Case report

Purtscher retinopathy after head trauma

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Abstract
Purtscher's retinopathy is an acute retinal microangiopathy primarily associated with severe trauma, characterized by occlusion of the retinal microvasculature that causes edema, hemorrhages, and whitish lesions with a sudden decrease in visual acuity. **Case presentation.** A man with a history of closed head injury following a fall from a height of six meters. He was initially evaluated at a tertiary care hospital and discharged without apparent neurological impairment; however, 24 hours after the event, he developed sudden bilateral vision loss. Fundus examination revealed optic discs with poorly defined borders, multiple intraretinal hemorrhages, and hypopigmented lesions in both eyes; these findings, when correlated with the patient's history of trauma, led to the diagnosis. **Treatment.** Treatment was initiated with systemic and topical corticosteroids, along with topical anti-inflammatory. **Clinical evolution**. During close follow-up, the patient showed progressive resolution of the retinal hemorrhages and improvement in visual acuity, indicating a favorable clinical course; however, there was no complete resolution at the five-month follow-up, as he presented with irreversible macular atrophy as a sequela.

Keywords
Retinal Diseases, Traumatic Brain Injuries, Maculopathy.

Resumen
La retinopatía de Purtscher es una microangiopatía retiniana aguda asociada principalmente con traumatismos graves, caracterizada por oclusión de la microvasculatura retiniana que produce edema, hemorragias y lesiones blanquecinas con disminución súbita de la agudeza visual. **Presentación del caso.** Se presenta el caso clínico de un hombre con antecedente de traumatismo craneoencefálico cerrado por caída desde seis metros de altura, evaluado inicialmente en un hospital de alta complejidad y egresado sin compromiso neurológico aparente, quien 24 horas después del evento, desarrolló pérdida visual bilateral abrupta. En el examen de fondo de ojo se evidenciaron discos ópticos con bordes mal definidos, hemorragias intrarretinianas múltiples y lesiones hipopigmentadas en ambos ojos, hallazgos que permitieron establecer el diagnóstico al correlacionarlos con la historia traumática. **Intervención terapéutica.** Se instauró tratamiento con corticosteroides sistémicos y tópicos, además de antiinflamatorios tópicos. **Evolución clínica.** Durante el seguimiento estrecho, el paciente mostró reabsorción progresiva de las hemorragias retinianas y mejoría de la agudeza visual, lo que evidenció una evolución clínica favorable; sin embargo, no hubo resolución completa a los cinco meses de seguimiento, ya que presentó como secuela atrofia macular irreversible.

Palabras clave
Enfermedades de la Retina, Traumatismo Craneoencefálico, Atrofia.

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Retinopatía de Purtscher posterior a traumatismo craneoencefálico

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Introduction

Purtscher's retinopathy is an occlusive vascular disorder described by Otmar Purtscher in 1910.[1] It is a retinopathy secondary to retinal capillary ischemia,[2] which occurs in patients with a history of trauma to areas distant from the eyes.[3] The underlying pathogenesis of Purtscher's retinopathy is unclear, but it may be related to embolism and occlusion of precapillary arterioles in the retina.[4] These ophthalmoscopic changes may also be present in various non-traumatic conditions (pancreatitis, amniotic fluid embolism, systemic vasculitis, etc.), a condition known as Purtscher-like retinopathy; this diagnosis is accompanied by multisystem organ failure and, therefore, carries a poor prognosis.[4-6]

For diagnosis, Miguel *et al.*, described cotton-wool spots (93 %), retinal hemorrhages (65 %), and Purtscher flecken (63 %) as the most common fundoscopic findings. They also proposed that a diagnosis of Purtscher retinopathy or Purtscher-like retinopathy can be established when at least three of five criteria are met: the presence of Purtscher flecken, mild to moderate retinal hemorrhages, cotton-wool spots confined to the posterior pole, a probable explanatory etiology, and ancillary tests consistent with the diagnosis.[7-9]

The objective of this case report is to describe and analyze a clinical manifestation of Purtscher retinopathy in the context of traumatic brain injury, highlighting its diagnostic and therapeutic importance. Its publication is considered relevant due to the relative rarity of this condition, the possibility of delayed diagnosis due to the initial absence of obvious ocular symptoms, and the need for a high index of clinical suspicion in patients with severe trauma who develop late-onset visual disturbances. Furthermore, the case provides an opportunity to reflect on the therapeutic approach using corticosteroids and its impact on visual outcome, offering a useful clinical lesson for ophthalmologists, neurologists, and emergency physicians in the early identification, timely management, and follow-up of this potentially disabling complication.

