Terson syndrome after footvolley injury

Sindrome de Terson após contusão no futevôlei

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How to cite:
Horowitz SA, Damasceno NA, Brasil OF, Damasceno EF. Terson syndrome following a traumatic brain injury. Rev Bras Oftalmol. 2025;84:e0015.

doi:
https://doi.org/10.37039/1982.8551.2025.0015

ABSTRACT

To report a case of Terson syndrome following a traumatic brain injury during a footvolley match. The patient was undergoing evaluation with the following tests: visual acuity, retinography, angiography, and optical coherence tomography. Follow-up was maintained for 3 months with conservative treatment. Terson syndrome associated with head trauma is poorly documented as fundoscopy is not performed in most patients.

RESUMO

Relatar um caso de síndrome de Terson por traumatismo cranioencefálico durante um jogo de futevôlei, que recebeu monitorização através dos exames de acuidade visual, retinografia, angiografia e tomografia de coerência óptica. O tratamento se manteve conservador por 3 meses. A síndrome de Terson associada a traumatismo cranioencefálico é pouco documentada devido à fundoscopia não ser realizada na maioria dos pacientes.

Keywords:
Retinal hemorrhage; Subarachnoid hemorrhage; Brain injuries, traumatic; Craniocerebral trauma; Tomography, optical coherence; Conservative treatment; Visual acuity

Descritores:
Hemorragia retiniana; Hemorragia subaracnóidea; Lesões encefálicas traumáticas; Traumatismos craniocerebrais; Tomografia de coerência óptica; Tratamento conservador; Acuidade visual

Received on:
Jun 21,2024

Accepted on:
Nov 21, 2024

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Conflict of interest:
no conflict of interest.

Financial support:
no financial support for this work.

INTRODUCTION

Terson syndrome was first described in 1881 by the German ophthalmologist Moritz Litten by observing the presence of intravenous hemorrhage with subarachnoid hemorrhage (SAH), and in 1900 it was named by the French ophthalmologist Albert Terson after the description of a syndrome of vitreous hemorrhage in association with SAH.[1,2] The syndrome is characterized by SAH, intracranial hemorrhage or traumatic brain injury (TBI) associated with intraocular hemorrhage that may be vitreous, sub-hyaloid, sub-retinal, internal limiting sub-membrane or intra-retinal hemorrhage.[3-5] There are no reports of involvement of the outer retina, ellipsoid zone, external limiting membrane, or retinal pigment epithelium, differing from Berlin edema.[6]

The cause appears to be a sudden increase in intracranial pressure (ICP), causing rupture of the retinal vessels, as in Valsalva retinopathy. However, in the case of Terson syndrome, ocular hemorrhage can occur hours or days after the acute event. The exact mechanism remains to be elucidated.[7,8]

CASE REPORT

The clinical case consists of an 18-year-old girl who suffered a TBI during a footvolley match, who developed headaches and dizziness, and 5 days later noticed a spot in the center of vision on the left. Visual acuity was maintained at 20/20 in both eyes, and fundoscopy showed an image of foveal retinal hemorrhage in the left eye (Figure 1).

Retinal assessment was performed and an image measuring 8 µm was found in the left macula. On angiography, it was located intraretinal (Figures 2). Using OCT, it was possible to evaluate the sub-internal limiting membrane of the retina and intraretinal locations. And according to the OCT (A) image, it was a hemorrhage in the superficial plexus of the retina (Figure 3).[9]

Follow-up was maintained for 3 months with conservative treatment and complete resolution.

DISCUSSION

It is believed that Terson syndrome may be caused by an increase in ICP, either by hemorrhage or TBI, and is characterized by ocular hemorrhage, including vitreous,

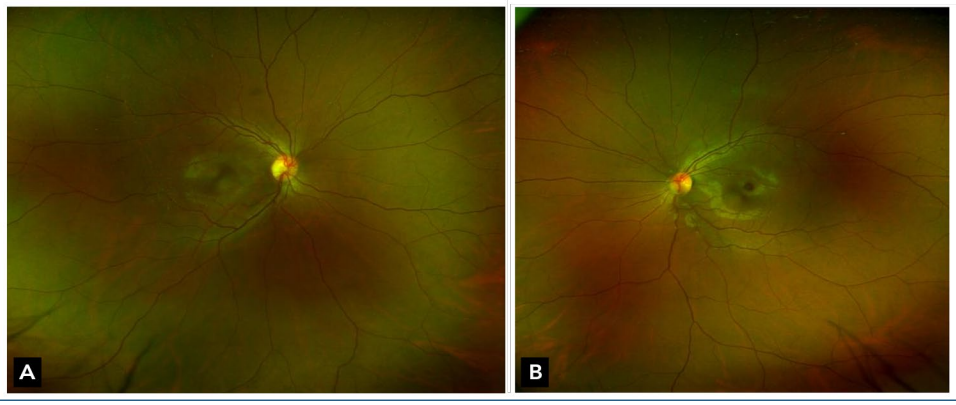

Figure 1. Wide field retinography (Optos): (A) right eye, normal; (B) left eye, rounded foveal retinal hemorrhage.

