Established in 1871

Swiss Medical Weekly

Formerly: Schweizerische Medizinische Wochenschrift

An open access, online journal • www.smw.ch

Original article | Published 17 September 2020 | doi:10.4414/smw.2020.20319
Cite this as: Swiss Med Wkly. 2020;150:w20319

Homonymous visual field defects in patients with multiple sclerosis: results of computerised perimetry and optical coherence tomography

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Summary

AIMS OF THE STUDY: Visual dysfunction is frequent in multiple sclerosis, usually resulting from retrobulbar optic neuritis or papillitis. Less frequently, demyelinating lesions can affect the retrochiasmal pathways. There are few reports of homonymous visual field defects (HVFD) in multiple sclerosis and little is known about their evolution. The purpose of this study was to better define both the clinical profile and the evolution of HVFD in patients with multiple sclerosis.

METHODS: We performed a retrospective study of all multiple sclerosis patients who presented HVFD and were examined by automated static perimetry. A subset of patients benefited from macular assessment with optical coherence tomography (OCT). We also reviewed the worldwide literature on the subject.

RESULTS: Twenty patients were retrieved from the neuro-ophthalmology database of the Hôpital Ophtalmique Jules-Gonin. There were 11 women and 9 men, and their average age was 35 ± 11 years. The relapsing-remitting form of multiple sclerosis was most common (18/20; 90%), the primary progressive form (1/20; 5%) and the secondary progressive form (1/20; 5%) were rare. HVFD were the presenting symptom of multiple sclerosis in seven patients (35%). The recovery was complete in 12/20 patients (60%), and the median time to recovery was 10 weeks (2-13 weeks). An incomplete recovery was found in 5/20 subjects (25%) and no recovery occurred in 3/20 subjects (15%). Magnetic resonance imaging disclosed a definite lesion explaining the HVFD in 7/11 patients: five within the optic radiations (71.4%), one within the optic tract (14.3%) and one within the lateral geniculate nucleus (14.3%). Our results were comparable to those compiled from our literature search (29 publications, totalling 70 cases). A recurrent episode of HVFD occurred in three patients (15%). OCT was performed in 10/20 patients. Retinal ganglion cell layer thickness was assessed and revealed a homonymous thinning in three patients, diffuse unilateral or bilateral thinning (resulting from previous episodes of optic neuritis) in six patients, and normal retinal ganglion cell layer thickness in one patient.

CONCLUSION: HVFD in multiple sclerosis are found mostly in young patients with relapsing-remitting multiple sclerosis, which is consistent with the epidemiology of multiple sclerosis. HVFD can be the first manifestation of multiple sclerosis and have a relatively good prognosis. Like optic neuritis, HVFD can recur. The incidence of HVFD in multiple sclerosis is unknown, as it is probably underdiagnosed. Systematic automated static perimetry and OCT could help to determine the true incidence of HVFD in multiple sclerosis.

Keywords: *multiple sclerosis*, *homonymous*, *hemianopsia*, *quadrantanopsia*, *scotoma*, *retrochiasmal visual pathway*, *OCT*

Introduction

Multiple sclerosis is an autoimmune inflammatory disorder characterised by demyelinating lesions of the central nervous system. The visual pathways are frequently involved. Whereas the antechiasmatic pathways are most commonly affected, resulting in either retrobulbar optic neuritis or papillitis, demyelination can affect any part of the visual pathway [1]. Approximately 30% of patients with multiple sclerosis present with optic neuritis/papillitis as the inaugural symptom of their disease, and 80% of patients will develop an optic neuritis along the course of their disease [2]. Although chiasmal syndrome has been reported in multiple sclerosis patients, it is rare [3]. Symptomatic retrochiasmal lesions of the visual pathways, resulting in homonymous visual field defects (HVFD), have been reported since 1890 [4]. However, there are only three published case series, which report five, eight and 18 cases of HVFD in multiple sclerosis respectively [5–7]. All other publications deal with single case reports. The frequency of HVFD in multiple sclerosis has been estimated at between 1.3% and 3.5% [5, 8–11]. Paradoxically, despite the low frequency of HVFD in multiple sclerosis, MRI and autopsy studies have shown that the retrochiasmal pathways are frequently affected by demyelinating lesions in multiple sclerosis (30-90%), with the periventricular area being the most common location of demyelinating lesions [8, 10]. According to Plant et al., the size of the demyelinating lesions is a predictor of HVFD, with only the largest

Author contributions
F-XB. designed and directed the study. LS collected the data. F-XB and LS analysed the data and wrote the manuscript.

