Short communication

Third cranial nerve palsy and Purtscher retinopathy in a child with multiple injuries

P. Larrañaga-Fragoso a,*, Z. del-Barrio a, S. Noval a, N. Pastora a, A. Royo b

a Servicio de Oftalmología, Hospital Universitario La Paz, IdiPaz, Madrid, Spain
b Servicio de Radiología, Hospital Universitario La Paz, IdiPaz, Madrid, Spain

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Abstract

Case report: A 4 year-old girl was referred to our hospital after have suffered a severe accident. The patient was diagnosed with complete third nerve palsy in her right eye and Purtscher retinopathy in her left eye.

Discussion: Purtscher retinopathy is a rare condition. The diagnosis is made on clinical ground and its treatment is not well defined although it is believed that systemic steroids could improve the visual outcome. Traumatic third nerve palsy has a poor spontaneous recovery. The use of botulinum toxin might be useful in children to improve the recovery rate, maintaining binocularity, and avoiding amblyopia in other cases.

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Parálisis del tercer nervio craneal y retinopatía de Purtscher en niña politraumatizada

Resumen

Caso clínico: Una niña de 4 años ingresa por politraumatismo grave por arrollamiento. Durante la evolución se le diagnostica de parálisis completa del tercer par del ojo derecho y retinopatía de Purtscher en ojo izquierdo.

Discusión: La retinopatía de Purtscher es poco frecuente. El diagnóstico es clínico y su tratamiento no está estandarizado aunque los corticoides sistémicos podrían mejorar el pronóstico visual. Las parálisis del tercer par traumáticas tienen baja tendencia a la recuperación espontánea. El uso de toxina botulinica en niños podría mejorar la tasa de recuperación total y podría ser útil para acelerar la resolución y así permitir binocularidad y evitar la ambliopía.

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* Corresponding author.
E-mail addresses: paulalarranaga@gmail.com, paulalac@hotmail.com (P. Larrañaga-Fragoso).

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Case report

Female, age 4, admitted to the Intensive Care Unit (ICU) for multiple traumatism after being run over by a train. The patient exhibited a severe systemic situation with cranioencephalic and hemiperenitoneal traumatism as well as pulmonary concussion, being administered intravenous corticoid, antibiotics as well as antiseizure drugs.

In the first ophthalmological examination carried out in the ICU, complete right eye (RE) palpebral ptosis and exotropia was observed with limitation in verticalduction and adduction. The right pupil exhibited medium midriasis with diminished direct photo-motor reflex, while the left pupil reacted normally. The anterior pole of both eyes did not exhibit pathological alterations, whereas the left eye (LE) exhibited a peripapillary retinal edema with retinal hemorrhages at varying depths and slight vascular ingurgitation and tortuosity (Fig. 1). Posterior pole retina whitening produced an appearance of cherry red spots. The final clinical diagnostic was complete palsy of the RE III cranial pair and Purtscher retinopathy in LE. Cerebral and orbital computerized axial tomography revealed fractures in the orbit as well as at the cranial base level (Fig. 2).

Two weeks later, visual acuity was of 0.5 in the RE and finger counting at 2 m in the LE. Anisocoria persisted and ptosis improved, exhibiting a margin-reflex 1 (DMR1) distance of 1 mm which it was decided to observe. RE remained in –45 DP exotropia. LE funduscopy revealed the absence of the posterior pole retinal whitening and normal vascular pattern, although with persistent hemorrhages. Optic coherence tomography (OCT) (Cirrus HD-OCT; Carl Zeiss Meditec, Dublin, CA) in LE evidenced diminished retinal thickness at the macular level with general layer de-structuring (Fig. 3). RE OCT was normal. Due to the persistence of exotropia and said restrictions it was decided to inject botulin toxin (Botox®, Allergan) in the lateral

Fig. 1 – Normal RE. LE exhibits peripapillary hemorrhages at the nervous fiber layer and deep hemorrhages in the retina. Retinal whitening around vessels with pseudocherry spots. Ingurgitation and vascular tortuosity in the superior temporal branch.

Fig. 2 – 2 D Coronal Reconstruction (A and B) and 3 D surface (C and D) over images acquired with helicoidal scanner. (A and C) Multiple fractures in the right orbit involving roof, natural and medial walls (yellow). (B and D) Sphenoid sinus left wall fracture (red).
rectus muscles. In addition, in view of persistent anisocoria and accommodation difficulties, progressive spectacles were prescribed with +2.5 D.

Two years after the accident, the patient exhibited a visual acuity of 0.9 in RE and 0.15 in the LE. Orthotropia was maintained in all gaze positions without restrictions. Right eyelid ptosis recovered although minimum asymmetry with the contralateral eyelid remained. Funduscopy evidenced LE macular atrophy.

Discussion

Purtscher’s retinopathy was described in 1910\(^1\) by Otmar Purtscher. This condition is characterized by the presence of multiple cotton-like exudates, retinal hemorrhages and optic nerve tumefaction. Typically, superficial retinal whitish areas appear which respect the 50 \(\mu\) margin around vessels, corresponding to the perivascular area without capillaries.\(^2\)

Purtscher’s retinopathy appears mainly after cranial traumatism without direct ocular trauma, although other mechanisms are possible.\(^3\) Physiopathology is unknown but it could be an embolic mechanism of fatty and platelet origin due to activation of the supplement cascade giving rise to vessel occlusion of the pre-capillary arterioles.

The visual prognostic of Purtscher’s retinopathy is variable, although the majority of patients exhibit spontaneous recoveries.\(^4\) Poor visual results are associated to significant macular ischemia, occasionally related to associated atypical central artery obstruction conditions.\(^5\) It has been postulated that systemic corticoids could assist functional recovery.

However, in the present case visual recovery was poor. Two weeks after the accident, OCT already exhibited macular atrophy with possible ischemic process involving central retinal artery obstruction in the context of Purtscher’s retinopathy. Occlusive therapy was not pursued due to poor recovery possibilities.

III pair palsy is infrequent in children. Traumatisms and congenital conditions are the most frequent causes.\(^6\) Traumatic palsy has poor visual prognosis not only due to the risk of amblyopia but to possible optic neuropathy and associated cortical damage.\(^7\)

It is recommended to delay surgical treatment of palsy at least 6 months due to the possibility of spontaneous recovery. Surgical management is complex and could require an average of 2-3 surgical procedures in 5-5 years.\(^8\)

Botulin toxin injections for traumatic III pair palsy have not demonstrated to improve complete recovery rates in the general population, although it shortens recovery time.\(^9\) This explains the early application of Botox in children to promote binocularity and preventing amblyopia.

In the present case, a prognostic of spontaneous exotropia recovering was deemed unlikely due to its traumatic cause and the sensory component secondary to LE Purtscher’s retinopathy. It was decided to inject botulin toxin at an early stage in order to shorten resolution time and thus avoid compensation torticollis and also in an attempt to prevent the persistence of exotropia as a sequel, which would be reinforced by the sensory component. The result was good although it could also be said that low LE visual acuity could have made a positive contribution by obliging RE to carry out ductions to increase visual field.
**Conflict of interests**

No conflict of interests was declared by the authors.

**REFERENCES**