Inapparent Visual Field Defects in Multiple Sclerosis Patients

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ABSTRACT

To assess inapparent visual field defects in patients with multiple sclerosis free from optic neuritis. During 5 years period 120 patients with multiple sclerosis were examined at the University Department of Ophthalmology, Zagreb University Hospital Center. They were divided into three groups with 40 patients each: patients with acute unilateral optic neuritis, referred to ophthalmologist and treated with pulsed steroid therapy; patients with subjective feeling of blurred vision, normal visual acuity and no signs of acute optic neuritis; and patients free from subjective signs of visual impairment. Study patients underwent standard ophthalmologic examination and visual field testing in photopia by use of quantitative kinetic Goldmann perimetry. The initial and control examination by visual field testing were performed at least 6 months apart. Study results showed 65% of multiple sclerosis patients to have visual field defects without subjective signs of impaired vision. The most common defects were mild to moderate visual field narrowing with blind spot enlargement and depression from above. The following results were recorded: acute optic neuritis group: normal in 13/40 (32.5%) for the affected eyes and 27/40 (67.5%) for fellow eyes; mild visual field narrowing in 4/40 (10%) for the affected eyes and 10/40 (25%) for fellow eyes; moderate visual field narrowing with blind spot enlargement in 14/40 (35%) for the affected eyes and 1/40 (2.5%) for fellow eyes; and paracentral and arcuate scotomata in 9/40 (22.5%) for the affected eyes and 2/40 (5%) for fellow eyes; subjective symptom group: normal in 8/40 (20%) for the affected eves and 11/40 (27.5%) for fellow eves; mild visual field narrowing in 11/40 (27.5%) for the affected eves and 16/40 (40%) for fellow eyes; moderate visual field narrowing with blind spot enlargement in 18/40 (45%) for the affected eves and 10/40 (25%); and paracentral and arcuate scotomata in 3/40 (7.5%) for both affected and fellow eves; and subjective symptom-free group: normal in 24/80 (30%), mild visual field narrowing in 22/80 (27.5%) moderate visual field narrowing with blind spot enlargement in 24/80 (30%); and paracentral and arcuate scotomata in 10/80(12.5%). The presence of subclinical form of optic nerve involvement could be demonstrated in a very early stage of multiple sclerosis by the introduction of visual field testing in the standard examination protocol.

Key words: visual field defect, multiple sclerosis

Introduction

At present, there is a general consensus among researchers and clinicians on a significant association between optic neuritis and multiple sclerosis (MS), as many patients with a clinically definitive form of MS have a history of acute optic neuritis during the course of the underlying disease, and the clinically definitive form of MS develops in many patients with a history of optic neuritis^{1,2}. Ebers believes that optic neuritis is a *forme fruste* of MS³. Although optic neuritis is considered to be the most common ocular manifestation in MS and could be the first sign of MS, visual system involvement may also be asymptomatic or very discrete, without any signs of visual acuity impairment or subjective signs of visual field defects^{4,5}. Using various functional tests and visual evoked potentials, many authors demonstrated involvement of the visual pathways in MS patients free from subjective signs of impaired vision^{6–9}. Study hypothesis was that visual field defects are also

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present in MS patients free from optic neuritis. Therefore, in the present study perimetry was chosen as a method to reveal vision function impairment. As these defects involve eccentric, peripheral areas of the visual field and progress at a slow rate, patients may frequently fail to notice them. Thus, the aim of the study was to investigate the prevalence, forms and extent of visual field defects in MS patients in order to assess the association between MS and most common visual field defects. It was also intended to evaluate the role of perimetry in the diagnosis of MS.

Patients and methods

Patients

This prospective study included 120 MS patients diagnosed according to Poser's criteria¹⁰. Neurologic deficit and dysfunction were recorded as scores according to the Expanded Disability Status Scale¹¹. During 5 years period from 1995 to 2000 patients were identified through Neuroophthalmological division of University Department of Ophthalmology, Zagreb University Hospital Center and were divided into 3 groups of 40 patients each: 1) acute optic neuritis group; 2) subjective symptom group; 3) subjective symptom-free group (Table 1). Patient selection was based on the following criteria: group 1 - acute optic neuritis group: unilateral acute optic neuritis, normal visual acuity on the fellow eye; normal visual acuity on the affected eye on control examination at 6 months of therapy introduction; and age 18-45 years. Only patients with unilateral optic neuritis and normal visual acuity on control examination were included.; group 2 - subjective symptom group: subjective symptoms occurring on one eye with normal visual acuity bilaterally; no pulsed therapy for at least 6 months preceding the onset of subjective symptoms, between initial and control examination, and at the time of control examination; and absence of subjective symptoms on 6-month control examination and age 18-45 years; and group 3 - subjective symptom-free group: no history data on optic neuritis; absence of subjective signs of impaired vision; normal visual acuity bilaterally; and no pulsed therapy for at least 6 months preceding the onset of subjective symptoms, between initial and control examination, and at the time of control examination and age 18–45 years. No case of optic neuritis recurrence was recorded during the time between initial and control testing.