Case presentation

A man with a history of closed head injury resulting from a fall from a height of six meters, who was evaluated at a tertiary care hospital and discharged without apparent neurological impairment. Twenty-four hours after the event, he developed sudden bilateral vision loss. Examination of the fundus revealed poorly defined optic nerves, multiple intraretinal hemorrhages, and hypopigmented lesions in the macular region and mid-periphery (Figure 1); these findings, when correlated with the history of trauma, led to the diagnosis.

The initial evaluation revealed visual acuity of 2.00 logMAR (hand motion) in the right eye and 1.70 logMAR (finger counting) in the left eye. Slit-lamp biomicroscopy revealed no abnormalities in the anterior segment. Fundoscopy (Figure 1) revealed a clear vitreous, poorly defined and elevated optic nerve heads, retinal hemorrhages, and hypopigmented spots (Purtscher spots) in the macular region and the mid-periphery. Optical coherence tomography (OCT) was performed on both eyes, revealing marked macular atrophy, with central macular thicknesses of 175 µm and 190 µm, respectively (Figure 2).

Treatment

Systemic anti-inflammatory treatment was initiated with oral prednisone at a dose of 100 mg/day (divided into two 50-mg doses) for three days. Subsequently, treatment continued with a gradual tapering regimen at a rate of 20 mg every three days until reaching 20 mg/day, followed by reductions of 5 mg every three days until the medication was definitively discontinued after 24 days of treatment, with a total cumulative dose of 990 mg. In addition, topical treatment was initiated with 1 % prednisolone, one drop every 4 hours for seven days with gradual tapering, and 0.1 % nepafenac, one drop every 12 hours in both eyes.

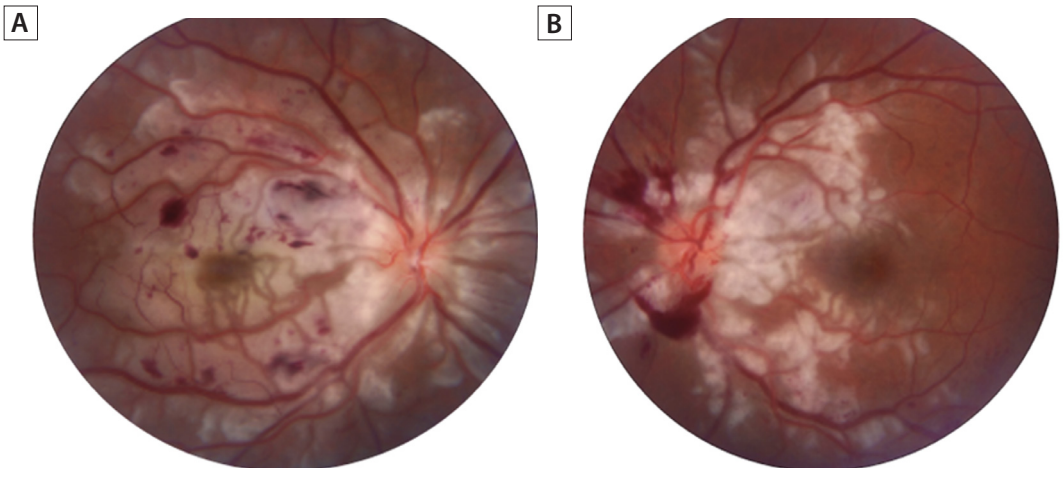

Figure 1. Bilateral fundus photography performed 24 hours after traumatic brain injury. (A) Right eye. (B) Left eye. Fundus photograph showing a clear vitreous, optic nerves with poorly defined and elevated margins, retinal hemorrhages, and hypopigmented spots (Purtscher spots) in the macular region and mid-periphery. Source: ZEISS VISUCAM 524, Ophthalmology Department, Salvadoran Social Security Institute.

