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Normal Pressure Glaucoma and CSF Compartmentalization Syndrome (About 3 Cases)

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Abstract:
Normal tention glaucoma and idiopathic intracranial hypertension have common characteristics, but do not clinically associate.
The physiopathological hypotheses seem to incriminate for these two pathologies a disorder of the dynamics of the cerebrospinal fluid. New data suggest that the cerebrospinal fluid that fills the spaces around the optic nerve has a composition and clean hydraulics, sometimes independent of those of other spaces of the central nervous system.
This pathological compartmentalization of the optic nerve sheath was assumed in both idiopathic intracranial hypertension and normal pressure glaucoma. The subject of this thesis enriches the literature on NTG with observations illustrating the coexistence of a NTG and compartmental syndrome documented by imaging. A review of the literature on this association is summarized in the discussion.
The present work is a retrospective study of 3 patient files presenting both "idiopathic intracranial hypertension" and "normal pressure glaucoma", whose clinical and paraclinical data were collected from patients' files collected within the ophthalmology department.

Introduction:
Normal tension glaucoma (NTG) is a set of clinical presentations that belong to the large family of open-angle glaucoma.
They raise the issue of risk factors other than intraocular pressure.
Described in 1857 by Albrecht Von Graefe as pathological cupping of the optic disc without intraocular hypertonia (IOH) [1].
Other authors have tried to redefine the NTG by integrating the notion of scalability, or a causal factor. It seems that to date, the strictly descriptive definition is the most unequivocal and the one accepted by most authors [2, 3], indeed, NTG remains a chronic anterior optic neuropathy, associating anomalies of the optic disc and subsequent perimetric alterations, accompanied by an intraocular pressure (IOP) remaining within the statistical norms. Although they differ moderately from high pressure open-angle glaucoma in their clinical expression, the only established treatment is similar, lowering intraocular pressure
Normal tension glaucoma and idiopathic intracranial hypertension have common features, but are not clinically associated.
The physiopathological hypotheses seem to incriminate for these two pathologies a disorder of the dynamics of the cerebrospinal fluid. New data suggest that the cerebrospinal fluid that fills the spaces around the optic nerve has its own composition and hydraulics, sometimes independent of those of the other spaces of the central nervous system.
This pathological compartmentalization of the optic nerve sheath has been hypothesized in both idiopathic intracranial hypertension and normal tension glaucoma.

Case report:

The present work is a retrospective study of 3 files of patients with both "idiopathic intracranial hypertension" and "normal pressure glaucoma", illustrating the coexistence of a NTG and a compartmentalization syndrome documented by imaging.

Case No. 1:

54-year-old patient diagnosed with normal tension glaucoma in 2014 and put on beta-blocker (eye drops). The patient's follow-up was marked by a rapid progression on the glaucogram with worsening of the visual field.

Faced with this rapid progression, a cerebral MRI was requested showing bilateral dilation of the peri-optic subarachnoid spaces, then a neurological opinion was sought, returning in favor of idiopathic intracranial hypertension according to Dandy'S criteria (Figure 1).

The rest of the clinical examination is unremarkable.

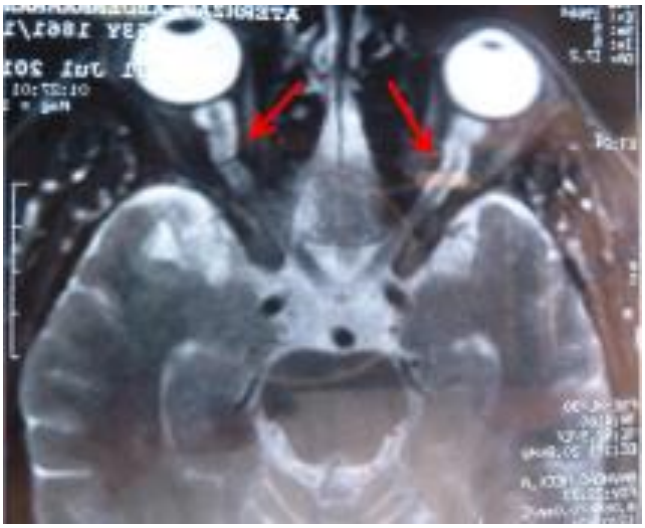

Figure 1: MRI sections showing dilation of the peri-optic sheaths (first patient)

Observation No. 2:

58-year-old patient with as an antecedent: intracranial hypertension under carbonic anhydrase inhibitor

Patient followed in neurology for 3 years for idiopathic intracranial hypertension. Referred to ophthalmology to assess the impact of his pathology on the optic nerve.

