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Dégénérescence transsynaptique rétrograde démontrée in vivo par Tomographie en Cohérence Optique (OCT) lors d'atteinte des voies visuelles rétrogéniculées

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"Dégénérescence transsynaptique rétrograde démontrée in vivo par Tomographie en Cohérence Optique (OCT) lors d'atteinte des voies visuelles rétrogéniculées"

THESE

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DOCTEUR EN MEDECINE

par

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Résumé

Une atteinte axonale résulte en une dégénérescence axonale antérograde et rétrograde et aboutit à l'apoptose du corps de la cellule ganglionnaire. Une atteinte des voies visuelles antégéniculées (chiasma optique, bandelettes optiques ou corps genouillé latéral) sera responsable de l'apparition retardée d'une certaine atrophie des deux nerfs optiques, visible lors de l'examen du fond d'œil. Par contre, il était classiquement admis qu'une atteinte des voies visuelles rétrogéniculées (radiations optiques, cortex occipital) ne s'accompagnait pas d'atrophie des nerfs optiques.

Cependant, une atteinte axonale peut parfois induire une dégénérescence transsynaptique rétrograde (TRD) avec dégénérescence d'un axone non lésé initialement et apoptose de sa cellule ganglionnaire. Expérimentalement, une telle dégénérescence transsynaptique rétrograde a été observée après des lésions sélectives des voies visuelles rétrogéniculées, avec une très nette perte homonyme des cellules ganglionnaires rétiniennes (RGCL) [1,2]. Malgré ces évidences expérimentales, la présence de TRD chez l'humain est restée sujet à controverse et il était admis qu'une lésion rétrogéniculée chez l'humain ne pouvait provoquer une atrophie optique que si la lésion survenait très tôt dans la vie [3,4].

La tomographie par cohérence optique (OCT) permet la mesure de l'épaisseur des différentes couches rétiniennes in vivo [5]. Ce dispositif permet au clinicien de détecter des variations d'épaisseur significatives au niveau de la rétine et de ses différentes couches, avec une sensibilité supérieure à l'examen du fond de l'œil par l'ophtalmoscope. Nous nous sommes posés la question de savoir si un phénomène de TRD pouvait être détecté chez l'humain après une lésion purement rétrogéniculée. Ce travail de thèse est constitué de deux articles (peer-reviewed) publiés entre 2015 et 2016. Le premier sous forme de « case report » décrit un amincissement sectoriel homonyme des cellules ganglionnaires maculaires suite à une lésion acquise) du cortex occipital. Le deuxième article, résume les résultats d'une étude rétrospective portant sur des patients ayant présenté une lésion isolée acquise des voies visuelles rétrogéniculées, confirmée par imagerie cérébrale. Tous les patients avaient bénéficié d'un champ visuel computérisé et d'un examen par OCT pour mesurer l'épaisseur des cellules ganglionnaires (RGCL) et des fibres nerveuses péripapillaires (RNFL). Etant donné la nature purement rétrogéniculée des lésions cérébrales, confirmée par imagerie cérébrale, la présence d'un amincissement homonyme du RGCL ne peut résulter que d'un TRD.

En conclusion, le phénomène de TRD existe chez certains patients avec lésions acquises des voies visuelles rétrogéniculées. Cette évidence est donnée par l'OCT, permettant de visualiser in vivo l'épaisseur de la couche des cellules ganglionnaires rétiniennes. Pourquoi certains patients présentent ce phénomène, et d'autres pas, reste pour l'instant inexpliqué.

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Homonymous Ganglion Cell Layer Thinning After Isolated Occipital Lesion: Macular OCT Demonstrates Transsynaptic Retrograde Retinal Degeneration

Paolo G. Meier, MD, Philippe Maeder, MD, Randy H. Kardon, MD, PhD, François-Xavier Borruat, MD

Abstract: A 48-year-old man was examined 24 months after medial and surgical treatment of an isolated well-circumscribed right occipital lobe abscess. An asymptomatic residual left homonymous inferior scotoma was present. Fundus examination revealed temporal pallor of both optic discs, and optical coherence tomography (OCT) revealed mild temporal loss of retinal nerve fiber layer in both eyes. No relative afferent pupillary defect was present. Assessment of the retinal ganglion cell layer demonstrated homonymous thinning in a pattern corresponding to the homonymous visual field loss. There were no abnormalities of the lateral geniculate nuclei or optic tracts on review of the initial brain computed tomography and follow-up magnetic resonance imaging. We believe our patient showed evidence of transsynaptic retrograde degeneration after an isolated right occipital lobe lesion, and the homonymous neuronal loss was detected on OCT by assessing the retinal ganglion cell layer.