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lesions resulting in symptomatic HVFD [7]. The discrepancy between clinical data (low frequency of HVFD) and anatomical data (high incidence of retrochiasmal demyelinating lesions found by either autopsy or magnetic resonance imaging [MRI]) suggests that either most retrochiasmal lesions are asymptomatic, or that HVFD may be underdiagnosed in patients with multiple sclerosis [10, 12].

As well as MRI, nowadays there is another method to detect evidence of previous demyelinating retrochiasmal lesions: optical coherence tomography (OCT). Spectral-domain OCT is a relatively novel, noninvasive technique which allows the retinal structures to be assessed with a high resolution of 5-10µm. Furthermore, retinal segmentation permits the isolation and analysis of separate layers of the retina, namely the retinal ganglion cell layer. Any lesion along the afferent visual pathway can result in RGCL thinning. All antegeniculate lesions, i.e. optic nerve, optic chiasm, optic tract and lateral geniculate body, will result in retrograde axonal degeneration, resulting in a thinning of the RGCL [13, 14]. Experimental and clinical studies have also shown that transsynaptic retrograde degeneration can occur after lesions of the postgeniculate visual pathway [15–20]. However, retrograde transsynaptic degeneration is not present in all patients, is not associated with any particular pathology, and its exact mechanism is not yet elucidated.

In order to better characterise both the clinical profile and the evolution of HVFD in patients with multiple sclerosis, we performed a retrospective study of such patients. We also compared our results with those compiled from a search of the worldwide literature on the subject.

Patients and methods

The study was ethically approved by the "Commission cantonale d'éthique de la recherche sur l'être humain" (CER-VD).

We analysed the charts of all multiple sclerosis patients who presented HVFD and were examined in the Neuro-Ophthalmology Unit of the Hôpital Ophtalmique Jules-Gonin. Patients were retrieved from the neuro-ophthalmology database and anonymised data were extracted from their clinical charts. The period of recruitment ranged from 1994 to 2016. From 2013, all patients were investigated with spectral-domain OCT (Cirrus 5000, Carl Zeiss AG, Oberkochen, Germany). Segmentation of the macular OCT allowed measurements of the retinal ganglion cell layer (RGCL). All patients were examined by one of the study authors (FXB). Inclusion criteria were a diagnosis of multiple sclerosis and at least one episode of HVFD, documented by static automated perimetry (Octopus 300, program G1 or M2, Haag-Streit AG, Köniz, Switzerland). The diagnosis of multiple sclerosis was independently established according to the results of a clinical examination by a neurologist and the results of both MRI and lumbar puncture. The exclusion criterion was the presence of another pathology which could have contributed to the HVFD.

We extracted information on general demographics (age and gender), visual function at the time of the episode of HVFD (visual acuity; colour vision; visual field) and specific information on multiple sclerosis (date of diagnosis; type of multiple sclerosis, i.e. relapsing-remitting, primary progressive, secondary progressive) from the medical charts. Previous episodes of optic neuritis were recorded. Snellen visual acuity (VA) was converted into logMar scores for statistical purposes [21]. Normal visual acuity was defined as ≤ 0.0 logMar (≥10/10). Colour vision was tested using Ishihara pseudo-isochromatic plates and normal colour vision was defined by a score ≥ 11/13. Automated static perimetry data was analysed both qualitative and quantitative, at initial examination and also at follow-up visits.

We categorised HVFD into three types: homonymous hemianopsia, homonymous quadrantanopsia and homonymous scotoma. These homonymous defects could be either complete or partial. Recovery from the HVFD, on follow-up visual field examination, was either complete (no HVFD), partial (improvement of HVFD) or absent (no improvement of HVFD). Recurrences of HVFD and the results of cerebral MRI were also recorded when available.

