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VISUAL FIELD DEFECT DUE TO NON-ANEURYSMAL ARTERIAL CONTACT: A CASE REPORT

ANEVRİZMAYA BAĞLI OLMAYAN ARTERİAL TEMASTAN DOLAYI GERÇEKLEŞEN GÖRME ALANI DEFEKTİ: BİR OLGU SUNUMU

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ABSTRACT

In this case, we describe a patient with visual field defect caused by non-ectatic/non- aneurysmatic arterial contact. A 29-year-old woman with no significant past medical history had noticed with the complaint of visual field loss in her right eye that started 2 years ago. Visual field examination showed partial visual field defects in the right eye. Radiologic examination has revealed a close relationship of right optic nerve with the first segment of anterior cerebral artery. However no typical space-occupying mass was revealed in the magnetic resonance imaging and no aneurysmal or ectatic arterial lesion was seen in the computed tomography angiography.

Key Words: *Arterial Contact, Visual Defect, Optic Neuropathy*

ÖZET

Olgumuzda, ektazik ve anevrizmatik olmayan arteriyal temasın neden olduğu görme alanı defekti olan bir hasta sunulmuştur. Hikayesinde özellik olmayan 29 yaşında bayan hastanın 2 yıl önce başlayan görme alanı defekti mevcuttu. Görme alanı muayenesinde sağ gözde kısmi görme alanı defekti tespit edildi. Yapılan radyolojik değerlendirmelerde anterior serebral arterin ilk segmenti ile sağ optik sinir arasında yakın temas tespit edildi. Ancak manyetik rezonans görüntülemesinde yer kaplayan bir kitle yada bilgisayarlı tomografi anjiografisinde anevrizmal yada ektazik bir arteriyal lezyon görülmedi.

Anahtar Kelimeler: *Arteriyal Temas, Görme Alanı Defekti, Optik Nöropati*

Introduction

Symptomatic compressive optic neuropathy (CON) which may lead vision loss because of the increased soft tissue content of the orbit, bone structures, tumoral mass lesions and vascular abnormalities. Rarely anterior clinoid mucocele, large anterior communican artery aneurysm, ophtalmic artery aneurysm, and ectatic internal carotid artery may cause monocular vision loss due to optic nerve (ON) compression (1).

Vision loss due to compression of ectatic intracranial artery was firstly described by Caramazza in 1932 (2). According to our knowledge, compressive optic neuropathy owing to the non-ectatic/non-aneurismatic vascular contact is a rare phenomenon. In this case report we aimed to present a rare form of CON in a patient who described vision field defect due to non-ectatic/non-aneurismatic arterial contact. We also

aimed to underline efficacy of computed tomography angiography (CTA) in the diagnosis.

Case Report

A 29 year old woman was admitted to our ophthalmology clinic with the complaint of visual field loss in her right eye. Her complaints started 2 years ago. The patient described right sided spots in her visual field compatible with scotomas. A complete ophthalmologic examination and visual field test were performed based on the patient's history. Uncorrected visual acuity was 1.0 in the right eye and 0.6 in the left eye, respectively (Snellen chart).

The assessment made by auto refractometer revealed left sided myopia and astigmatism (-0.25 - 1.50 X 170°) and best corrected visual acuity in the left eye could be developed to 0.7. We thought that the visual acuity loss in the left eye was related to ametropic amblyopia. Neither biomicroscopic anterior segment evaluation nor retinal examination revealed any significant pathology. Intraocular pressures were measured 11, and 12 mmHg by applanation tonometry for the right and left eyes respectively and these values were in the normal limits. After ocular examination a visual field test was performed. Visual field examination showed partial visual field defects in the right eye, visual field of the left eye was normal with computerized perimetry (Fig. 1A-B).

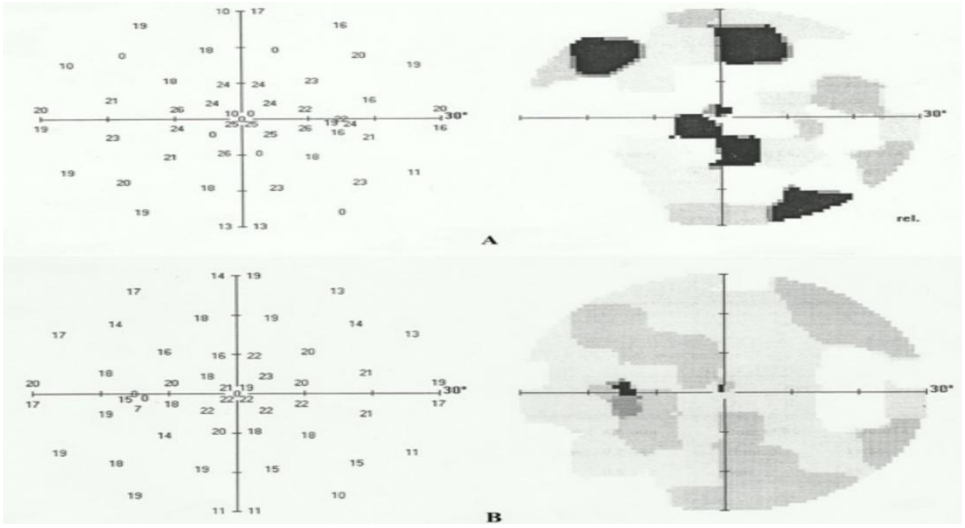

Figure 1: Visual field examination of right (a), and left eye (b). The left eye is in normal limits, but scotomas are seen in various parts of the right visual field.