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Transsynaptic retrograde degeneration (TRD) of the visual pathways, that is loss of undamaged retinal ganglion cells occurring upstream from a retrogeniculate lesion, has been convincingly reported in experimental conditions (1–3). Some patients with occipital lesions sparing the lateral geniculate nucleus and optic tract can develop TRD with bow tie optic atrophy in the eye contralateral to the lesion and temporal pallor in the fellow eye (4). However, optic nerve changes can be subtle and sometimes overlooked on ophthalmoscopy. Histological evidence of TRD in humans was provided by Beatty et al (5) who reported such a case 40 years after occipital lobectomy. The development of optical coherence tomography (OCT) allows measurement of the retinal nerve fiber layer (RNFL), and this technology has provided evidence of TRD after isolated occipital lesions (6–8). Additional evidence of TRD with occipital lesions has been demonstrated with magnetic resonance imaging (MRI), revealing atrophy of the ipsilateral optic tract (9,10).

The development of segmentation software of macular OCT provides thickness maps of the retinal ganglion cell layer–inner plexiform layer complex (RGCL-IPL). This approach is advantageous for discerning patterns of homonymous loss corresponding to TRD from postgeniculate lesions. We report homonymous thinning of RGC-IPL in the macula of a patient with an isolated occipital lobe abscess producing a corresponding homonymous visual field scotoma within the central 15°.

CASE REPORT

A 46-year-old man in good health reported a 10-day history of headaches and scintillating scotomata. The patient was afebrile with normal vital signs, and general physical examination was normal. Visual acuity was 20/20 bilaterally but visual field examination revealed a dense left homonymous hemianopia (Fig. 1). There was no relative afferent pupillary defect (RAPD). Ophthalmoscopy was normal. Brain computed tomography disclosed a well-demarcated lesion within the right occipital lobe with perifocal edema thought to represent a neoplasm (Fig. 2).

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The authors report no conflicts of interest.

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Transsynaptic Retrograde Degeneration: Clinical Evidence with Homonymous RGCL Loss on OCT

Darstellung von transsynaptischer retrograder Degeneration bei homonymem RGCL-Verlust mittels OCT

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Key words
- retrogeniculate lesion
- homonymous retinal ganglion cell loss
- homonymous hemianopia
- spectral domain optical coherence tomography

Schlüsselwörter
- retro-genikuläre Läsion
- homonymer Ganglienzellenverlust
- homonyme Hemianopsie
- Spektral-Domänen Optische Kohärenztomografie

Abstract

Background: Retinal thinning after a retrogeniculate lesion (transsynaptic retrograde degeneration) was first described 50 years ago, but has long been a controversial issue. It is now possible to use OCT for the in vivo measurement of retinal thickness.

Material and Methods: This was a retrospective study of patients with homonymous visual field loss, with SD-OCT assessment (RNFL and RGCL measurements) in isolated retrogeniculate lesions, subsequently confirmed by a neuroradiologist.

Results: Nine patients with vascular, inflammatory or tumour brain lesions were included in the study. Homonymous RGCL thinning was found in all patients, and correlated with the visual field defect. No correlation was found with RNFL.

Conclusions: The homonymous defect of RGCL in patients with retrogeniculate lesions demonstrates the presence of transsynaptic retrograde degeneration. RGCL is a better predictor of visual field defects than RNFL measurement.

Zusammenfassung

Hintergrund: Die makuläre Dickenabnahme als Folge einer retrogenikulären Läsion wurde vor 50 Jahren beschrieben, war aber lange Zeit umstritten. Heutzutage ermöglicht das OCT die Netzhautdicke in vivo zu messen.

Patienten und Methoden: Retrospektive Studie von Patienten mit homonymem Gesichtsfeldverlust, SD-OCT Untersuchung (RNFL und RGCL Messung) bei neuroradiologisch isolierten retrogenikulären Läsionen.

Ergebnisse: Neun Patienten mit vaskulär-, entzündungs- oder raumforderungs- bedingten Hirn Läsionen wurden ausgewertet. Ein homonymer RGCL-Verlust, korrelierend zum Gesichtsfeldverlust, war in allen Individuen vorhanden. Die entsprechenden RNFL Messungen waren weniger aussagekräftig.

Schlussfolgerungen: Ein homonymer RGCL-Verlust, bei rein retrogenikulären Läsionen, weist auf eine transsynaptische retrograde Degeneration hin. Die Messung der RGCL gibt bessere Rückschlüsse auf Gesichtsfelddefekte als die RNFL-Analyse.

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Background

Neuronal degeneration after axonal lesions occurs in both directions, toward the cell body (retrograde degeneration) and toward axon terminal (anterograde or Wallerian degeneration). Usually, insult to the axon of a neuron results in neuronal death limited to the injured cell.