Literature review

We performed a literature review of HVFD in multiple sclerosis. We searched relevant papers on Pubmed and Google Scholar using the following keywords "multiple sclerosis, homonymous hemianopsia, homonymous quadrantanopsia, homonymous scotoma, visual homonymous defect, homonymous visual field defect, homonymous defect, visual pathway lesion, optic tract lesion, lateral geniculate body lesion, optic radiation lesion, retrochiasmal lesion". Whenever possible, we retrieved from the article the following data: age and gender of the patients; type of multiple sclerosis; whether the HVFD was the first manifestation of multiple sclerosis; type of HVFD, its severity and evolution; location of the associated lesions.

Results

In the neuro-ophthalmology database, 547 patients were coded as "multiple sclerosis" and 44/547 were also coded as "HVFD". Amongst the 44 selected patients, we excluded 24 patients due to either another cause of the HVFD found on MRI, absence of follow-up, or lack of computerised visual field examination. Twenty patients were included in this study (table 1). There were 11 women (55%) and 9 men, and their median age was 35 years (range 16–52). Relapsing-remitting multiple sclerosis was the most common type (18/20; 90%), whereas primary progressive multiple sclerosis (1/20; 5%) and secondary progressive multiple sclerosis (1/20; 5%) were less frequent. In seven patients (35%), the HVFD was the inaugural symptom of multiple sclerosis. For the remaining thirteen patients, the HVFD occurred at a median time of 5.5 years (range 1 month to 16 years) after the diagnosis of multiple sclerosis. An HVFD was found fortuitously in five patients without any visual symptoms.

The results of central visual function tests at initial examination are summarised in table 2. Visual function of eyes without a previous history of optic neuritis (30 eyes) was normal for both visual acuity and Ishihara, whereas visual acuity and color vision were abnormal for the group of 10 eyes with previous episodes of optic neuritis.

Eleven patients (55%) presented with homonymous quadrantanopsia, seven patients (35%) exhibited homonymous hemianopsia and two patients (10%) showed homonymous

scotoma (fig. 1). An example of each type of homonymous defect is shown in figure 2. The majority of patients (17/20; 85%) presented with a partial HVFD. One patient exhibited a bilateral partial HVFD.

Results of MRI at the time of the HVFD were available for only 11 patients. Seven of them exhibited a lesion explaining the visual field defect. Lesions were present within the optic radiations (5/7; 71.4%), the optic tract (1/7; 14.3%) and the lateral geniculate nucleus (1/7; 14.3%).

Macular spectral-domain OCT was obtained for 10 patients (all examined after 2013). Analysis of RGCL thickness revealed three patients with homonymous thinning of the RGCL, six patients with diffuse unilateral or bilateral thinning of the RGCL resulting from previous episodes of optic neuritis, and one patient with normal RGCL thickness.

All patients benefited from follow-up automated static perimetry examinations. Follow-up duration varied from two weeks to 11 years, with a median time of 12 weeks. The recovery was complete in 12 patients (60%), and the median time to complete recovery was 10 weeks (2–13 weeks). One patient recovered completely but the time to full recovery could not be determined as the patient was examined only four years after the initial episode of HVFD. Incomplete recovery was documented in five subjects (25%) after a median follow-up time of 28 weeks (16–58 weeks). No recovery was found in three subjects (15%), despite a long median follow-up time of 17 weeks (10 weeks to 7.6 years). Worsening of the HVFD was documented in one of these three patients, who had a significant increase in mean defect during the follow-up period. Examples of VF recovery are depicted in figure 3. Overall, evolution of the HVFD was favourable in 85% of our patients.

A recurrent episode of HVFD occurred in three patients (15%). Each of these three patients had recovered from the initial episode when the second one occurred. All three patients eventually fully recovered.

Case report of Patient 10 (patient previously but partially reported [22])

A 28-year-old woman woke up with painless bilateral visual disturbance. Examination revealed a normal visual acuity and colour vision, but automated static perimetry revealed a left homonymous inferior quadrantanopsia. Cerebral MRI revealed a lesion on the right posterior thalamic region, also affecting the right optic radiations. A

Table 1: Demographics of multiple sclerosis (MS) patients with homonymous visual field defects (HVFD).

