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Short communication

Retinal ischaemia and delayed fibrovascular proliferation associated with an optic nerve coloboma☆

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ABSTRACT

Case report: A patient with a left optic nerve coloboma with late development (at 15 years of age) of vitreoretinal fibrovascular proliferation and tractional retinal detachment (TRD). Sectorial retinal photocoagulation was performed with regression of the proliferative tissue and exudation.

Discussion: Congenital optic nerve anomalies may be associated with significant retinal avascularity, ischaemia and late fibrovascular proliferation. Laser is effective for regression of the neovascular tissue and preventing TRD progression.

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Isquemia retiniana y proliferación fibrovascular tardía asociada a coloboma de nervio óptico

Palabras clave:
Isquemia retiniana
Coloboma de nervio óptico
Anomalía congénita de nervio óptico
Desprendimiento de retina traccional
Fotocoagulación retiniana

RESUMEN

Caso clínico: Paciente con coloboma de nervio óptico izquierdo, que desarrolla tardíamente (a los 15 años de edad) proliferación fibrovascular vitreorretiniana y desprendimiento de retina traccional (DRT). Se realiza fotocoagulación retiniana sectorial con regresión del tejido proliferativo y la exudación asociada.

Discusión: Las anomalías congénitas del nervio óptico pueden asociar avascularidad e isquemia retiniana significativa y proliferación fibrovascular de aparición tardía. El tratamiento láser es efectivo para obtener la regresión del tejido neovascular y prevenir la progresión del DRT.

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Introduction

Optic nerve (ON) coloboma is a stationary congenital alteration caused by the anomalous closure of the fetal fissure in the optic cup.[1] The typical location of colobomae is inferonasal, and their severity is highly variable ranging from small changes in the retinal pigment epithelium to large cups. In addition, they could associate additional ocular colobomae.

As with other congenital ON anomalies such as hypoplasiae, peripapillary staphyloma or *morning glory*, ON colobomae could develop complications such as subretinal exudation, extra-retinal neovascularization, peripapillary choroidal neovascularization and tractional retinal detachment (TRD).[2,3] This paper presents the case of a patient with ON coloboma who developed late fibrovascular proliferation and TRD and with good response to sectorial retinal photocogulation.

Clinic case report

Patient, 4 months old, referred due to strabismus. Examination revealed fixing and following objects with the right eye (RE). The left eye (RE) exhibited light perception and endotropia of +45 prism diopters. Intrinsic ocular motility and anterior pole did not provide pathological findings in any eye. In funduscopy, the RE exhibited a posterior segment within normal limits and an inferior ON coloboma with adjacent tiger-like appearing retina. Retinal neovascularization in the inferotemporal sector of the RE was incomplete, ending at the equatorial level in the temporal zone and retro-equatorial level in the inferior zone. Hourly RE occlusions were prescribed but at one year of age, treatment was discontinued due to poor patient management with the LE. Regular ophthalmological controls were carried out, registering a visual corrected acuity (VA) of 20/15 in RE and finger counting in RE. At the age of 15, a vitreoretinal fibrovascular proliferation was identified in a scheduled visit, exhibiting intra- and subretinal exudation as well as TRD in the inferior zone of the RE (Fig. 1). Fluorescein angiography revealed retinal avascularity in the temporal-inferior periphery with well-defined limits and neovascularization in the anterior edge (Fig. 2).

It was decided to perform sectorial photocoagulation with argon laser over the avascular retina. Subsequently, the exudation progressively disappeared together with vascular proliferation component regression. The fibrous proliferation component partially regressed. Bilateral VA remained stable 24 months after photocoagulation (Fig. 3).

Discussion

Recently, optic disk congenital anomalies have been linked to avascularity and primary peripheral retinal ischaemia in children.[3] This involves incomplete terminal vascularization of the retina which could subsequently give rise to complications such as fibrovascular proliferations, vitreous hemorrhages and TRD, similarly to other infant primary ischemic-proliferative retinopathies, such as retinopathy of prematurity or familial exudative vitreoretinopathy.

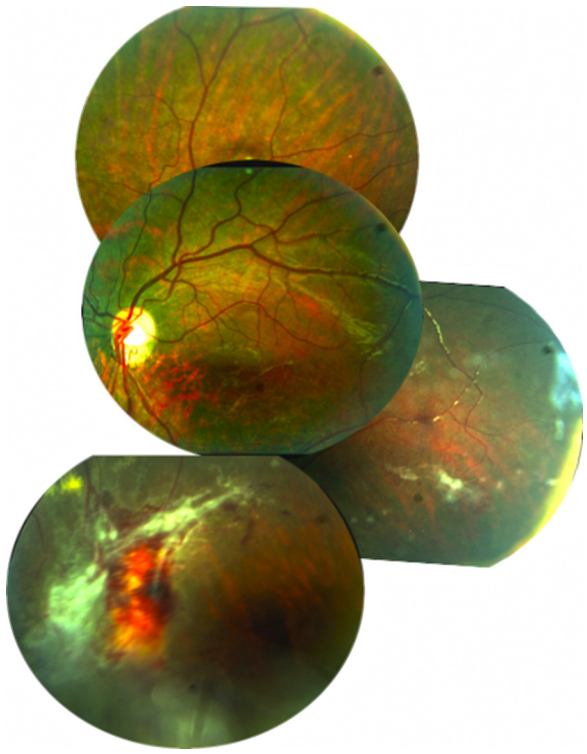

Fig. 1 – Left funduscopy with optic disk inferior coloboma, inferior retinal hemi-atrophy with foveal involvement and peripheral retinal avascularity at approximately 3–8 o'clock, with fibrovascular tissue and inferior tractional retina detachment. The proliferation exhibits hard exudate and associated subretinal dense exudate in the inferior zone.

It is believed that the presence of incomplete retinal vascularization in optic disk malformations is caused by the influence of ON development on retinal angiogenesis. The growing activity of retinal neurons during development produces physiological hypoxia that stimulates normal retinal angiogenesis. Said hypoxia "directs" the retinal vascular precursors toward the avascular periphery. Reduced ganglion cells, nerve fiber layer or other neural elements could involve less demand for oxygen and lower physiological hypoxia, leading to the alteration of this hypoxia-triggered signal.[4]

During development, axons and vascular endothelium utilize the same signaling molecules. Knockout experimental models of these molecules have exhibited anomalies in the development of both components.

On the other hand, it has been suggested that anatomical changes in the disk could also produce mechanical alterations in the migration of vascular precursors from the papilla to the retinal periphery in the angiogenesis process due to alteration in the peripheral migration of astrocytes.[3]

In a previous series with patients exhibiting ON anomalies, fibrovascular proliferation and/or TRD were diagnosed at a mean age of 46 months (range, 0–245 months).[3] In the present case, the proliferation became apparent when the patient was over 15 years old. It is possible that the congenital retinal avascularity could have caused silent ischaemia during many years and/or that puberty hormonal changes influenced the retinal

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