Transsynaptic retrograde degeneration (TRD) occurs when, following the death of an injured neuron, a previously intact neuron connecting to the injured one will transsynaptically undergo changes (atrophy, apoptosis). Retinal ganglion cells (RGC) send their axons to the lateral geniculate body where they synapse with neurons sending their axons to occipital primary visual cortex. For the ophthalmologist, it is well known that optic atrophy will be visible during fundus examination several weeks/months after injury to either optic nerve, optic chiasm, optic tract or lateral geniculate body. On the other hand, lesions affecting only the retrogeniculate pathways should not induce visible optic nerve atrophy at fundus examination, even years later, unless TRD occurs. TRD was demonstrated experimentally more than fifty years ago, but clinical evidence of TRD was debated up to recently [1–8]. Recent advances in OCT technology allow to image in vivo the retinal nerve fibers and RGC layers (RNFL, RGCL). Patients with antegeniculate lesions demonstrate loss of

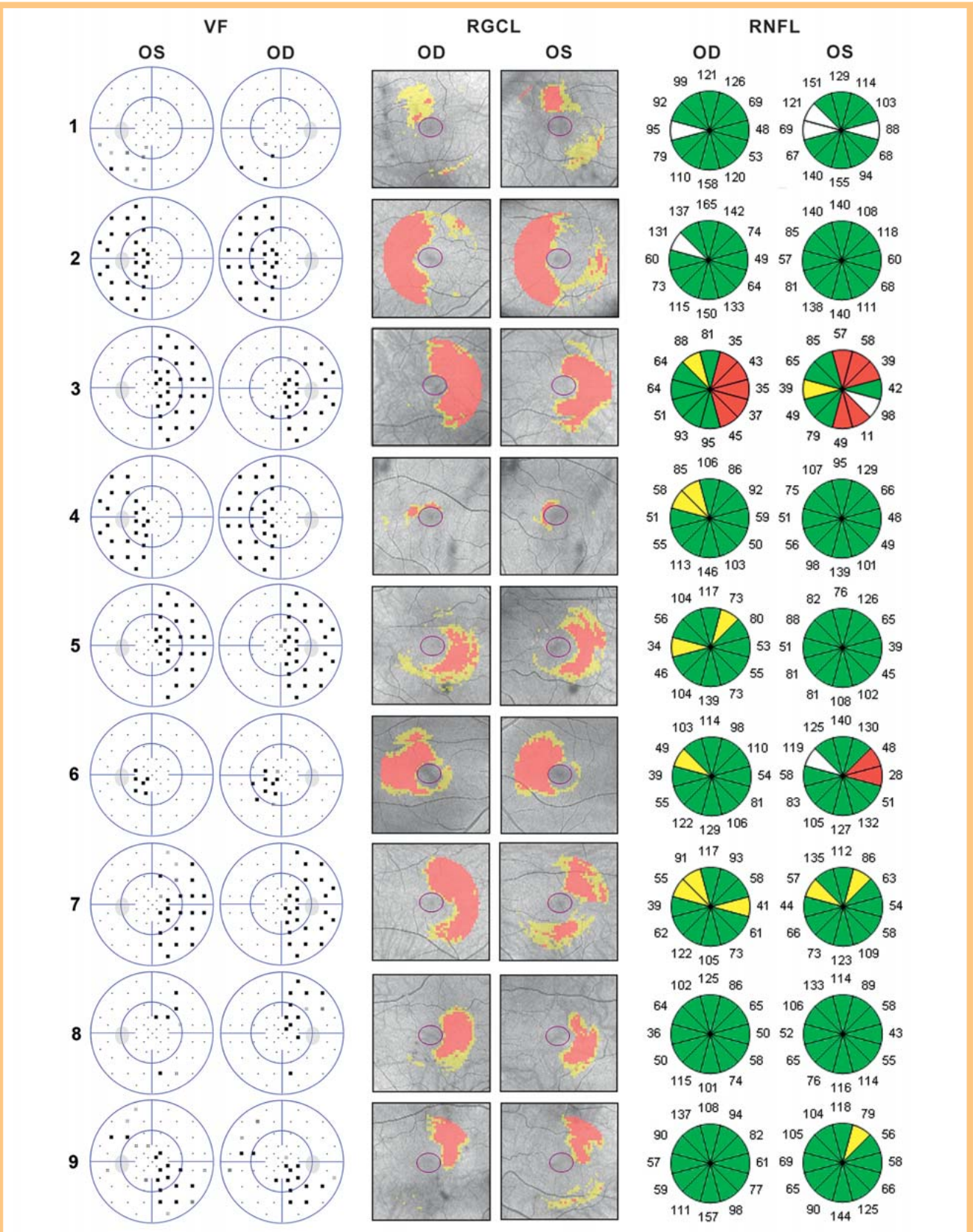

Fig. 1 Results of automated perimetry and OCT. Left two columns – Visual fields (VF) are displayed as seen by the observer. Right VF (OD) is on the right and left VF (OS) is on the left. Only the corrected probability plots are displayed (Octopus, Haag Streit AG, Schlieren bei Köniz). Middle two columns – OCT deviation maps of the retinal ganglion cell layer (RGCL) is displayed as seen in the fundus. All patients exhibited homonymous thinning of RGCL (see text). Right two columns – Clockhours retinal nerve fiber layer (RNFL) measured by OCT is displayed as seen in the fundus. Normal values are in green, borderline values in yellow and values outside the normal range in red. Four results were completely normal (Patient 1,2,8,9) and only one (Patient 3) showed a frank homonymous loss of RNFL.

both RNFL and RGCL, but the homonymous character of the loss is frequently more obvious for RGCL loss than for RNFL loss. In vivo, loss of RGCL might result from TRD in patients with retrogeniculate lesions, and homonymous loss of RGCL should be detectable by OCT.

We report a series of 9 TRD patients who presented with homonymous RGCL loss resulting from an isolated unilateral retrogeniculate lesion.

Material and Methods

We performed a retrospective chart review of patients satisfying the following inclusion criteria: homonymous visual field (VF) defects, SD-OCT with RNFL and RGCL measurements (Cirrus 4000 HD-OCT; Carl Zeiss Meditec, Dublin, CA), and isolated retrogeniculate lesion. The study variables were: age, sex, etiology of CNS lesion. Neuroimaging was reviewed by one of us (PM) to ensure that the lesion was purely retrogeniculate. Exclusion criteria were: MRI unavailable for review, evidence of geniculate or antegeniculate lesions, and presence of any other maculopathy or optic neuropathy.

Results