Pt	Age	Sex	MS	HVFD as presenting symptom	Previous optic neuritis	VA (/10)		Ishihara (/13)		HVFD	Recovery		Recurrence
						RE	LE	RE	LE		Type	Within	
1	44	F	RR	–	R+L	7	7	8	1	PHH R	C	10 weeks	–
2	25	F	RR	+	R+L	5	2	7	1	PHQ R	C	12 weeks	–
3	41	F	RR	–	–	10	10	11	11	PHH L	C	3 weeks	–
4	22	F	RR	–	–	9	9	12	11	PHQ R	X	10 weeks	–
5	16	F	RR	–	–	10	10	13	13	PHQ L	C	2 weeks	I
6	35	M	RR	–	–	10	10	13	12	CHH R	P	58 weeks	–
7	37	M	RR	+	–	15	12.5	12	12	PHQ R	C	12 weeks	–
8	49	F	RR	–	–	12	12	13	13	PHQ L	P	16 weeks	–
9	35	M	PP	–	L	12	1	11	0	PHH R	P	28 weeks	–
10	28	F	RR	+	–	10	10	13	13	PHQ L	C	12 weeks	CI
11	28	M	RR	–	R	10	10	13	13	CHQ R	C	13 weeks	–
12	51	F	RR	–	R	6	8	10	13	PHQ L	C	4 weeks	–
13	52	F	RR	+	–	10	12	13	13	CHH L	P	34 weeks	CI
14	41	M	RR	–	L	12	12	13	13	HS R	C	12 weeks	–
15	24	M	RR	+	–	10	10	13	13	PHH L	C	10 weeks	–
16	33	M	RR	+	–	10	10	13	13	HS R	C	3 weeks	–
17	47	M	SP	–	–	10	8	12	11	PHQ R+L	X	92 months	–
18	26	M	RR	+	–	10	10	13	13	PHH R	C	NA	–
19	47	F	RR	–	L	12	10	12	1	PHQ R	X	16 weeks	–
20	28	F	RR	–	–	10	10	11	11	PHQ L	P	28 weeks	–

C = complete recovery; CHH = complete homonymous hemianopsia; CHQ = complete homonymous quadrantanopsia; CI = contralateral; F = female; HS = homonymous scotoma; I = ipsilateral; L = left; LE = left eye; M = male; NA = not available; PHH = partial homonymous hemianopsia; P = partial recovery; PHQ = partial homonymous quadrantanopsia; PP = primary progressive; presenting symptom = HVFD as presenting symptom of MS; Pt = patient; R = right; RE = right eye; RR = relapsing-remitting; SP = secondary progressive; VA = visual acuity; X = no recovery

Table 2: Visual function of patients with multiple sclerosis and homonymous visual field defects.

	All eyes (n = 40)		Eyes without ON (n = 31)		Eyes with previous ON (n = 9)	
	Right eye	Left eye	Right eye	Left eye	Right eye	Left eye
Average VA (LogMAR)	+0.01 ± 0.10	+0.10 ± 0.30	0.00 ± 0.00	0.00 ± 0.00	+0.17 ± 0.11	+0.39 ± 0.48
Median VA (LogMAR)	0.00	0.00	0.00	0.00	0.18	0.14
Min–max VA (LogMAR)	+0.30 to −0.18	+1.18 to −0.09	+0.05 to −0.18	+0.10 to −0.09	+0.30 to 0.00	+1.18 to −0.08
Average Ishihara (/13)	12 ± 2	10 ± 5	12 ± 1	12 ± 1	10 ± 2	3 ± 5
Median Ishihara (/13)	12.5	12.5	13	13	9	1
Min–max Ishihara (/13)	7–13	0–13	11–13	11–13	7–13	0–13

ON = optic neuritis; VA = visual acuity

pseudotumoural form of multiple sclerosis was diagnosed. The visual field defect spontaneously resolved after three months (fig. 4). Two years later, she presented a recurrent episode of HVFD with a normal visual acuity and colour vision. An MRI showed two lesions in the white matter of the right hemisphere (periventricular region and optic radiations). Four months later, she had fully recovered. Four years after the inaugural HVFD, the patient was again symptomatic. She presented bilateral complete inferior visual field defect and MRI revealed bilateral lesions within the optic radiations. Recovery was again complete after four months. Thickness of the RGCL was normal in both eyes.