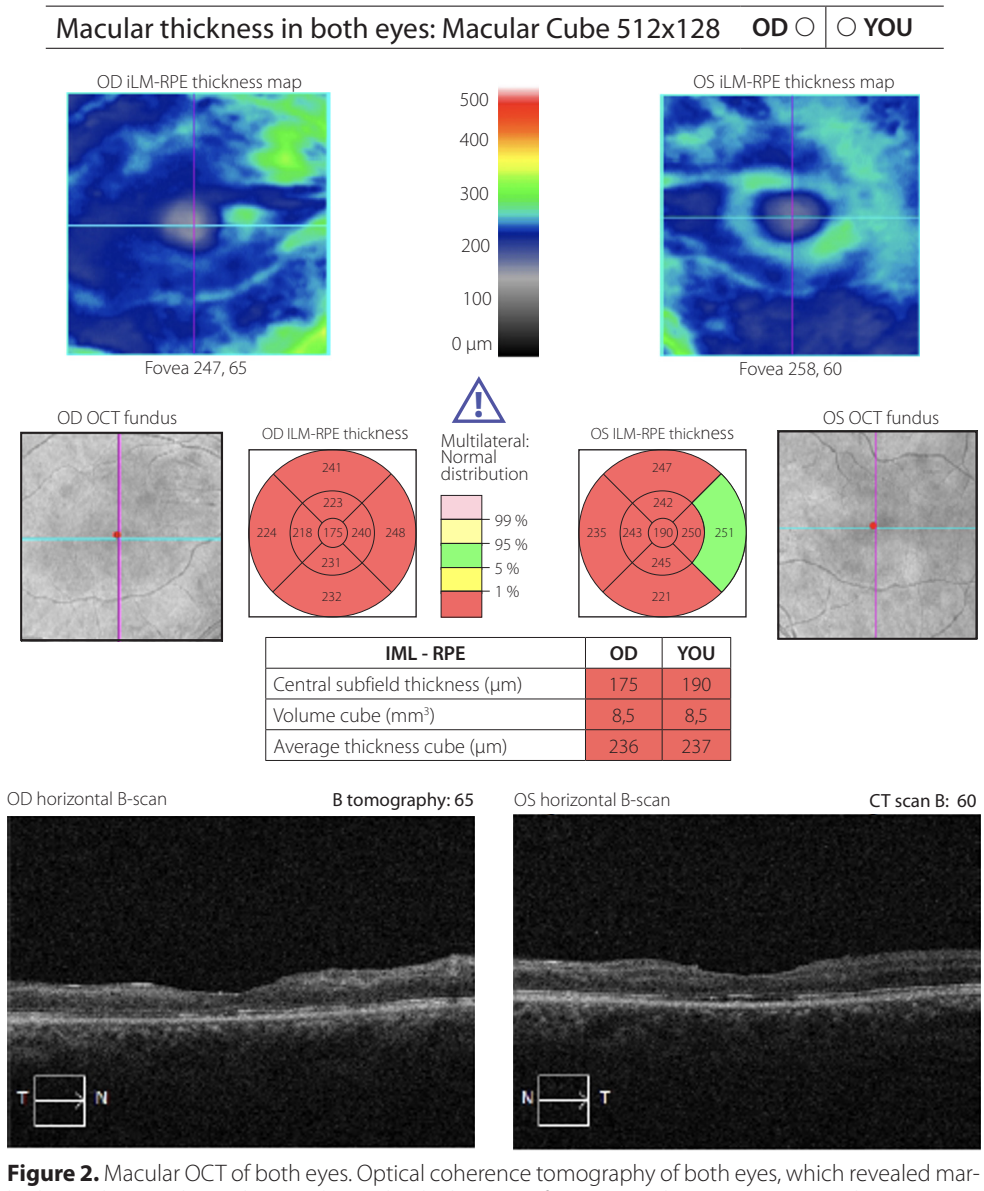

Figure 2. Macular OCT of both eyes. Optical coherence tomography of both eyes, which revealed marked macular atrophy, with central macular thicknesses of 175 µm and 190 µm, respectively.
Source: ZEISS CIRRUS 6000, Ophthalmology Department, Salvadoran Social Security Institute.

Clinical diagnosis

At the five-month follow-up, visual acuity was 1.30 logMAR (20/400) in the right eye and 0.54 logMAR (20/70) in the left eye (Figure 3), along with a significant improvement in clinical retinal findings, including resorption of the hemorrhages and a reduction in ischemic lesions. However, despite the favorable course and the visual gain achieved, recovery was not complete, and macular atrophy persisted as a sequela, as observed in follow-up studies (Figure 3).

Discussion

Purtscher's retinopathy is an occlusive microvasculopathy associated with some form of external trauma to the eyeball, such as traumatic brain injury or chest trauma during chest compressions. Purtscher's-like retinopathy presents with an identical clinical picture in the absence of trauma; the precipitating event is some type of inflammatory condition, such as pancreatitis or HELLP syndrome.[11–13]

This condition is a rare cause of acute bilateral vision loss resulting from occlusion of the precapillary arterioles of the retina. There are multiple theories regarding the mechanisms underlying the lesions. Microembolization secondary to fat, air, leukocytes, platelets, and fibrin has been considered a possible cause.[14]

To establish the diagnosis, four of the five diagnostic criteria must be present: Purtscher's spots, low to moderate numbers of retinal hemorrhages (1 to 10), a probable explanatory etiology, and supportive diagnostic findings; only cotton-wool spots were absent.