On planning the study, 3 groups of 45 subjects were anticipated. During the study, three patients from the subjective symptom group had to be excluded for worsening of their neurologic symptoms and initiation of pulse steroid therapy. Another two patients from the same group failed to show up for control testing. Two patients from the subjective symptom-free group were excluded due to acute optic neuritis. In order to preserve the balance design of the study, each group finally comprised of 40 patients. Patients with ocular lesions leading to visual function defects such as optic medium opacity, retinal detachment, inflammatory lesions, degenerative chorioretinal lesions, glaucoma, strabismus and amblyopia were excluded. Patients with refraction errors greater than 3 diopters were also excluded.

Methods

Patients were examined at University Department of Ophthalmology, Zagreb University Hospital Center in a standardised procedure. Standard ophthalmologic examination consisted of visual acuity measurement with a Snellen chart, biomicroscopy, ophthalmoscopy, and applanation tonometry. Pupillary reactions and bulbar motoricity were also examined. Visual field was tested by kinetic perimetry according to Goldmann¹².

Main outcome measures

In the acute optic neuritis group, the clinical diagnosis of optic neuritis was based on the following criteria: sensation of blurred vision, decreased visual acuity, retrobulbar pain, relative afferent pupillary defect and visual field defects. The eye involved by optic neuritis was considered affected, and the contralateral one as fellow eye. In the subjective symptom group, the eye involved by subjective discomforts was considered affect-

Group	Acute optic neuritis group	Subjective symptoms group	Subjective symptoms-free group		
n	40	40	40		
Age (years)	18–45 (mean 29.225±SD 6.9)	18–45 (mean 29.125±SD 8.1)	18–43 (mean 29.75±SD 8.3)		
Female (%)	25~(62.5%)	28 (70%)	24 (60%)		
Male (%)	15 (37.5%)	12 (30%)	16 (40%)		
MS duration (years)	$1-17 \pmod{6.025 \pm SD \ 3.8}$	$2-16 \pmod{5.85 \pm SD 4.5}$	$1-13 \text{ (mean 5.575 \pm SD 3.8)}$		
Symptoms duration (hours)	$12-55 \pmod{33.425 \pm SD \ 13.3}$	6–90 (mean 44.1±SD 24.5)	0		
EDSS^*	1–5 (mean 2.9625±SD 0.9)	1–3.5 (mean 2.45±SD 0.8)	1–3.5 (mean 2.25±SD 1.0)		

 TABLE 1

 DEMOGRAPHIC AND CLINICAL DATA

* EDSS – An expanded disability status scale according to Kurtzke (11)

ed, and the contralateral one as fellow eye. Subjective discomforts were described as blurred vision on one eye, occasionally accompanied by painful eye movements. The eye exhibiting subjective symptoms is below referred to as affected eye, and the contralateral eye as fellow eye. In the subjective symptom-free group there were no subjective signs of impaired vision and all subjects had normal visual acuity, thus there was no differentiation between affected and fellow eye.

Assessment of a visual field defect is based on the following criteria:

- grade 1, normal finding
- grade 2, mild narrowing of isopters by 10–15°
- grade 3, moderate narrowing of isopters by more than 15° with blind spot enlargement and occasional absence of inner isopters
- grade 4, moderate narrowing of isopters by more than 15° with blind spot enlargement, occasional absence of inner isopters and presence of paracentral or arcuate scotomata
- grade 5, narrowing with the presence of central or cecocentral scotomata, blind spot enlargement and absence of inner isopters

Ophthalmologic examination and visual field testing were performed on two occasions at least 6 months apart. In the acute optic neuritis group, initial examination and visual field testing were performed at the onset of optic neuritis, whereas control examination and visual field testing were performed 6 months from therapy completion. In the subjective symptom group, initial examination and visual field testing were performed at the time of the occurrence of subjective discomforts, whereas control examination and visual field testing were performed at the time free from subjective discomforts. In the subjective symptom-free group, ophthalmologic examination and visual field testing were performed on two occasions at a 6-month interval.