After the neurological examination, a magnetic resonance imaging (MRI) was performed. In MRI a close relationship of right ON with the first segment (A1) of anterior cerebral artery (ACA) was detected. But, no typical space-occupying mass or enhancing lesion was revealed in the MRI examination (Fig. 2).

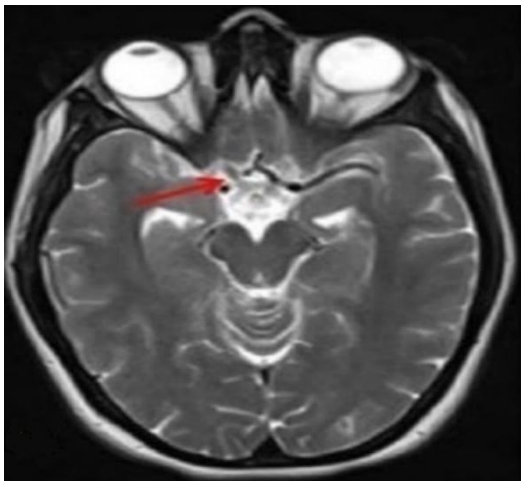

Figure 2: Axial T2-weighted MRI reveals close relationship of first portion (A1 segment) of anterior cerebral artery (ACA) with right optic nerve. The optic nerve crosses over the A1 segment of ACA (red arrow).

Multidetector computed tomography was performed for elucidate vascular structure close to right ON. CTA demonstrated chronic total occlusion of the right A1 due to direct nerve contact (Fig. 3A-B).

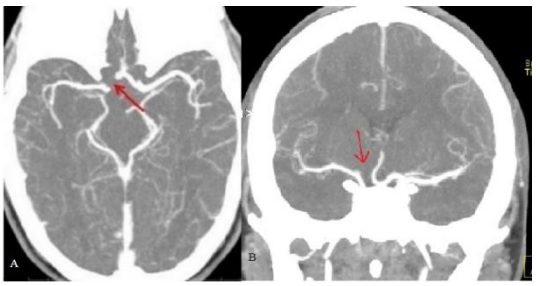

Figure 3: Axial and coronal maximum intensity projection (MIP) images obtained from source computed tomography angiography (CTA) image demonstrates total occlusion of first A1 (red arrows). Note that the A1 segment finishes as a stump. No aneurism or ectasia is detected in intracranial arterial structures.

No space-occupying mass lesion was detected during MRI and no aneurismal or ectatic arterial lesion was revealed in the CTA, the patient was prescribed eyeglass for the refraction error of the left eye and recommended 3 months visits. In the patient's control visits, any detoriation was not observed in the visual field defect.

Discussion

Asymptomatic ON contact with internal carotid artery is a common imaging finding. Jacobson et al. (3). reviewed 100 MRI of asymptomatic patients retrospectively. According to this study, 70% of all study group demonstrated unilateral or bilateral supraclinoid ICA contact on ON. However, symptomatic compression of the ON owing to ectatic internal carotid artery (ICA)-ophthalmic artery complex was rarely reported in literature (4-6).
Another form of retrobulbar optic neuropathy is caused by aneurysmal compression of intracranial arteries. Chang et al. (7) has reported CON owing to the anterior communicating artery aneurysm with CTA firstly . Bakker et al. has reported two cases with A1 aneurism that caused compression of the visual pathway (8). Interestingly all CON caused by vascular problems involves abnormaly enlarged arteries, however symptomatic contact of ON from non-aneurysmal/non-ectatic vessels is not a well-recognised entity. We belive that anatomic proximity of ACA can be an explanation as to why right optic–nerve was affected in our patient and why A1 segment of right ACA was totally occluded. Anatomically, the ON lies above the diaphragma sellae and the cavernous sinus. and ON is in close relationship with ophthalmic artery inferolaterally, internal carotid artery laterally and anterior cerebral artery superiorly (9). Direct contact of A1 to right ON caused total occlusion. But interestingly, our patient did not experienced a stroke involving the territory of the ACA.

Digital subtraction angiography (DSA) is considered the most sensitive tool for detection of intracranial vascular pathologies. But, this method does not supply additional information about the brain and nerve parenchyma and DSA is an invasive procedure provided by arterial catheter insertion which means additional risk to the patient. We have successfully demonstrated vascular status of the patient non-invasively as described in the literature (10).
Clinically Multiple Sclerosis (MS) was considered in the differential diagnosis, however a demyelinating processes was ruled out by normal signal intensity of white matter and we did not detected pathologic contrast enhancement in post-contrast MRI series supporting MS. Radiologically hypoplastic A1 segment of ACA can be considered in differential diagnosis, but in this situation no vision field defect could be expected.

In conclution ectatic or aneurysmal symptomatic ON compression is an expected clinical situation. Rarely, ON may compress A1 segment of ACA which leads total occlusion and this rare phenomenon can affect both optic nerve and A1. Cross sectional imaging techniques including MRI and particularly CTA give valuable information about the vascular reason of CON.

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Conflict of interest

The authors declare no conflict of interest.