Nine patients met all the inclusion criteria. There were six men and three women, mean age was 50 years (range 27–78). Etiology of CNS lesion included: stroke (4), trauma (1), haemorrhage from arterio-venous malformation (1), tumour (1), cortical abcess (1) and multifocal progressive leukoencephalopathy (1). The median delay between diagnosis and OCT was 23 months, ranging 3–64 months. For the two patients examined at 3 and 6 months, the diagnosis was tumour and arterio-venous malformation, implying that the duration of visual pathway dysfunction was possibly longer than 3 and 6 months. Homonymous VF loss was right-sided in five patients and left-sided in four others. Six patients presented homonymous hemianopia and 3 homonymous quadrantanopia. All patients presented with RGCL loss in a homonymous pattern which was ipsilateral to the side of the retrogeniculate lesion. The extent of RGC loss was variable amongst patients and roughly depended on the amount of VF loss with some exceptions (more VF loss than RGCL loss in patients 4 and 5). Normal RNFL was found in 4 patients (Patients 1,2,8,9) and the pattern of RNFL loss was frankly homonymous in only one patient (Patient 3) (**Fig. 1**).

Conclusions

Transsynaptic retrograde degeneration has been recognized for more than 50 years from experimental data and was clinically suspected but challenged for several years [1–8]. With the development of modern OCT, fast and precise evaluation of retinal thickness, as well as retinal segmentation, is now available to the clinician.

Our study demonstrated homonymous RGCL thinning in all patients presenting with pure retrogeniculate lesions. There was no age, sex or etiology predilection for developing TRD. These were also the results of recent publications [9,10]. Although all our patients exhibited a homonymous pattern of RGCL loss, the extent of homonymous VF loss could not be readily predicted from the extent of RGCL loss and vice versa. Most patients showed a good correlation between the degree of VF loss and the extent of RGC loss, but Patients 4 and 5 exhibited a greater loss of VF than what could be found by OCT. Both patients were examined relatively early after the onset of retrogeniculate lesion. As the time course of TRD has not been determined yet, it is then possible that further loss of RGC will be noticed later on.

Assessment of RNFL was less useful in our patients as RNFL was found to be normal in four patients and a clear homonymous loss of RNFL was present in only one patient (**Fig. 1**.). In our series RGCL seems to be more sensitive than RNFL to assess TRD. In other pathologies such as optic neuritis or compression of the pregeniculate visual pathway, it is our experience that RGCL assessment provides, in some patients, not only earlier information regarding visual pathway dysfunction, but also is more sensitive to detect such dysfunction than RNFL.

Transsynaptic retinal degeneration is not as rare as previously thought, but does not happen in every patient with purely retrogeniculate lesions. Larger case series or, better, prospective studies could help determine not only the possible variables influencing the development of TRD, but also the true incidence and the time course of TRD.