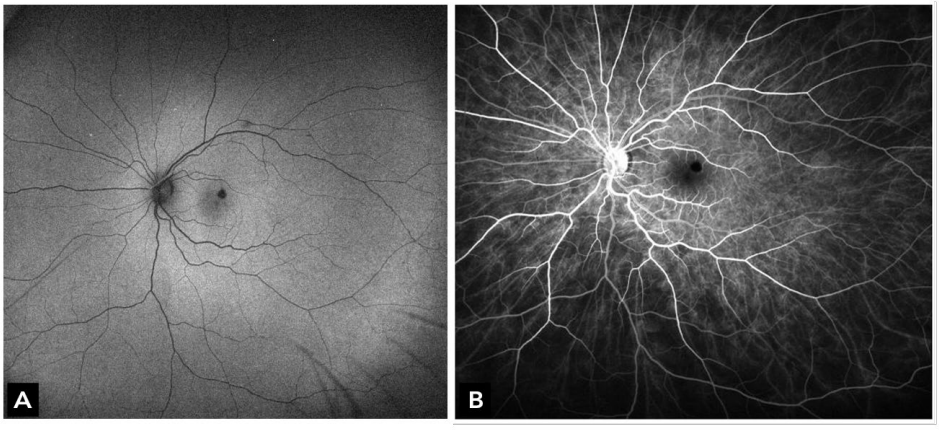

Figure 2. Wide field retinography (Optos). (A) Fundus autofluorescence; (B) angiography with fluorescein.

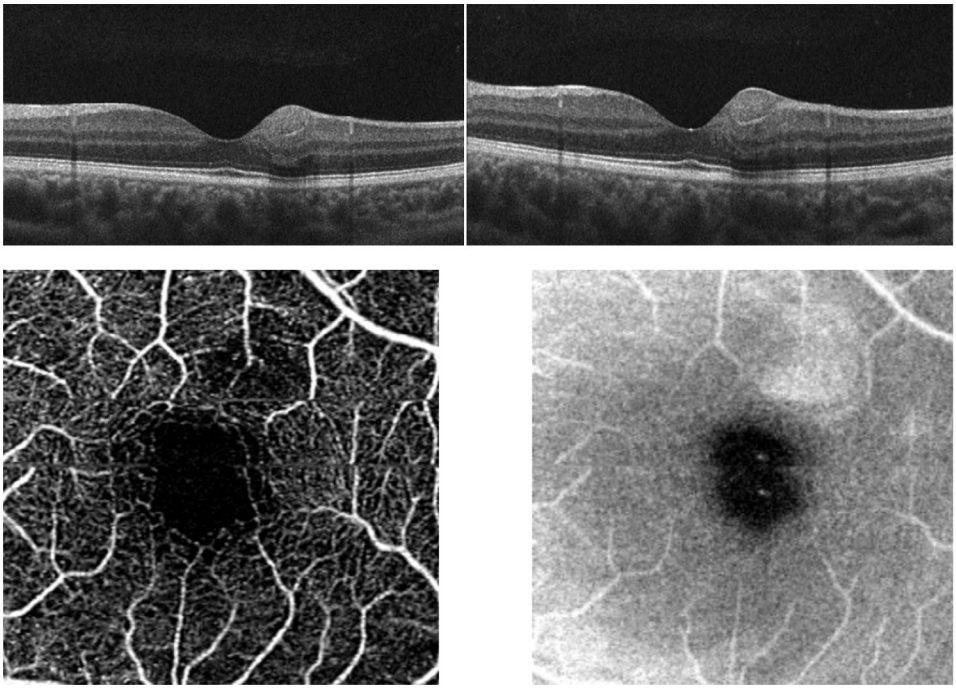

Figure 3. Stratus optical coherence tomography scans: sub-MLI and intraretinal hemorrhage. Optical coherence tomography angiography images: superficial retinal capillary plexus.

preretinal, intraretinal, or subretinal hemorrhage. The ophthalmological presentation is variable, and there may be hemorrhage from the subretinal space to the vitreous cavity, with the most frequent presentations being vitreous or pre-retinal hemorrhage.[3]

The origin of retinal hemorrhages and their connection with increased ICP is still controversial, but there are many theories, such as compression of the central retinal vein (which is in the subarachnoid space of the optic nerve sheath), high cerebrospinal fluid pressure, and/or accumulated blood. This would result in retinal venous stasis and increased venous pressure in the retinal veins, leading to venous engorgement, rupture of retinal capillaries, and retinal hemorrhages. Terson syndrome may develop several days or even weeks after the sudden increase of ICP, unlike Valsalva retinopathy, where a sudden increase in ICP causes retinal vessels to rupture.[3,5,10]

Delay in diagnosis may affect the visual prognosis. Surgical treatment with posterior retinal vitrectomy is indicated when there is extensive intraocular hemorrhage that does not resolve spontaneously or when visual acuity at presentation is very low.[11-13]

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