Literature results

Our literature search found 29 publications, totalling 70 patients with multiple sclerosis and HVFD [4–7, 11, 22–46]. The gender of the patients was known in 57 cases. There were 35 women (61.4%) and 22 men. Their median age was 30 years (range 19-57). The type of multiple sclerosis was described in 56 cases: the relapsing-remitting type was the most common (98.2%) and only one case of primary progressive type was described. An HVFD was the inaugural symptom of multiple sclerosis in eight patients (11.4%).

Figure 1: Visual field and optical coherence tomography findings in multiple sclerosis patients with homonymous visual field defects.

Pt	Time 0				Time R				OCT performed 5.4yrs after HVFD (0-12; median 5yrs)
			Mean Defect (db)				Mean Defect (db)		
	VF LE	VF RE	RE	LE	VF LE	VF RE	RE	LE	
1			NA	NA			NA	NA	NA
2			11	8.7			1.9	3.0	NA
3			3.3	3.9			2.3	2.5	NA
4			3.9	3.7			3.4	2.7	NA
5			3.6	2.9			0.4	0.9	
6	VF by confrontation						10.1	13.4	
7			6.4	3.1			-1.5	0.1	NA
8			1.1	0.6			0.6	0.4	NA
9			9.6	22.5			2.3	11.5	
10			4.5	4.6			0.3	-0.1	
11			7.6	10.5			1.0	1.6	NA
12			5	3.5			0.8	0.0	NA
13			16.6	17.9			13.8	17	
14			1.2	4.3			-1.0	-0.7	NA
15			3.2	6.3			2.7	3.0	
16			3.3	3.5			1.4	1.6	NA
17			3	5.8			5.1	8.9	
18			4	1.1			0.4	-0.7	
19			3.5	8.7			5.2	8.1	
20			7.6	8.6			5.1	3.8	

Pt = patient, VF = visual field, RE = right eye, LE = left eye, Time 0 = time of HVFD, Time R = time of recovery of HVFD
NA = not available, OCT = Optical Coherence Tomography

Figure 2: Examples of homonymous visual field defects in patients with multiple sclerosis. Top – right partial homonymous hemianopsia (30° visual field, program G1). Middle – partial right homonymous quadrantanopsia (30° visual field, program G1). Bottom – right paracentral inferior homonymous scotoma (central 10° visual field, program M2). MD = mean defect expressed in decibels (dB).

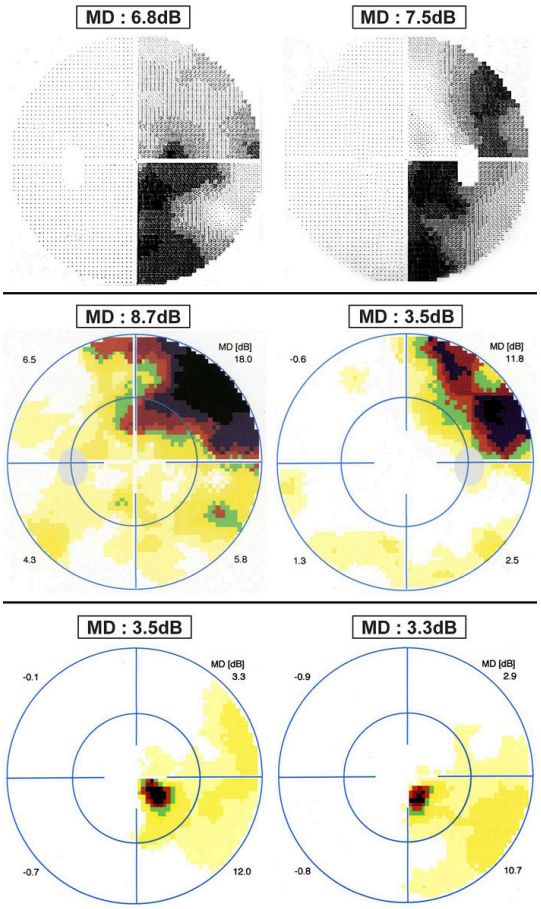
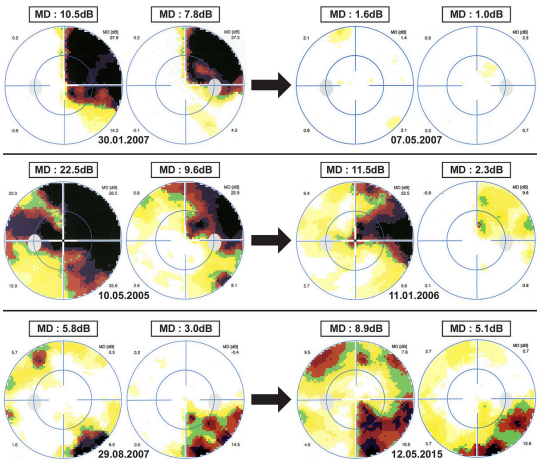