Statistical analysis

Differences in the prevalence of particular grades of visual field defects (grades 1–5) among the three patient groups were determined by χ^2 -test or Fisher exact test for either eye in separate at initial and control examination. In case of statistically significant differences, Bonferroni method of multiple comparison was used to identify the groups of patients with statistically significant between-group differences¹³.

The difference between the affected and fellow eye in groups 1 and 2 was tested by Friedman test for dependent samples on initial and control examination. Friedman test was also used to assess differences between the results of initial and control visual field testing for the affected and fellow eye in separate¹⁴. P-values <0.05 were considered statisticaly significant. All statistical analysis were done using the SAS 8.0^{15} .

Results

On initial testing, all patients had normal visual acuity except for the affected eve in the acute optic neuritis group patients. Nine of patients in the acute optic neuritis group had visual acuity worse than 5/60, 26 patients had visual acuity from 6/60 to 6/15, and five patients from 6/12 to 6/10 according to Snellen chart. On control testing, all patients had normal visual acuity. Comparison of the results obtained by visual field testing according to study groups revealed grades 1 and 2 defects, i. e. mild to moderate visual field narrowing with blind spot enlargement, to be the most common form of impairment, except for the acute optic neuritis group where narrowing with central or cecocentral scotomata was the most common form of visual field defect on the affected eve, recorded in 60% of patients (Table 2). In the subjective symptom group consisting of patients with subjective signs of visual impairment but with normal visual acuity on initial testing, the following results were recorded for the affected and fellow eyes: mild visual field narrowing in 30% and 55%; moderate visual field narrowing with blind spot enlargement in 47.5% and 22.5%; and paracentral and arcuate scotomata in 15% and 10% of subjects, respectively (Table 2). In the subjective symptom-free group, mild visual field narrowing and moderate visual field narrowing with blind spot enlargement were most common defects (Table 2).

In the acute optic neuritis group, recovery of the visual field on the affected eve (Friedman test=29.688. p=0.001) and fellow eye (Friedman test=14.222, p= 0.001) was recorded, (Table 2) whereas visual field defects were more pronounced on the affected eye at both initial (χ^2 =62.424, p=0.001) and control (χ^2 =23.193, p= 0.001) testing (Table 2). On control testing, visual field recovery on both the affected eye (Friedman test= 12.000, p=0.001) and fellow eye (Friedman test=8.00, p=0.05) was also observed in the subjective symptom group (Table 2). In this group, higher grade visual field defects were recorded on initial testing on the affected eye (Fisher exact test=7.571, p=0.058), whereas on control testing these defects were comparable between the affected and fellow eyes (Fisher exact test=3.685, p= 0.305) (Table 2).

In Subjective symptom-free group there were no significant visual field changes between the initial and control testing (Table 2). Analysis of difference in the results of visual field control testing between the affected and fellow eyes in the subjective symptom group and of the two eyes in the subjective symptom-free group showed a comparable distribution of defect severity in the two groups (χ^2 =6.3, p=0.39) (Table 3). Analysis of difference in the results of visual field control testing between the affected and fellow eyes in the acute optic neuritis group and of the two eyes in the subjective symptom-free group pointed to a higher proportion of the moderate form of visual field defect with blind spot enlargement and moderate defect with paracentral and arcuate scotoma on both eyes in the subjective symptom-free group (χ^2 =29.36, p=0.01). Multiple post hoc

