

Vascular Compression of the Optic Chiasm Resembling Glaucoma-Like Visual Field Defects*

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Open-angle glaucoma with normal intraocular pressure, also known as normal-tension glaucoma, is estimated to constitute 10–25% of all glaucoma cases. NTG is a diagnostic challenge, as various lesions affecting the visual pathways including compression by normal or abnormal intracranial blood vessels can mimic its clinical findings. In recent years, several studies have shown a statistically significant percentage of patients with NTG who were shown to have compression of the optic nerves by normal or abnormal carotid arteries compared with controls or patients with high pressure glaucoma. We report two patients who initially presented with clinical signs imitating bilateral NTG, with non-progressive binasal visual field defects due to optic chiasm compression by low positioned anterior cerebral arteries. We show the radiologic findings and discuss possible explanations for these observations.

Patient Descriptions

Two cases of lateral chiasmal compression were identified in the Rambam Medical Center Neuroophthalmology outpatient clinic. Each underwent a repeated eye exam that comprised a full neuroophthalmologic status, including dilated fundus examination and automated visual field perimetry testing. Each patient had undergone brain and visual pathways imaging by magnetic resonance imaging with contrast material.

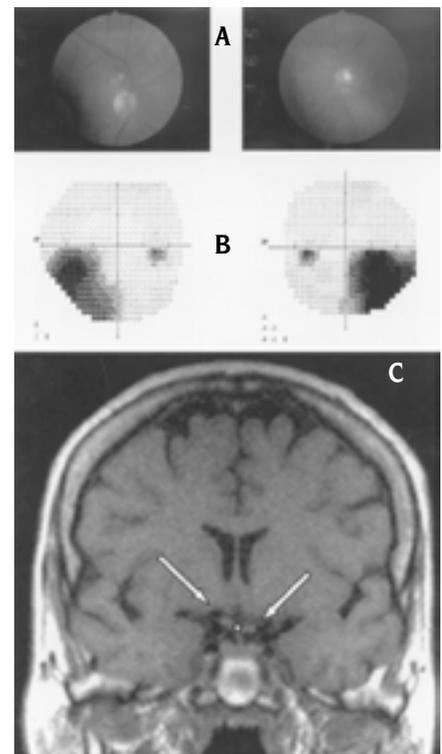
Patient 1

A 53 year old man had been under ophthalmic care some 4 years prior to his referral to our clinic for non-specific visual disturbances. The patient was a chronic smoker and suffered from arterial hypertension and type 2 diabetes. Ophthalmic examination revealed visual acuity of 6/6 without correction and applanation tonometry of 10–14 mm Hg in each eye on different measurements. Fundus examination demonstrated bilateral shallow excavation more prominent on the left side. The diagnosis at this stage was NTG. Visual field follow-up during 4 years revealed a non-progressive lower quadrant binasal defect, more prominent in the left eye. Computed tomography study of the brain was described as normal. Axial and coronal magnetic resonance imaging demonstrated a bilateral downward course of both anterior cerebral arteries abutting the lateral portions of the optic chiasm (MRI images are not shown due to technically low print quality).

Patient 2

A 64 year old diabetic woman was diagnosed as having NTG and treated with timolol 0.5% twice daily and pilocarpine 2% four times a day for 5 years. Physical examination revealed visual acuity of 6/6 in each eye; maximal intraocular pressure was 19 mmHg in each eye. A fundus examination disclosed bilateral temporal disk pallor without excavation [Figure A]. Serial visual field examinations during 5 years showed a stable binasal visual field defect in the lower quadrant, more

prominent in the left eye [Figure B]. A coronal MRI through the supracellar cistern demonstrated low positioned anterior cerebral arteries, touching the right lateral segment of the optic chiasm, and compressing the left one while pushing it



[A] Case 2. Fundus photos after 9 years of follow-up showing temporal optic disk pallor in the absence of deep or wide optic disk cupping. **[B]** Corresponding lower binasal quadrantanopia on automated visual fields testing. **[C]** A typical coronal T1-weighted MRI demonstrating the right anterior cerebral arteries (white arrows) abutting the optic chiasm (asterisk) and the pressure caused by the left carotid siphon on the left chiasmatic segment.

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NTG = normal-tension glaucoma

to a lower position than the right segment [Figure C].

Comment

NTG constitutes a significant part of any glaucoma clinic. Previous studies have reported compression of the optic nerve by normal or diseased carotid arteries in NTG. The anterior cerebral arteries pass above the dorsal surface of the optic nerves as they converge to form the optic chiasm. This close proximity between the anterior cerebral arteries and anterior visual pathways may lead to compressive effects of the vessels on the optic chiasm. This is especially true for atherosclerotic or ectatic vessels. This study described the follow-up of two patients with a diagnosis of NTG based mainly on visual field studies. The absence of progression in visual field defects led us to question the diagnosis of normal-tension glaucoma. Neuroimaging proved the visual field defects to be the result of optic pathway compression by low positioned anterior cerebral arteries.

Visual field defects resulting from vascular compression on neural optic pathways have previously been reported. Reports relevant to our findings include tortuous ectatic anterior cerebral arteries forming redundant loops into the optic chiasm, causing bitemporal superior

quadrantanopia and optic atrophy [1], anterior cerebral artery aneurysm causing unilateral compression of the optic nerve with various visual defects (unilateral hemianopia, complete anopia) [2,3], and unilateral compression of the optic chiasm causing nasal quadrantic hemianopia [4]. Visual improvement after surgical manipulation of ectatic anterior cerebral arteries has also been reported [5].

We believe that the signs and symptoms in our study were not only secondary to the close contact of the low positioned anterior cerebral arteries to the optic chiasm, but might have been, in part, the outcome of other factors such as vascular rigidity. The first patient had diabetes and arterial hypertension, and the second patient was diabetic; hence both had up to two known risk factors for the development of atherosclerosis, which results in increased arterial rigidity.

To the best of our knowledge this is the first report in the English-language medical literature on bilateral nasal visual field defects caused by bilateral optic chiasm compression by anterior cerebral arteries. Although NTG may occur in the presence of vascular compression of the optic nerves, the etiology of vascular compression by anterior cerebral artery should be considered as a rare but valid possibility in cases of binasal visual field

defects, especially when the defect is non progressive and the patient has atherosclerotic risk factors. Further large-scale epidemiologic studies are needed to estimate the prevalence of this pathology.

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