

Case Report

Optical Coherence Tomography Angiography in Chiasmitis

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Keywords

Optical coherence tomography angiography · Neuro-ophthalmology · Multimodal imaging · Chiasmal optic neuritis · Bitemporal hemianopia

Abstract

A 29-year-old girl presented complaining of acute bilateral visual loss associated with mild headache and retrobulbar pain. She was diagnosed with chiasmal optic neuritis caused by multiple sclerosis. Her visual acuity and visual field defect promptly improved after steroid therapy. However optical coherence tomography angiography showed a progressive reduction of superficial capillary plexus density of the retina and optic nerve consistent with the progressive impairment of the retinal ganglionar cell layer. Contrary to chiasmal compression, in chiasmal optic neuritis, the superficial capillary plexus density reduction is diffuse and does not reflect the peculiar anatomy of the chiasm.

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Introduction

Optical coherence tomography angiography (OCTA) is mainly used for the study of retinal diseases, but it is being recognized as a useful tool in assessing the vascular alterations of optic disc and retina in optic neuropathies. We report a case of a patient affected by chiasmal

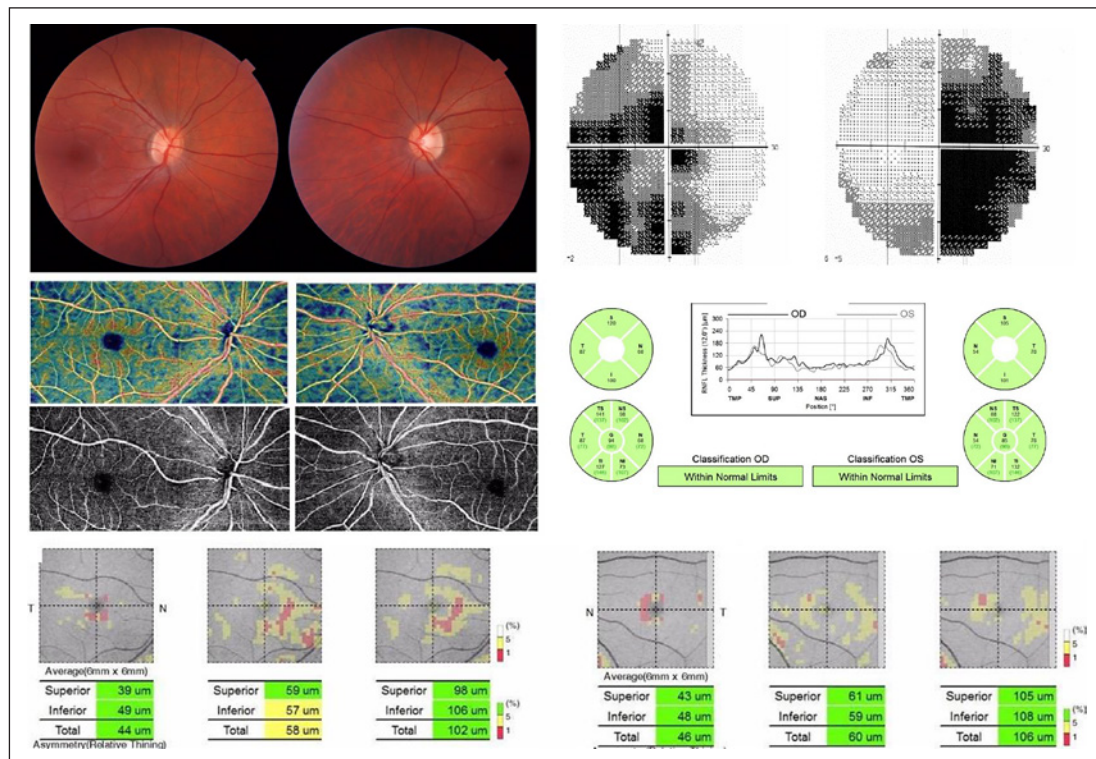


Fig. 1. At presentation, fundus (upper left) is normal. Standard computerized perimetry (upper right) shows a bitemporal hemianopia. RNFL (middle right) is normal OU, while ganglion cell complex (GCC) (bottom row) shows mild bilateral peripapillary impairment. Superficial capillary plexus as seen in OCTA of the right eye and left eye. Densitometric map is shown.

optic neuritis caused by multiple sclerosis (MS) studied by means of OCTA. Possible explanations for OCTA findings are discussed.