Conflict of Interest

None.

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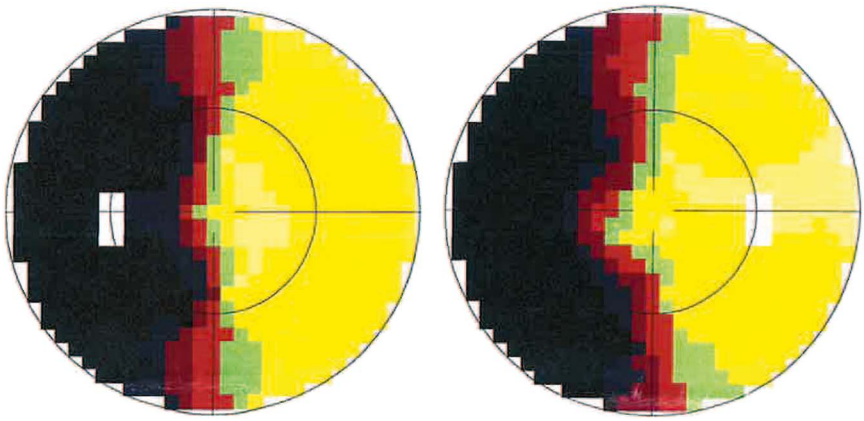

FIG. 1. Automated static perimetry (Octopus, program G1, Haag Streit, Schlieren, Switzerland) reveals a left homonymous hemianopia.

The patient was treated with intravenous methylprednisolone (125 mg 3 times a day) for 48 hours followed by craniotomy. At surgery, a cortical abscess was drained, and bacteriologic analysis revealed *Actinomyces Meyeri* and *Aggregatibacter aphrophilus*. Systemic intravenous antibiotic therapy (ceftriaxone 2 g twice a day and metronidazole 0.5 g 3 times a day) was given for 6 weeks. The patient was found to have a patent foramen ovale that was surgically closed. Six weeks after surgery, ophthalmic examination revealed partial resolution of the left homonymous hemianopia, absence of a RAPD, and no evidence of optic atrophy. Twelve months after initial presentation, MRI showed right occipital cortical atrophy with mild ipsilateral white matter gliosis (Fig. 3). Review of all MRI studies revealed no evidence of damage to the lateral geniculate nuclei or optic tracts.

Two years later, the patient was referred for neuro-ophthalmic evaluation. Visual acuity was 20/15 in each eye, pupils reacted normally, and color visual testing and slit-lamp examination were normal. There was temporal pallor of both optic discs (Fig. 4). Automated visual field testing showed a congruous left inferior homonymous congruous scotoma (Fig. 5).

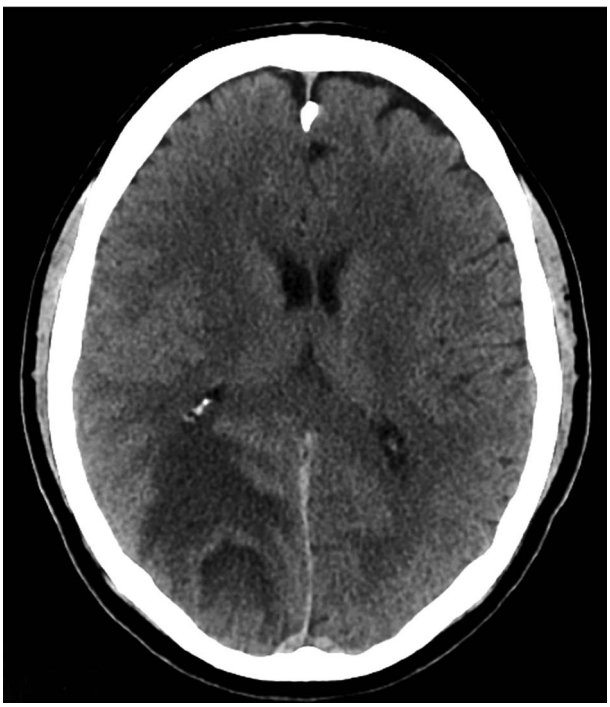

FIG. 2. Axial computed tomography shows a right occipital lobe lesion with surrounding edema.