Figure 3: Examples of the types of evolution of homonymous visual field defects. Top – complete recovery of a right homonymous quadrantanopsia in 13 weeks (30° visual field, program G1). Middle – partial recovery of a partial right homonymous hemianopia after 28 weeks (30° visual field, program G1). Bottom – no recovery of a right inferior homonymous quadrantanopsia after more than 7 years (30° visual field, program G1). MD = mean defect expressed in decibels (dB).



Fifty-four patients had an HVFD detected by manual kinetic perimetry and 11 patients were examined with automated static perimetry. For the remaining five patients, no information was found regarding perimetric techniques. Forty-three patients (61.4%) showed homonymous hemianopsia, 13 patients (18.6%) exhibited homonymous quadrantanopsia, and homonymous scotoma was present in 14 patients (20%). The HVFD was partial in 31 patients (44.2%).

A definite lesion explaining the visual defect was reported in 44 patients, localised at either the optic radiations (30/44; 68.2%), the optic tract (12/44; 27.3%), or the lateral geniculate nucleus (2/44; 4.5%).

Regarding the evolution of the HVFD, only fifty-two patients were followed-up. The recovery was described as complete in 31 patients (59.6%), with a median time to recovery of 9 weeks (range 2 weeks to 12 months). An incomplete recovery was found in 16 subjects (30.8%). No recovery was found in five subjects (9.6%). No recurrent episodes of HVFD in multiple sclerosis patients were described in the literature.

Discussion

Patients with multiple sclerosis who present an HVFD are mostly young patients with relapsing-remitting multiple sclerosis, which is consistent with the epidemiology of multiple sclerosis. Both men and women can be affected, with a slight female preponderance (three women, two men), and an HVFD can occur at any time during the course of the disease. One third of our patients (7/20) presented an HVFD as the inaugural symptom of multiple sclerosis, which is three times higher than the proportions previously reported in the literature. This could be related to the fact that nowadays, computerised automated static perimetry is mostly used to evaluate visual fields. As automated static perimetry is more sensitive than other perimetric techniques, it allows more subtle defects to be detected.

Regarding the type of HVFD, we found that partial HVFD were more frequent than complete HVFD. The size and location of a demyelinating lesion are two critical factors which determine whether a lesion will result in a partial or a complete HVFD. Within the optic tract, axons are compacted into a small area and lesions at this location are more likely to produce a complete HVFD. In contrast, within the optic radiations, axons are spread over a larger area and lesions at this location are more likely to a produce partial HVFD. In multiple sclerosis, most lesions associated with HVFD are small and located within the optic radiations. This can explain why most HVFD are partial. Furthermore, optic radiation lesions can be asymptomatic, as MRI studies have revealed that retrochiasmal demyelinating lesions are found more frequently than HVFD [8, 47].

After an episode of HVFD due to a demyelinating lesion, most patients experienced complete recovery of their visual fields (59% in our series and 60% in the literature) in a relatively short time. The median recovery time was ten weeks in our study, which is comparable with previous reports (median of nine weeks with only one patient taking more than 12 months to recover) [7]. A partial recovery was found in 23% of our patients, with a median follow-up of three years. This suggests that if a full visual field recovery is not achieved at three months, it will not occur.

Two patients in our study showed worsening of their HVFD without new clinical relapse. They were both diagnosed with a secondary progressive form of multiple sclerosis. Worsening of an HVFD without new relapses in a patient with relapsing-remitting multiple sclerosis could be a sign of the disease evolving towards a secondary progressive phase. Due to both the highest incidence of relapsing-remitting multiple sclerosis and the relatively long follow-up time in our study (average two years, maximum ten years), it is not surprising that we encountered relapses of HVFD (fig. 4). In our study 3/20 patients presented with recurrences of HVFD.