No. (%) of eyes in the group										
	Optic neuritis group			Subjective symptoms group			Subjective symp- toms-free group			
Visual field changes	Affected eye		Fellow eye		Affected eye		Fellow eye			
	Initial testing (n=40)	Control testing (n=40)	Initial testing (n=40)	Control testing (n=40)	Initial testing (n=40)	Control testing (n=40)	Initial testing (n=40)	Control testing (n=40)	Initial testing (n=80)	Control testing (n=80)
Normal finding	0	13 (32.5)	16 (40)	27 (67.5)	2 (5)	8 (20)	5 (12.5)	11 (27.5)	24 (30)	24 (30)
Mild narrowing	0	4 (10)	16 (40)	10 (25)	13 (32.5)	11 (27.5)	22(55)	16 (40)	22(27.5)	22(27.5)
Moderate narrowing with blind spot enlargement and occasional absence of inner isopters	5 (12 5)	14 (35)	6 (15)	1 (2.5)	19 (47.5)	18 (45)	9 (22.5)	10 (25)	24 (30)	24 (30)
Narrowing with the presence of arcuate or paracentral scotoma with blind spot enlargement	10 (25)	9 (22.5)	2 (5)	2 (5)	6 (15)	3 (7.5)	4 (10)	3 (7.5)	10 (12.5)	10 (12.5)
Narrowing with the presence of central or cecocentral scotomata	25 (62.5)	0	0	0	0	0	0	0	0	0

 TABLE 2

 RESULT OF VISUAL FIELD TESTING IN ALL GROUPS

TABLE 3

COMPARISON OF RESULTS OF CONTROL VISUAL FIELD TESTING IN ACUTE OPTIC NEURITIS GROUP AFFECTED AND FELLOW EYES, OF SUBJECTIVE SYMPTOM GROUP AFFECTED AND FELLOW EYE, AND OF CONTROL VISUAL FIELD TESTING IN SUBJECTIVE SYMPTOM-FREE GROUP

No. (%) of eyes in the group						
Visual field changes	Acute optic neuritis group affected eye Visual field control testing (n = 40)	Acute optic neuritis group fellow eye Visual field control testing (n = 40)	Subjective symptom group affected eye Visual field control testing (n = 40)	Subjective symptom group fellow eye Visual field control testing (n = 40)	Subjective symp- tom-free group Visual field control testing (n = 80)	
Normal finding	13 (32.50)	27 (67.50)	8 (20.00)	$11\ (27.50)$	24 (30.00)	
Mild narrowing	4 (10.00)	10 (25.00)	11(27.50)	16 (40.00)	22 (27.50)	
Moderate narrowing with blind spot enlarge- ment and occasional ab- sence of inner isopters	14 (35.00)	1 (2.50)	18 (45.00)	10 (25.00)	24 (30.00)	
Narrowing and presence of arcuate or paracentral scotoma with blind spot enlargement	9 (22.50)	2 (5.00)	3 (7.50)	3 (7.50)	10 (12.50)	

mutual testing showed a higher proportion of the defects described above on the affected eye in the acute optic neuritis group (p=0.03) and on both eyes in the subjective symptom-free group (p<0.001). Also, there was similar distribution of the visual field defects on the affected eye in the acute optic neuritis group and on both eyes in the subjective symptom-free group (p=0.378) (Table 3).

Discussion

Although optic neuritis is considered to be the most common form of visual function impairment in multiple sclerosis patients⁵, this study revealed that visual field defect may also be present in those multiple sclerosis patients who have never had optic neuritis. In our subjective symptom-free group, 57.5% of patients had visual field defect although they had no history of optic neuritis or any subjective signs of impaired vision. A similar finding has also been recorded by Patterson and Heron¹⁶, who report on the majority of their study subjects to have visual field defect without a history of optic neuritis. However, Burde and Gallin¹⁷ found their study subjects to have normal field of vision. This discrepancy could be attributed to different techniques of visual field testing used in the two studies. In the latter, visual field was tested by use of I2–I4 isopters, whereas we used and a I1 isopter (lower relative intensity test spot). The behavior of I1 isopter is of special importance, as it is located in the intermediary zone which is sensitive to various pathologic conditions¹².

In the subjective symptom-free group, mild to moderate narrowing and blind spot enlargement were the most common defects. Such asymptomatic visual field defects (negative scotomata) could be explained by their localization in the eccentric, peripheral visual field segments, bilateral occurrence and slow progression, thus being quite inapparent. Such a picture is opposite to that of acute optic neuritis, which is considered the most common visual lesion in patients with multiple sclerosis, where it is the reason for their visit to an ophthalmologist.