The diagnosis of normal tension glaucoma was retained after a follow-up of 5 years. The request for re-reading of the cerebral MRI initially interpreted as normal objectified a bilateral dilation of the peri-optic sheaths and an empty sella turcica.

Observation No. 3:

62-year-old patient, with a history of intracranial hypertension under carbonic anhydrase inhibitor, as well as beta-blocker.

The patient was referred to ophthalmology where the diagnosis of normal tension glaucoma was retained, an MRI was requested objectifying bilateral dilation of the optic nerve sheaths in favor of a compartmentalization syndrome (Figure 2 and 3)

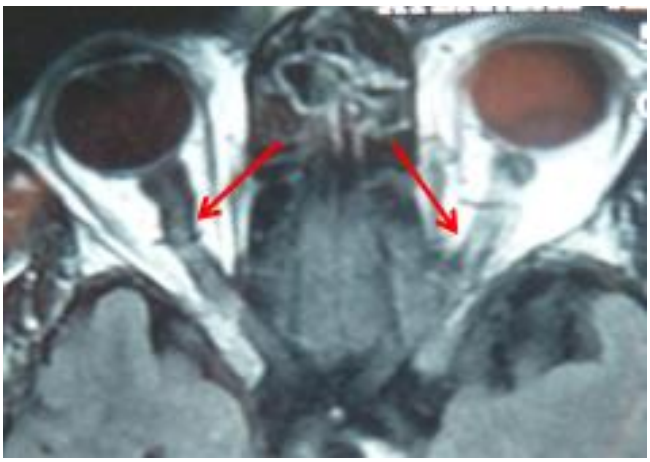

Figure 2: Orbito-cerebral MRI showing bilateral peri-optic gains dilation

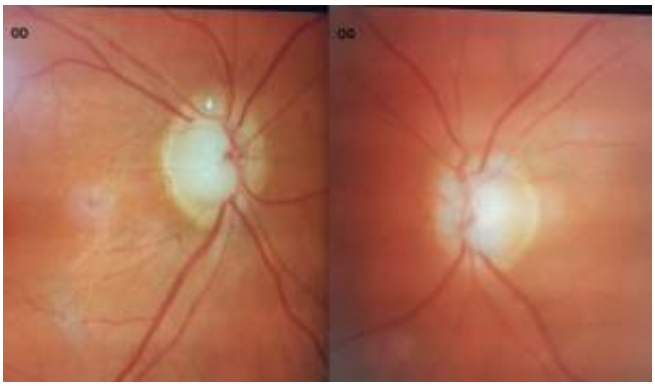

Figure 3: Control fundus after 5 years of follow-up showing asymmetric excavation

Discussion:

Glaucoma, a leading cause of blindness worldwide, is a condition characterized by damage to the optic nerve that is clinically manifested by cupping of the optic nerve head and progressive visual field defect.

Elevated intraocular pressure (IOP) has been identified as the main risk factor for progressive visual field loss and blindness. However, despite this well-documented association, some patients develop visual field loss without elevated IOP and are said to have "low pressure" or "normal pressure" glaucoma (NPG) [4].

Normal pressure glaucoma and idiopathic intracranial hypertension have common features, but are not clinically associated. The physiopathological hypotheses seem to incriminate for these two pathologies a disorder of the dynamics of the cerebrospinal fluid [5].

Through this present work we expose the hypothesis of the dysfunction of the CSF circulation at the level of the sheath of the optic nerve, in 3 subjects with normal pressure glaucoma and idiopathic intracranial hypertension. This implies a reduction in the renewal of the CSF responsible for a defect in the elimination of neurotoxic substances as well as a disturbance of the pressure of the CSF within the peri-optic meningeal sheath. We support this hypothesis with a review of the literature.

Conclusion:

This concept of optic nerve sheath compartmentalization syndrome offers an entirely novel approach to understanding the pathophysiology of visual loss in patients with idiopathic intracranial hypertension and, by extension, optic nerve atrophy in patients with normal pressure glaucoma.

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