The sensitivity of OCT for detecting abnormalities can sometimes be higher than that of automated computerised static perimetry. For example, following full recovery after an episode of optic neuritis, all patients exhibited RGCL loss ranging from 5-27% [48]. A higher sensitivity of OCT (namely the measurement of RGCL thickness) compared to computerised visual field examinations was also suggested by our study. Four years after the onset of a completely resolved mild partial right homonymous hemianopsia, OCT revealed the persistence of homonymous thinning of the RGCL (fig. 5, Patient 18). In addition, OCT can sometimes reveal subclinical damage to the retrochiasmal visual pathways, as illustrated by a visually asymptomatic patient with multiple sclerosis (not from the current study) who exhibited a homonymous thinning of the RGCL on OCT, without any history of visual impairment in the past (fig. 6). Homonymous hemiatrophy of the ganglion cell layer was recently reported in a retrospective series of 47 patients with HVFD of various aetiologies [49]. The authors reported that such homonymous hemiatrophy could result from either direct retrograde axonal degeneration or transsynaptic retrograde axonal degeneration. They

Figure 4: Recurrence of a homonymous visual field defect (HVFD), previously partially reported [22]. Top – inaugural left homonymous inferior quadrantanopsia, which disappeared completely after three months. Middle – 2 years later, recurrence of the left homonymous inferior quadrantanopsia, which this time resolved over four months. Bottom – 4 years after the initial episode, a second HVFD recurrence occurred with a bilateral inferior homonymous quadrantanopsia. Complete recovery of the HVFD was obvious after 3.5 months. MD = mean defect expressed in decibels (dB).

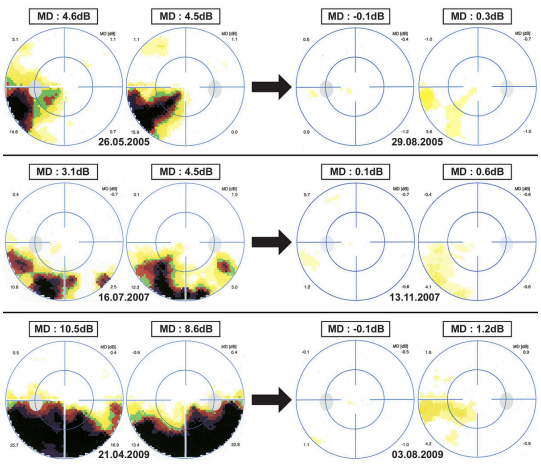
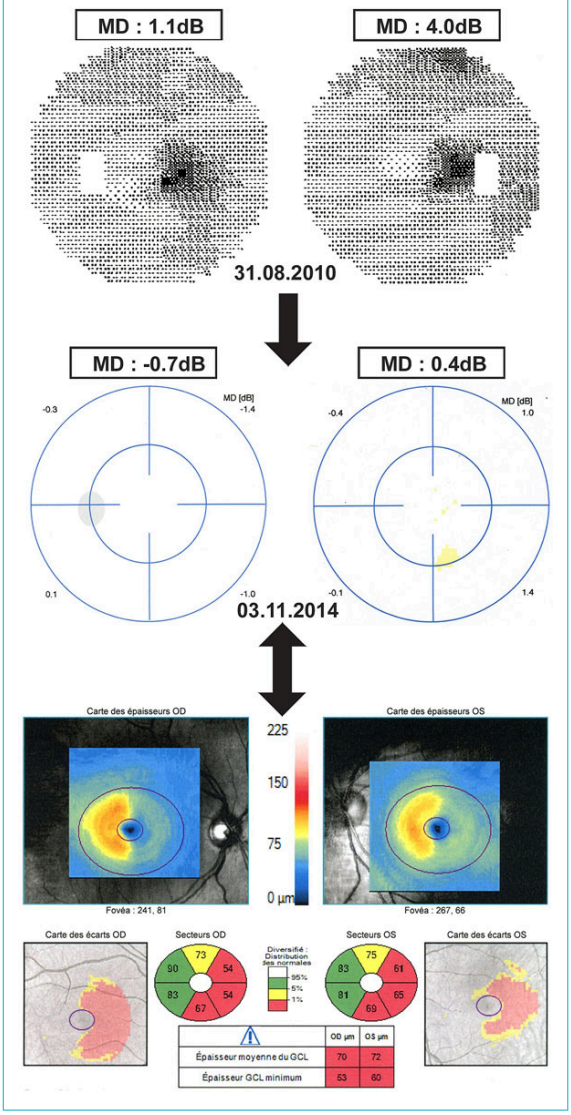