Case Report

A 29-year-old woman developed acute bilateral visual loss, mild headache, and retrobulbar pain for 1 week. Past medical, social, surgical, and family histories were noncontributory. She was taking no medications. On examination, the best corrected visual acuity was 20/40 in each eye (OU). Color vision was impaired (5/14 OU on Ishihara color plates). Ocular motility was full. The pupils were equal with no relative afferent defect but were sluggishly reactive to light OU. Slit lamp biomicroscopy and fundus examination were unremarkable. The optic nerve showed greater excavation in the left eye. In previous visits, this difference in terms of cup/disc between the two eyes had already been reported, we believe it is an anatomical conformation. Automated perimetry (Humphrey visual field) showed a bitemporal hemianopia consistent with chiasmal disease; Optical coherence tomography (OCT) of the peripapillary retinal nerve fiber layer (RNFL) was normal OU; the first OCTA scan of retina and optic nerve was considered normal (shown in Fig. 1). Contrast brain magnetic resonance imaging (MRI) revealed chiasmal enhancement (shown in Fig. 2). She was treated with IV methylprednisolone (1,000 mg/day for 3 days) with resolution of her symptoms, improvement of the visual field defect, and a best corrected visual acuity of 20/20 OU after 1 month. Serum aquaporin-4 (AQP4) channel antibody for neuromyelitis optica was negative. Cerebrospinal

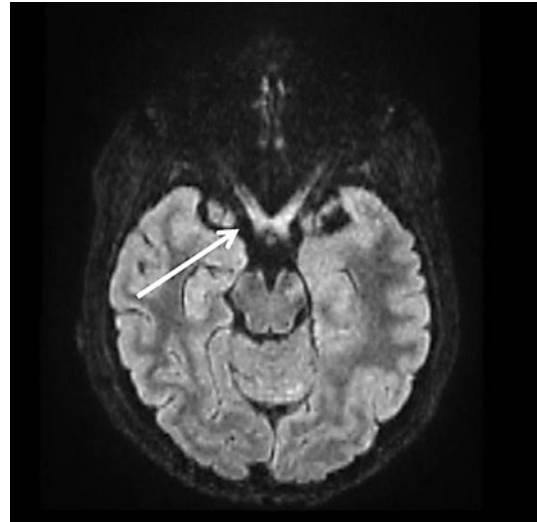


Fig. 2. Brain MRI at presentation shows high signal at the level of the chiasm (arrow).

fluid was positive for oligoclonal bands consistent with MS. Serial multimodal imaging and OCTA of the retina and optic nerve were performed for a period of 3 years. Over time, however, there was progressive reduction of the superficial capillary plexus in the macula and progressive loss of the ganglion cell complex and RNFL (shown in Fig. 3) despite 20/20 visual acuity OU and stable ocular examinations otherwise.

Discussion

In 1975, Bell documented the first case of a pathologic correlation between MS and chiasmatic involvement by exploratory craniotomy [1]. In 1987, Rosenblatt reported chiasmatic neuritis (CN) using MRI [2]. CN like optic neuritis is commonly associated with inflammatory and demyelinating disorders like MS. Patients with CN typically present with bilateral simultaneous or rapidly sequential visual loss, a bitemporal visual field defect, and a normal fundus initially. Contrast enhancement of the optic chiasm is often seen on MRI but is sometimes absent [3].