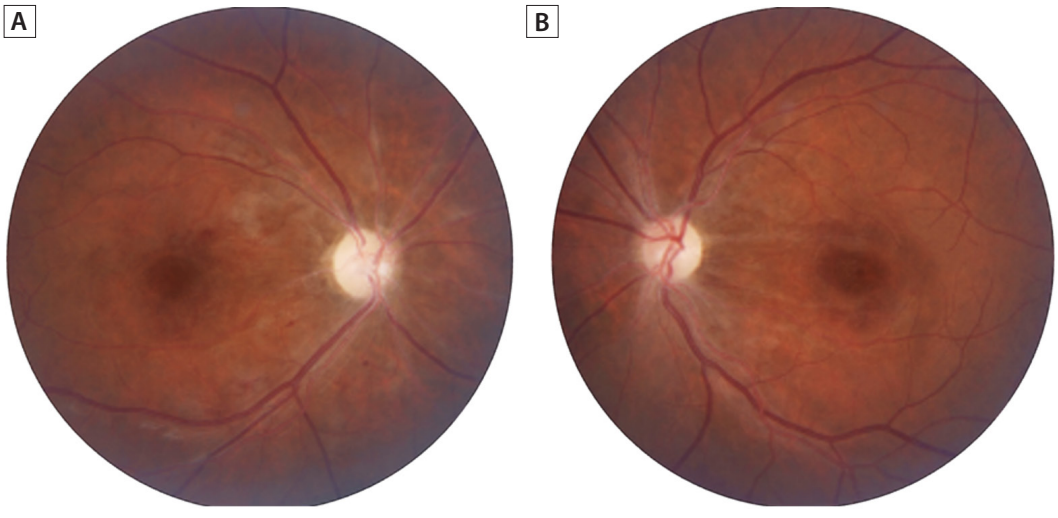

Figure 3. Bilateral fundus photography performed five months after the traumatic brain injury. (A) Right eye. (B) Left eye. Resolution of hemorrhages and reduction in ischemic lesions. Source: ZEISS VISUCAM 524, Ophthalmology Department, Salvadoran Social Security Institute.

Purtscher's retinopathy is a clinical diagnosis whose presentation typically includes unilateral or bilateral vision loss of varying severity and visual field defects. It can be spontaneous or may occur within 24 to 48 hours after the underlying condition,[10] which also corresponds to the course of the patient's illness.

Regarding treatment, cases related to the use of corticosteroids have been reported in the literature; however, there is no definitive treatment guideline that can prevent irreversible damage.[15,16]

This case provides relevant clinical lessons for medical practice: from a diagnostic standpoint, Purtscher's retinopathy should be considered in any patient with a recent history of traumatic brain injury or chest compression who develops acute or subacute vision loss, even in the initial absence of evident neurological involvement. From a therapeutic perspective, early initiation of systemic and topical corticosteroids, along with topical anti-inflammatory agents, proved crucial in promoting the reabsorption of retinal hemorrhages and enabling partial visual recovery. Regarding prognosis and follow-up, the need for close monitoring is emphasized to detect optic atrophy and irreversible macular damage in a timely manner and thus avoid delays in post-traumatic ophthalmologic evaluation.

The observed clinical course is explained by traumatic retinal occlusive microangiopathy, with capill y ischemia, edema, and hemorrhages that led to neuroretinal damage; the diagnostic delay was influenced by the initial absence of ocular symptoms and the lack of early ophthalmologic evaluation despite a high-risk mechanism of injury. The key clinical reasoning was the correlation between the recent history of trauma and the characteristic fundoscopic findings, while therapeutic decisions were aimed at reducing retinal inflammation and edema in order to limit ischemic damage and optimize visual prognosis.

This case is significant because it demonstrates a post-traumatic presentation with delayed onset of visual symptoms, which may delay diagnosis in the absence of high clinical suspicion. Unlike other reports where vision loss occurs immediately after trauma, in this patient symptoms appeared 24 hours later, highlighting that the absence of early visual manifestations does not rule out ischemic retinal damage.

The fundoscopic findings were decisive for the diagnosis and highlight the value of early fundus examination in patients with traumatic brain injury who develop visual disturbances, enabling this condition to be distinguished from others such as central retinal artery occlusion, traumatic optic neuropathy, or post-contusion macular edema.

This report provides evidence supporting the need for active visual monitoring during the first 72 hours after trauma and can help clinicians in emergency medicine, neurology, and ophthalmology recognize this condition and take timely action. Overall, it reaffirms that Purtscher's retinopathy should be considered in the differential diagnosis of acute post-traumatic vision loss and that early ophthalmologic evaluation is key to optimizing management and visual prognosis.

Ethical considerations

This case report has been prepared in accordance with the principles of the Declaration of Helsinki, thereby ensuring the confidentiality of the patient, who authorized its publication through informed consent.

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