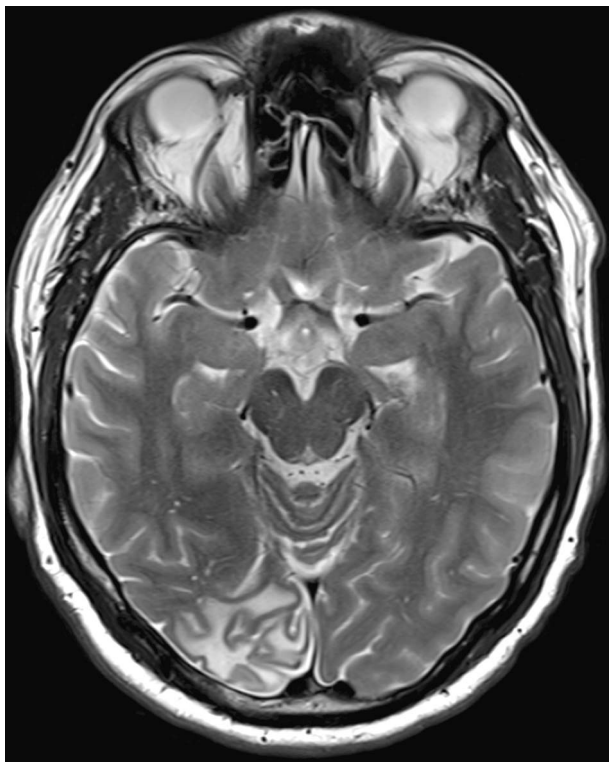

FIG. 3. T2 axial magnetic resonance imaging 12 months after draining of brain abcess shows cortical atrophy of the right occipital lobe with moderate gliosis.

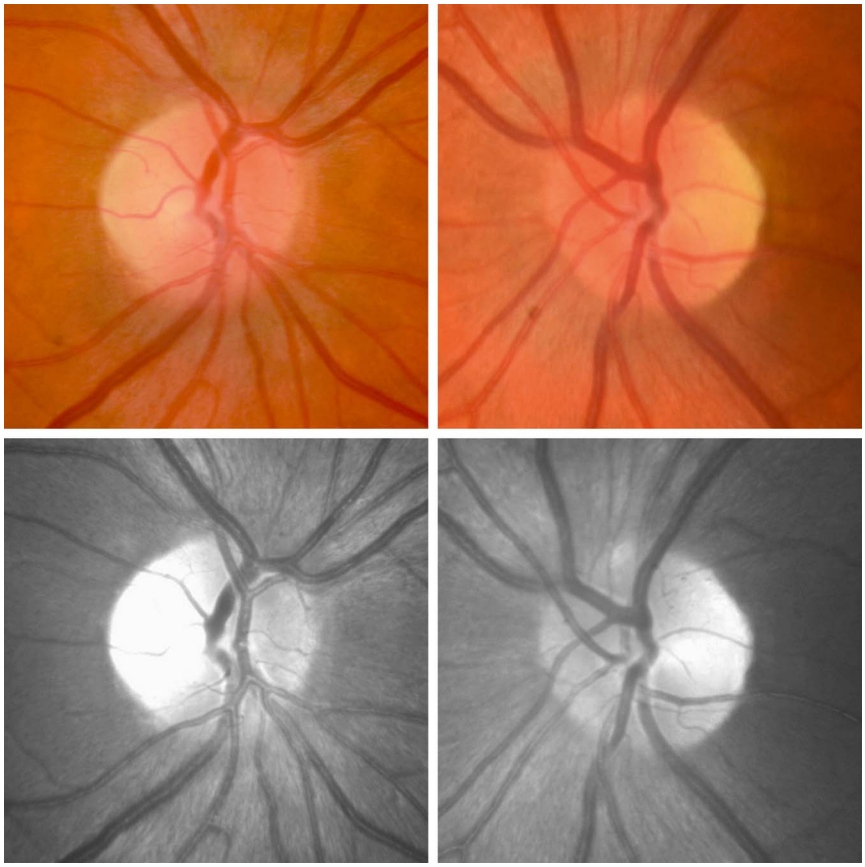

FIG. 4. Two years after initial presentation, color (*top*) and red-free (*bottom*) fundus photographs shows temporal pallor of both optic discs.

Using spectral-domain OCT (Cirrus 4000 HD-OCT; Carl Zeiss Meditec, Dublin, CA), average RNFL thickness was normal in both eyes (right eye: 95 μm; left eye: 98 μm) with slight thinning of temporal quadrant in each eye. Macular OCT showed no significant thinning of the total retinal thickness. Analysis of the RGC-IPL complex with the Ganglion Cell Analysis module (Zeiss Cirrus software, version 6.5.0; Zeiss, Dublin, CA) disclosed a moderate homonymous thinning in both eyes on the total thickness RGC-IPL map and significant to less than the 1% level on the probability map (Fig. 6). The pattern of the RGCL thinning was homonymous, affecting the temporal retina in the right eye and the nasal retina in the left eye. The RGC-IPL thinning respected the vertical meridian. Although the homonymous visual field defect extended to approximately 16°, ganglion cell thinning appeared to extend to approximately 6°.