The visual prognosis of CN is usually favorable after corticosteroid treatment. In one retrospective study of 20 consecutive patients with CN at a single institution, 40% later developed MS [4]. While MRI is the imaging modality of choice for a radiological accurate evaluation of the optic chiasm, ophthalmologists nowadays have a rich armamentarium for the functional and anatomical evaluation of the optic nerves and retina of these patients. Moreover, the study of the circulation of the optic nerve head and parafoveal areas of the eye in MS patients may provide insight into the role of the more global vascular changes in the pathogenesis of MS.

Different methods have been used to detect the ocular blood perfusion in clinical practice and experimental research. Fluorescein angiography and indocyanine green angiography provide qualitative evaluation of retinal and choroidal circulation but do not provide objective quantitative measurements. As a noninvasive imaging technique, OCT has emerged as a research and clinical tool in MS. OCT has demonstrated retinal structural defects in MS including thinning of the RNFL and retinal ganglion cell and inner plexiform layers [5]. These changes are due to retrograde axonal degeneration of the optic nerve axons after clinically apparent and subclinical ON, and possibly due to primary neurodegenerative pathology. OCT technology can provide retinal and papillary structural information.

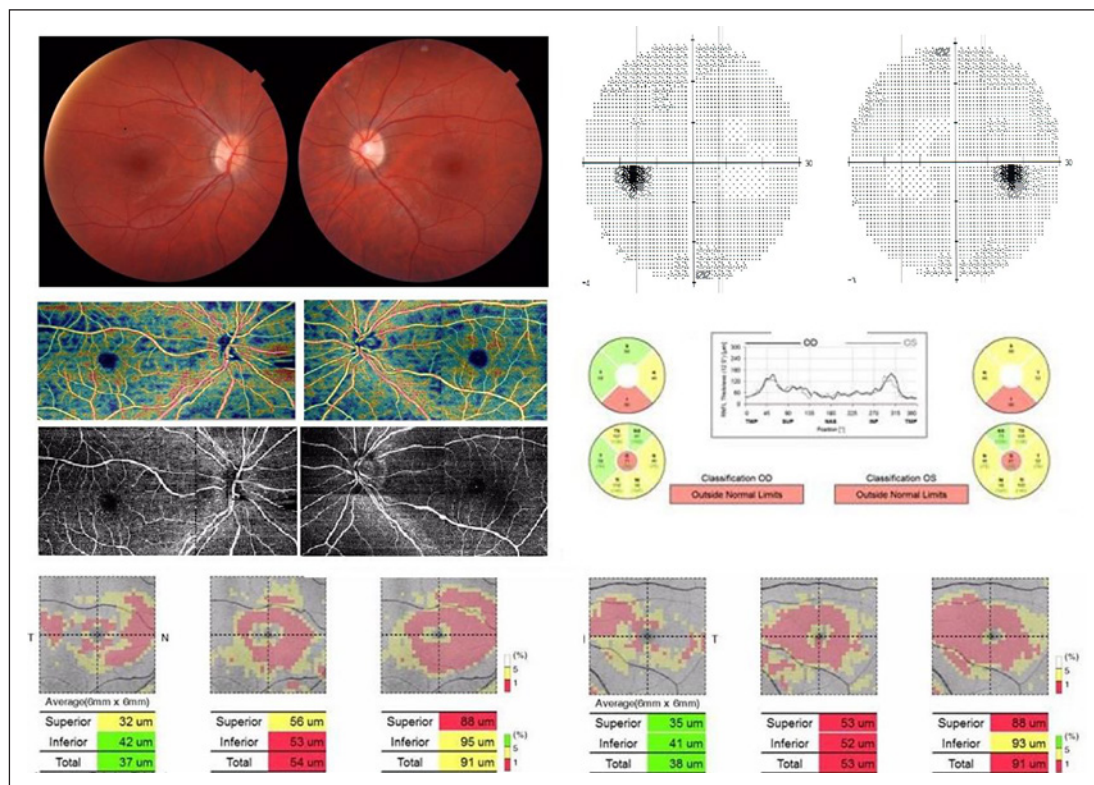


Fig. 3. At 3 years, fundus examination (upper left) shows a mild temporal pallor of the optic nerves. Standard computerized perimetry (upper right) is normal. Both RNFL thickness (middle right) as well as GCC (bottom row) studies show a bilateral impairment (more evident in the left eye). OCTA at 3 years show reduction in the capillary density of the superficial capillary plexus of the right eye and left eye also evident in the densitometric map.

