhages, and retinal edema are most commonly seen around the optic disc.[7] The fluorescein angiography shows evidence of arteriolar obstruction and leakage. Occasionnaly, patients may show disc edema and afferent pupillary defect. Vision may be permanently lost from ischemia, and optic atrophy may develop.

Figura 1. Fundus revealed large peripapillary yellow-white patches at the level of the inner retina and superficial retinal hemorrhage in both eyes.

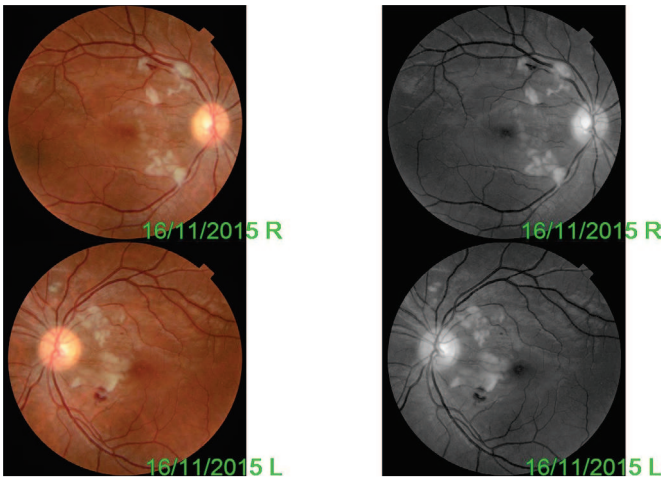


Figura 2. Fluorescein angiography showed blockage of the choroidal fluorescence in the early stages which was associated with the yellow-white patches and hemorrhage.

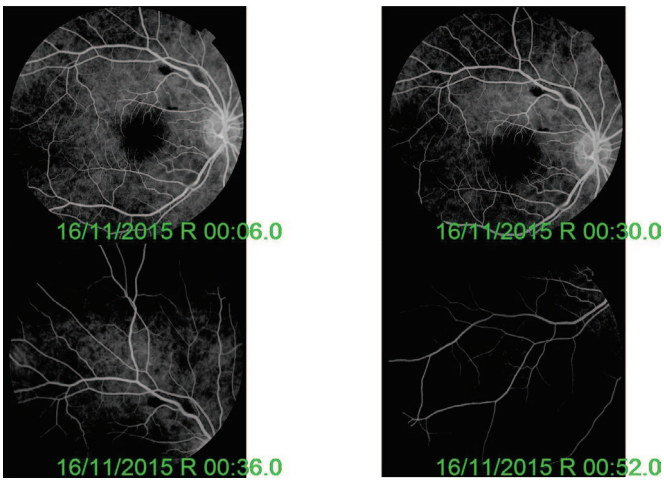
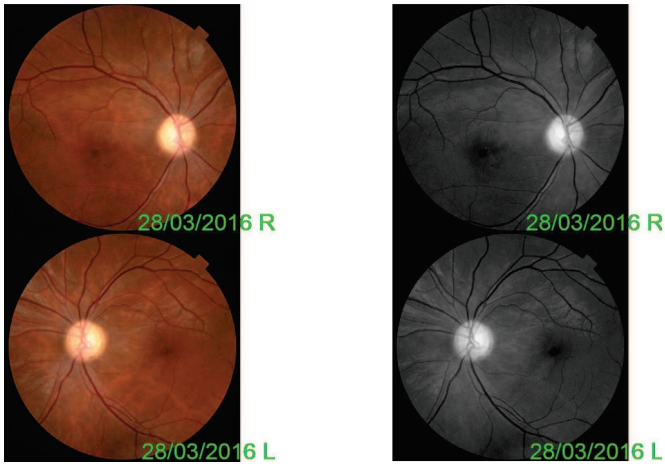


Figura 3. Fundoscopy showed optic nerve atrophy in both eyes.



The patophysiology is thought to be a result of injury-induced complement activation, which may cause a platelet-leukocyte aggregates blocking arterioles as seen in the peripapillary retina.[8] Purtscher-like retinopathy in association with acute pancreatitis had already been well documented in several cases reports. Pancreatitis increases trypsin activity, which in turn activated the complement system, which is thought to be one of the mechanism for the development of the retinopathy in acute pancreatitis.[4] Clinicopathologic studies demostrate occluded retinal arterioles and choroidal vessels, focal areas of edema within the inner retinal layers, cystoide spaces, small hemorrhages, and disruption of the photoreceptors. Fat emboli, increased venous pressure, retinal arteriolar spams, fluctuations in blood pressure, and anemia has also been related to the retinopathy. Although Purtscher's retinopathy is diagnosed typically after the onset of acute pancreatitis, Sanders[9] et al describe a patient with hystory of alcohol abuse with Purtscher's retinopathy 2 weeks berofe symptoms of acute pancreatitis, Sharma et al also report a case of a patient with chronic pancreatitis, who developed Purtscher's retinopathy 6 months before the development of fulminat acute pancreatitis.[7]

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