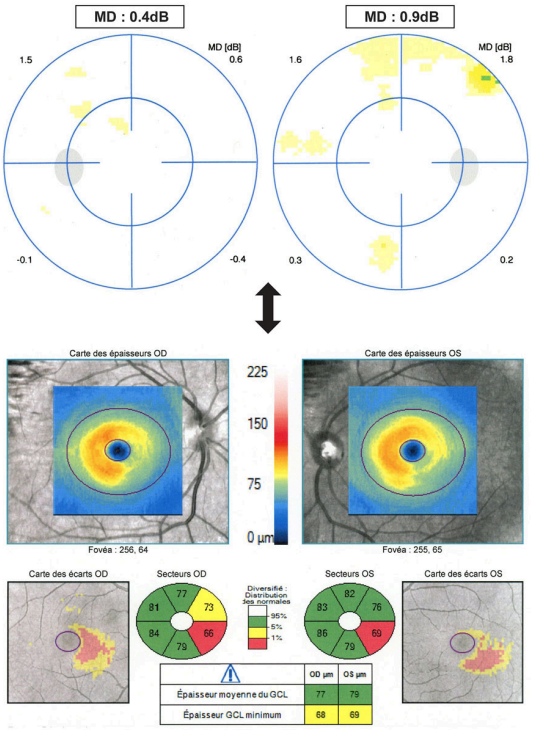
Figure 5: Persistence of homonymous retinal ganglion cell layer thinning after normalisation of the visual field. Top – initially (31 August 2010), the patient presented with a partial right homonymous scotoma and the mean defect (MD) was moderately elevated in both eyes. Middle – 4 years later (3 November 2014), the visual field was completely normal, with a normal MD in both eyes. Bottom – despite a full recovery of visual field, optical coherence tomography on 3 November 2014 showed a homonymous thinning of the retinal ganglion cell layer, visible on the thickness maps, the deviation maps and the sector maps. MD = mean defect expressed in decibels (dB).



also reported a higher sensitivity of RGCL thickness measurement compared to peripapillary retinal nerve fibre layer measurement.

The incidence of HVFD in multiple sclerosis is probably underestimated. Some patients can exhibit only mild and partial HVFD, which could go unnoticed unless examined by static computerised perimetry. In our series, incidental partial HVFD were found in five visually asymptomatic patients at their regular ophthalmologic check-up. Systematic automated static perimetry in multiple sclerosis patients could potentially detect more cases of asymptomatic HVFD. Furthermore, it is possible that prospective OCT examinations could reveal additional cases of subclinical dysfunction of the retrochiasmal visual pathway, as illustrated in figure 6.

Figure 6: Detection of asymptomatic retrochiasmal damage by optical coherence tomography (OCT). Top – normal visual field of a patient with multiple sclerosis who was never visually symptomatic. Bottom –OCT of the same patient, performed at the same date, demonstrated a homonymous left inferior thinning of the retinal ganglion cell layer, visible on the thickness maps, the deviation maps and the sector maps. MD = mean defect expressed in decibels (dB).



The weaknesses of our study are due to its retrospective nature. Not all patients benefited from a cerebral MRI at the time of HVFD diagnosis, and OCT was performed at the time of diagnosis in only 4/10 patients. A prospective study addressing these points would certainly allow to better determination of both the incidence and the clinical profile of HVFD in multiple sclerosis.

Homonymous visual field defects can occur in patients with multiple sclerosis. Their visual prognosis is good overall. The incidence of HVFD in multiple sclerosis might be higher than previously reported. Systematic examination with automated static perimetry and OCT might help to establish the true incidence of involvement of the retrochiasmal pathway in multiple sclerosis.

Acknowledgments
We would like to thank Mr Yann Leba for his assistance in making our figures.

Disclosure statement
No financial support and no other potential conflict of interest relevant to this article was reported.

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