An emerging new technology, OCTA, has the capability of evaluating the density of the retinal and nerve capillary and choroidal network in much more detail than traditional fluorescein angiography [6]. OCTA detects particle (blood cell) movement that can be used to construct microvascular density maps [7, 8]. A number of reports indicate that there is decreased optic disc perfusion and peripapillary capillary density on OCTA in patients with chronic glaucoma [9–11] and other optic neuropathies [12–16]. Chen et al. [12] demonstrated RNFL thinning and decrease in the peripapillary capillary density on OCTA in various optic neuropathies. The authors suggested that the decrease in peripapillary retinal capillary flow density was secondary to optic nerve injury and subsequent RNFL loss as a secondary effect. Decreased optic nerve blood flow on OCTA has also been reported in optic neuritis [13], autoimmune optic neuritis, nonarteritic ischemic optic neuropathy, dominant optic atrophy, and Leber hereditary optic neuropathy [14]. Mase et al. [17] found that the radial peripapillary capillary density is proportional to the RNFL thickness in the superficial peripapillary retina of normal eyes, supporting the notion that the radial peripapillary capillaries are the vascular network responsible for supplying the RNFL. In these cases, chronic optic neuropathy leads to a reduction in RNFL which, in turn, reduces metabolic need and reduced or absent capillary blood flow through a neurovascular coupling mechanism.

OCT also allows a detailed analysis of macular ganglion cell complex that has been reported [18] to be superior to measurements of RNFL in optic neuropathy (e.g., Leber hereditary optic neuropathy, neurodegenerative disease, MS, and NAION). Macular ganglion cell

complex analysis also demonstrates high correlation with perimetry and might serve as an early structural indicator of irreversible neuronal loss [18]. Higashiyama et al. [19] reported OCTA in 4 patients with chiasmal compression and found a decrease in peripapillary retinal perfusion in the quadrants of the visual field defects due to chiasmal compression. This finding can be easily explained by the peculiar anatomic structure of the chiasm in which the nasal fibers of each optic nerve decussate. Therefore, a mass compressing the chiasm would cause these fibers to suffer from compression producing the classic bitemporal visual field defect.

In contrast to these OCTA findings in compressive chiasmopathy, although a classic bitemporal field defect was found in our case of CN, we did not observe a selective peripapillary or retinal capillary plexus density reduction as would be expected by a selective damage of the crossing nasal fibers composing the chiasm. We hypothesize that demyelinating CN may have been more diffuse producing more generalized optic nerve damage than in compressive chiasmal disease. More cases and further studies are needed to evaluate the role of OCTA in CN. Our case demonstrated diffuse capillary plexus density reduction on OCTA over time in CN, and to our knowledge, this is the first such case in the English language ophthalmic literature.

Statement of Ethics

The patient involved in the present study gave her written informed consent to publish the case (including publication of images). Ethical approval is not required for this study in accordance with local guidelines. Our research is in accordance with the World Medical Association Declaration of Helsinki.

Conflict of Interest Statement

The authors have no conflicts of interest to disclose.

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Author Contributions

Alessandra Cuna as first and corresponding author made substantial contributions to the conception; the design of the work; the acquisition, analysis, and interpretation of data, and the final revision of the paper; Francesco Pellegrini, Emanuela Interlandi, Erika Mandarà, and Alessandro Papayannis made substantial contribution to the design, analysis and interpretation of data of the work, and the final revision of the paper; Marco De Luca, Rocco De Marco, Cristina Ciabattini, and Antonio Zappacosta contributed by the final revision of the paper. All authors approved the submitted version and have agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even those in which the author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature.

Data Availability Statement

Research data are not publicly available on legal or ethical grounds. All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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