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Short Communication

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Carotid Artery Atheromatosis Detected With Doppler Ultrasonography In Patients With Normal Tension Glaucoma

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ABSTRACT

Normal Tension Glaucoma (NTG) is an optic progressive neuropathy with intraocular pressures <21 mmHg. It is a disease with multifactorial proposed pathogenetic mechanisms, one of them being intracranial or systemic vasculopathy. Seventy-seven percent of patients with NTG showed some degree of carotid atheromatosis when scanned with carotid US-Doppler. Six percent of patients showed significant burden to carotid blood flow.

KEYWORDS: Normal tension glaucoma; Carotid artery atheromatosis; US-Doppler.

INTRODUCTION

Glaucoma is the leading cause of blindness in the world, affecting approximately 60 million people.[1] In this disease there is a progressive loss of retinal ganglion cells and their axons associating remodeling of the Optic Nerve Head (ONH). Left without control this disease produces progressive visual field deterioration in the area of anatomical ONH and Retinal Nerve Fiber Layer (RNFL). It is a multifactorial disease, with pathogenic mechanisms not fully understood. Intraocular pressure (IOP) is the most important factor in the development and progression of glaucoma, but reducing IOP does not mean ending of the disease. Some patients suffer from glaucoma progression despite low IOP.[2]

Normal Tension Glaucoma (NTG) is an optic progressive neuropathy with IOP lower than 21 mmHG, with no known cause. Diagnosis is established once other optic neuropathies have been excluded. One of the proposed pathogenetic mechanisms in the development of the disease is the reduction in ocular and cerebral blood flow. It could be a manifestation of either intracranial or systemic vasculopathy rather than an isolated orbitary process. Coexistent micro- and macro-vascular disorders have been observed in NTG patients. The impact of these alterations and the relationship between them are in part unknown.[3]

MATERIALS AND METHODS

Series of 61 patients, 18 males and 43 females with ages between 39-88 years (mean age: 63 years) referred for Carotid-Doppler US with clinical diagnosis of NTG.

Common Carotid Artery (CCA) and Internal Carotid Artery (ICA) ultrasound scan was performed using a Siemens Antares Sonoline (Elangen, Germany) platform with a V10-5 lineal probe, insonation frequency 4.71 MHz, dynamic range 55 dB and PRF set in the 3000-4000 Hz range. The scan is practiced with the patient supine, the head slightly extended with a contralateral rotation of 15-20 degrees. One of the patients was scanned sitting in her wheel chair, with the rest of the exam keeping the same parameters.

The internal carotid stenosis degree was set using criteria established by the Society of

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Radiologists in Ultrasound Consensus Conference.[4] As patients were examined with a usual clinical diagnostic method informed consent was not considered necessary.

RESULTS

Atheromatous plaques were present in 47 patients (77%). Twenty-nine patients (41%) had calcific plaques and 18 patients (29%), fibro-lipidic plaques. Two patients (3%) had complete occlusion of one of their ICA. Two patients (3%) presented significant stenosis (higher than 70%) of at least one of the ICA; 7 patients (11%) presented carotid luminal reduction between 50-69%; 50 patients (82%) had 0-49% reduction of ICA diameters.

DISCUSSION

It has been estimated that 15-25% of patients with Primary Open Angle Glaucoma (POAG) suffer from NTG. In the Baltimore Eye Study, 50% of patients with glaucomatous ONH and VF changes had IOP lower than 21 mmHG in one visit and 33% had IOP lower than 21 mmHg in two determinations. This finding is important because it influences treatment of this group of patients.[5,6]

Glaucoma patients show loss of retinal ganglion cells with thinning of the retinal nerve fiber layer, deformity of optic nerve and of the head of optic nerve. Degenerative changes in lateral geniculate nucleus and in central visual pathways have been associated. RM scans showed higher prevalence of white matter lesions and ischemic changes secondary to small vessel disease in NTG patients when compared with control groups. These findings, along with coexistence of vascular risk factors in patients with optical nerve glaucomatous lesion suggest ischemia as an important factor in the progression of glaucoma.[7]

Patients with recently diagnosed NTG present signs of subclinical vascular abnormalities at micro- and macro-vascular levels, making necessary to consider circulation system pathologies in the development and progression of this disease.[3]

Patients with suspected NTG comprised about 10% of patients referred to our Radiology Department for a US-Doppler carotid scan. An important part of them (77%) presented atheromatous lesions in carotid artery walls. About 6% of patients had significant carotid artery permeability impairment, proportion slightly greater than that published in other studies.[8] In our opinion these data make necessary screening for carotid artery lesions in patients with NTG.

CONFLICTS OF INTEREST: None.

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