In the present study, visual field defects were categorized according to the prevalence of particular forms of these defects. On control testing, mild to moderate narrowing with blind spot enlargement was the most common form of visual field defects. The visual field defects found in our patients were comparable to those reported elsewhere^{18–20}. Although a higher prevalence of arcuate scotomata is being reported elsewhere 16,18,21 . the variation could be attributed to different methods of visual field testing, since some authors used automated perimetry and others employed Bjeruum's screen. As patient selection is also important, it should be noted that only patients with normal visual acuity were included in our study. Nevertheless, the results appear to be quite similar. Narrowing and occasionally absence of inner isopters accompanied by blind spot enlargement were recorded in the majority of our patients. Inner isopters reflect changes in the very 10°-15° area, in which other authors found defects¹⁸. Some of our results are quite comparable with those reported by Nizankowska et al²¹, because depression from above was also recorded in some of our patients, however, in contrast to our study, these authors found no blind spot enlargement. This variation could also be ascribed to the use of a different method of visual field testing, i.e. automated perimetry^{19,21}. They performed visual field testing within 30°, which is highly relevant in the light of our detecting defects in peripheral areas of the visual field, not only in central areas within 30°. Comparison of the results of control visual field testing performed during the quiescent stage showed visual field narrowing and blind spot enlargement to be the most common defects in all study groups. The higher proportion of normal visual field in the acute optic neuritis group could be ascribed to the administration of pulsed therapy, which was not used in the other two groups. Visual function impairment in the acute phase is explained by inflammation and oedema that lead to conductivity interruption. The possible visual function recovery occurs due to restored conductivity following regression of oedematous and inflammatory lesions, and partially due to early remyelination. Such recovery usually occurs within 3-6 months²². Late recovery of visual function could be attributed to longterm remvelination. Demveliantion, which may be followed by axonal degeneration, is known to be involved in plaque evolution 23,24 . An active process of demvelination is accompanied by myelin and oligodendrocyte destruction within the plaque. Oligodendrocytes were found to be significantly reduced in MS patients who died within one month of the clinical manifestation of the disease. Also, demyelination may be followed by remyelination, which can halt the degeneration of demyelinating axons, i.e. axonal destruction²⁵. Prineas et al²⁵ found the rate of plaques with signs of remyelination (shadow plagues) to markedly increase only 12 weeks of the initial clinical manifestation of the disease. However, these shadow plaques may be exposed to demveliantion and be converted to classic demvelinating plaques²⁶. Jones et al explain the recovery of VEP parameters by the process of remyelination, redistribution of ion channels, and potential cortical restructuring, emphasizing that this pattern of recovery can only be observed in 2-3 years²².

The presence of fellow eye lesion in the acute optic neuritis group on initial testing (during the course of acute optic neuritis) could be explained by dissemination of the inflammation from the affected $eye^{23,24}$. Other authors also report on visual field impairment on the fellow $eye^{27,28}$. As visual field recovery was also recorded on the fellow eye, these impairments were not likely to be caused by longstanding lesions^{23,24}. Beck et al²⁷ also describe recovery of the visual field on the fellow eye with steroid therapy.

It cannot be stated for sure whether the inapparent visual field impairments were caused by optic nerve demyelination or involvement of other segments of the visual pathway⁵. Optic tract involvement is frequently found on autopsy and magnetic resonance neuroradiology in multiple sclerosis patients. The tight fiber structure of the optic nerve sheath is highly sensitive to pathologic lesions. Although posterior segments of the vision pathway are also frequently involved, there is great disparity between the pathologic lesions and clinical signs, which could be explained by anatomic dispersion of the visual fiber in the sphere of vision. A minor plaque in this posterior segment of the vision pathway may involve a smaller number of fibers than in the optic nerve itself. This would result in a relatively rare manifestation of such characteristic visual field lesions. Also, such plaques may be asymptomatic as they cause changes in the peripheral segment of the visual field. Minor scotomata cause only mild changes in the field of vision without affection of the central visual acuity. Subjectively inapparent visual field defects may also be caused by subtle degenerative processes, oligodendropathy, and loss of axons^{5,29-31}. Multiple sclerosis is not only associated with demyelination but also with axonal damage and degeneration as well as with anatomic and biochemical changes of axons within the MS lesion². Brusa et al²⁴ think that visual function impairment occurs consequentially to irreversible axonal degeneration rather than demyelination. They believe that the failure of visual field recovery in their study was due to irreversible axonal degeneration. Accordingly, the presence of visual field impairment in our subjective symptom-free group could have also been due to the presence of irreversible axonal degeneration. In this group there were no significant visual field changes between the initial and control testing.