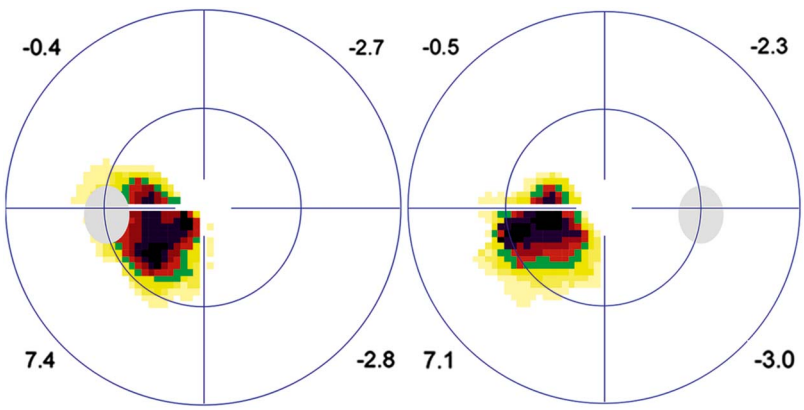

FIG. 5. Two years after initial presentation, automated static perimetry (Octopus, program G1, Haag Streit) discloses a residual left inferior homonymous scotoma.

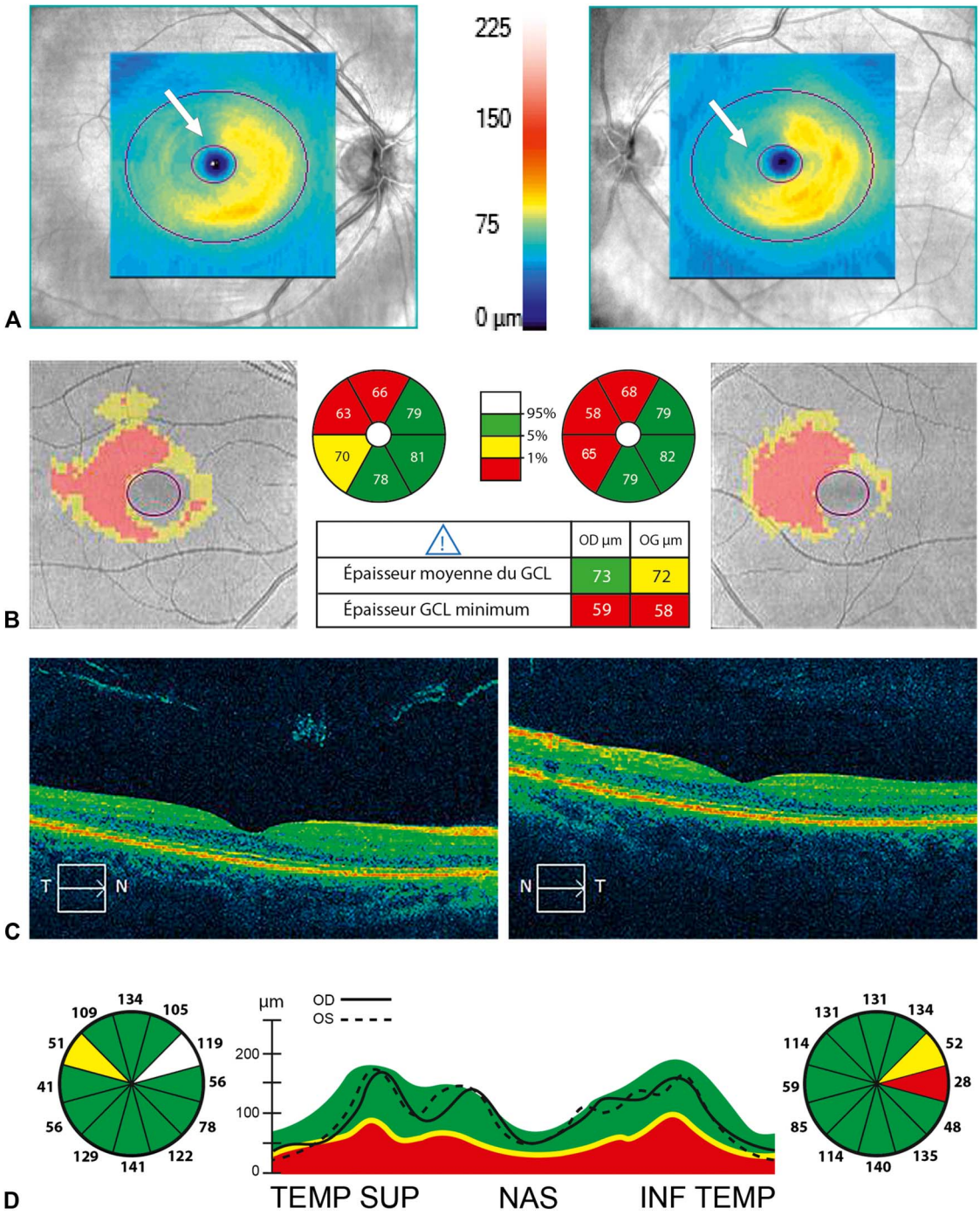

FIG. 6. Optical coherence tomography performed 2 years after initial presentation (Cirrus 4000 HD-OCT; Carl Zeiss Meditec). **A**. Fundus diagram with the thickness map of RGCL-IPL for right eye (*left*) and left eye (*right*). Homonymous thinning of RGCL-IPL complex (i.e., loss of RGCL-IPL in the temporal retina of the right eye and in the nasal retina of the left eye indicated by *arrows*) is present in a pattern corresponding to the residual homonymous left visual field defect. **B**. Deviation maps of the RGCL-IPL loss demonstrates the homonymous defect of RGCL-IPL. Note that the extent of the RGCL-IPL thinning on the deviation maps (approximately 6°) does not extend as far peripherally as the visual field defect (16°). Sector maps illustrated the degree of homonymous RGCL-IPL thinning in each eye. **C**. Macular profiles with horizontal OCT B-scans through the fovea appear normal in both eyes. **D**. RNFL analysis shows loss of nerve fibers in the temporal quadrant of both optic discs more marked in the left disc. RGCL-IPL, retinal ganglion cell layer–inner plexiform layer complex; OCT, optical coherence tomography; RNFL, retinal nerve fiber layer.

DISCUSSION

The combination of bilateral optic atrophy and homonymous visual field loss usually implies the presence of a lesion of the pregeniculate visual pathways. Typically, this results in temporal atrophy of the optic disc ipsilateral to the lesion and a bow tie atrophy of the contralateral eye. This pattern arises from loss of noncrossing fibers in the ipsilateral eye and the loss of crossing fibers in the contralateral eye (4). The presence of an RAPD in the contralateral eye indicates a lesion of the optic tract, whereas the absence of an RAPD signifies a lesion beyond the point where the pupillomotor fibers leave the optic tract.

OCT can demonstrate thinning of the RNFL in optic tract lesions, but the pattern of RNFL loss does not readily demonstrate a homonymous nature of the lesion. A bow tie pattern of RNFL loss also is not usually apparent. The availability of software to provide segmentation of the retinal layers in the macula allows the analysis of the RGC-IPL complex. Homonymous thinning of the RGC-IPL in a patient with an optic tract lesion due to neuromyelitis optica has been published (11).