Results of the study showed the standard ophthalmologic examination with manual visual field testing to record mild defects in patients free from subjective symptoms but to provide inadequate data on the visual system lesion. This points to the need of comparative ophthalmologic functional diagnosis in patients with multiple sclerosis, and especially in those free from acute optic neuritis episodes. Further studies are needed to evaluate the introduction of contrast sensitivity testing, visual field testing with automated perimetry and visual evoked potential testing to provide addi-

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In conclusion, the study showed the visual field defects to be also present in MS patients free from a history of optic neuritis. In contrast to other literature reports, three patient groups were compared. In addition to the groups of MS patients with optic neuritis and those without a history of optic neuritis, that have been most largely investigated to date, we differentiated a third group of patients with subjective signs of vision impairment and normal visual acuity. A similar group has also been described by Frederiksen et al³² however, without comparing them with other patient groups. Furthermore, all our patients had normal visual acuity on control testing. Our study has entailed highly relevant findings of a similar distribution of visual field defects across all study groups in the quiescent stage of the disease, when there were no signs of vision function impairment, and of the high prevalence of peripheral visual field defects. As the study showed the visual field defects to be located in the peripheral areas of the field of vision and to progress slowly, they proceed unnoticed by MS patients. This points to the role of perimetry in the early recognition of MS patients while still free from subjective disturbances. As visual field testing has to date been mostly focused on the central part of the field of vision, this study emphasizes the need of including peripheral areas in visual field testing.

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NEZAMIJEĆENA OŠTEĆENJA VIDNOG POLJA U BOLESNIKA KOJI BOLUJU OD MULTIPLE SKLEROZE

SAŽETAK

Utvrditi nezamjećena oštećenja vidnog polja u bolesnika s multiplom sklerozom koji nisu imali optički neuritis. Tijekom 5 godina pregledano je 120 bolesnika koji boluju od multiple skleroze na Klinici za očne bolesti Medicinskog fakulteta KBC Rebro zagreb. Oni su podijeljeni u tri skupine sa po 40 ispitanika svaka: ispitanici koji su se javili oftalmologu zbog akutnog optičkog neuritisa i liječeni pulsnom steroidnom terapijom, ispitanici sa subjektivnim znacima zamućenja vida, urednom vidnom oštrinom i bez znakova optičkog neuritisa, te ispitanici bez subjektivnih znakova oštećenja vida. U istraživanju je učinjen standardni oftalmološki pregled, te testiranje vidnih polja Goldmann kinetičkim perimetrom. Inicijalno i kontrolno testiranje učinjeno je u razmaku od 6 mjeseci. Istraživanje je pokazalo da 65% bolesnika s multiplom sklerozom bez subjektivnih znakova ima oštećenje vidnog polja. Najčešći oblici oštećenja su blaže do umjereno suženo sa proširenjem slijepe pjege uz depresiju odozgo. Utvrđeni su slijedeći rezultati: acute optic neuritis group: uredan nalaz u 13/40 (32.5%) za zahvaćeno oko, te 27/40 (67.5%) za prateće oko; blaže suženje vidnog polja u 4/40 (10%) za zahvaćeno, te 10/40 (25%) za prateće oko; umjereno suženje uz proširenje slijepe pjege u 14/40 (35%) za zahvaćeno oko, te 1/40 (2.5%) za prateće oko; paracentralni i arkuatni skotomi u 9/40 (22.5%) za zahvaćeno oko, te 2/40 (5%) za prateće; subjective symptom group: uredan nalaz u 8/40 (20%) za zahvaćeno oko, te 11/40 (27.5%) za prateće oko; blaže suženje vidnog polja u 11/40 (27.5%) za zahvaćeno oko te 16/40 (40%) za prateće oko; umjereno suženje uz proširenje slijepe pjege u 18/40 (45%) za zahvaćeno te 10/40 (25%) za prateće oko; i paracentralni i arkuatni skotom u 3/40 (7.5%) za oba oka; i subjective symptom-free group: uredan u 24/80 (30%), blaže suženje u 22/80 (27.5%) umjereno suženje s proširenjem slijepe pjege u 24/80 (30%); te prisutnost paracentralnih i arkuatnih skotoma u 10/80 (12.5%) ispitanika. Prisutnost supkliničkog oblika zahvaćanja vidnog živca može se utvrditi u vrlo ranoj fazi multiple skleroze uvođenjem testiranja vidnog polja u standardni protokol.