Our patient had a right occipital lobe abscess. Both before and after neurosurgical drainage, neuroimaging studies did not detect any abnormality of the optic tracts or lateral geniculate nuclei. The homonymous thinning of the RGC-IPL on OCT exhibited by our patient most likely represents TRD. Interestingly, the peripheral extent of the homonymous ganglion cell-inner plexiform layer thinning (6°) on the probability plot was not as extensive as the visual field defect (16°). Possibly, the central macular fibers near fixation are more likely to show TRD.

More than 50 years ago, Van Buren (1) created a focal surgically-induced lesion in 1 occipital lobe of the adult macaque monkey. Forty-eight months later, he demonstrated homonymous thinning of the retina supportive of the concept of TRD. Similar experiments in other nonhuman primates confirmed this observation (2,3). However, documentation of optic atrophy in humans after retrogeniculate lesion is rare, and the concept of TRD in human adults has been challenged (12). However, recent studies with OCT have demonstrated the possibility of TRD in adult humans with acquired retrogeniculate disorders. Jindahra et al (6) demonstrated thinning of RNFL in both eyes in a series of 26 patients with both congenital and acquired homonymous hemianopia. Patients exhibited a significant loss of RNFL mean thickness as compared to controls, and those with congenital lesions exhibited greater RNFL loss than individuals with acquired lesions. In a later study (7), the same investigators studied 38 patients with purely acquired retrogeniculate lesions and reported a decelerating rate of RNFL loss of 9.08 μm per log-years. Yet, no thinning of RNFL was detected when small visual field defects were present. Park et al (8) reported a constant pattern of RNFL loss in patients with cerebral infarction with the optic nerve ipsilateral to the lesion losing uncrossed (temporal) fibers and the fellow eye losing crossed (nasal) fibers.

In a cohort of patients with autosomal-demonstrated optic atrophy, Rönnbäck et al (13) showed that measurement of RGCL-IPL complex was more sensitive than RNFL thickness in detecting structural loss. The OCT results in our patient support a similar conclusion in patients with TRD due to a retrogeniculate lesion.

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