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VISUAL FIELDS IN GIANT CELL ARTERITIS (HORTON'S DISEASE)

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Abstract

Background: The purpose of this study was to investigate visual field defects in different clinical presentations of giant cell arteritis.

Methodology: Retrospective study of 36 patients from 1996 – 2010 with giant cell arteritis (67% female, average age 79, range 62-92 years), with typical clinical picture and/or positive biopsy (78%, 18/23 performed). Visual fields charted by Goldmann perimeter were categorized into specific categories. Respect for horizontal meridian was noted.

Results: Loss of vision was unilateral (65%) or bilateral (35%). The most common ischaemic lesions were anterior ischaemic optic neuropathy (AION; 16 unilateral, 4 bilateral) and posterior ischaemic optic neuropathy (PION; 5 unilateral, 5 bilateral). Other lesions included central retinal artery occlusion (CRAO) and internuclear ophthalmoplegia (INO). More than half of the affected eyes (53%) had visual acuity of counting fingers to no light perception. We observed 11 types of visual field defects. AION presented most commonly with peripheral island (35%) and sector defect (35%). Inferior altitudinal defect occurred in 18%. There was a tendency to affect nasal and inferior halves of visual fields. PION occurred in 31% of patients and most often presented with scotoma with or without peripheral defect.

Conclusions: AION most commonly presented with peripheral island (35%) or sector defect (35%). Visual field defects in AION almost universally showed respect for horizontal meridian, except where only small residual island remained. Inferior altitudinal defect occurred less commonly than expected (18%). PION was more prevalent in our study (31%) compared to others and most often presented with scotoma with or without peripheral defect.

Keywords

• Giant cell arteritis • Horton's disease • Temporal arteritis • Visual field defects • Goldmann kinetic perimetry • Ischaemic optic neuropathy • AION • PION • Neuroophthalmology

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1. Introduction

Giant cell arteritis (GCA, temporal arteritis, Horton's disease) is the most common primary vasculitis of adulthood. Incidence among people older than 50 years is estimated at 3-30/100.000 per year [1]. Women are more often affected than men [2]. The hallmark of GCA is inflammation of vessel walls that induces intimal hyperplasia and luminal occlusion. Most patients have lesions in branches of the carotid and vertebral arteries. Clinical symptoms are caused by end-organ ischaemia and systemic inflammation [3], and include headache, jaw claudication, visual loss, rheumatic polymialgia and systemic signs such as fever, fatigue and weight loss [4].

Ocular symptoms are present in 30-75% of patients and include visual loss, amaurosis fugax, ophthalmoplegia, ptosis, miosis, diplopia, ocular pain and Horner's syndrome [5-7]. Approximately 15-50 % of patients suffer from permanent visual loss [5,8-10] which is caused by ischaemia of any part of visual pathway [11]. The anterior part of the optic nerve is most commonly affected (anterior ischaemic optic neuropathy, AION), caused by occlusion of the posterior ciliary or ophthalmic arteries [5]. Another cause of visual loss is ischaemia of the posterior part of the optic nerve (posterior ischaemic optic neuropathy, PION) due to occlusion of arteries that supply the retrobulbar part of optic nerve (Figure 1). Other causes of visual loss include occlusion of central retinal artery (CRAO) or cilioretinal artery which supply the retina [5,12,13]. Visual loss is often severe and most patients present with visual acuity of counting fingers to no light perception [5].

In the differential diagnosis it is important to consider nonarteritic ischaemic optic neuropathy, which is not associated with giant cell arteritis. The nonarteritic form is presumably caused by non-perfusion due to fluctuation of arterial blood pressure in people with systemic risk factors, particularly nocturnal arterial hypotension [14]. In AION, visual field loss tends to be altitudinal and has a sharp demarcation at the horizontal meridian [15]. The most common visual field defect in nonarteritic AION (NA-AION) is a combination of a relative inferior altitudinal defect with absolute inferior nasal defect [16]. This finding can be explained by the common location of the temporal part of the optic disc in the watershed zone between the posterior ciliary arteries [17-20]. In the arteritic AION (A-AION) eyes visual field defect depends upon the area of the optic nerve head supplied by the occluded posterior ciliary artery, which varies widely from eye to eye [21]. The most common visual field defect in arteritic and nonarteritic PION is central visual loss, alone or in combination

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with other types of visual field defects, and much less commonly the reverse pattern, i.e., the central field normal with marked loss of peripheral fields [22]. The purpose of this study was to investigate visual field defects in different clinical presentations of giant cell arteritis. It is commonly accepted that visual field loss in AION tends to be altitudinal with sharp demarcation at the horizontal meridian [15]. For A-AION there is limited literature on specific types of visual field defects. A study of 25 patients with A-AION showed a wide variety of visual fields abnormalities, the most common of them being inferior altitudinal defect [23]. In a study of 45 patients with GCA, the majority of testable eyes, aside from central scotomas associated with loss, showed altitudinal or arcuate patterns [24]. For NA-AION it was shown with a detailed study that the most common specific visual field defect is inferior nasal defect, which together with inferior nasal step occurred in 36% of cases. Thus, contrary to the prevalent impression, an absolute inferior nasal sector defect is the most common type of visual field defect in NA-AION [16].

2. Experimental Procedures

We retrospectively studied 36 consecutive patients (50 eyes) from years 1996 – 2010 who presented with visual loss due to giant cell arteritis. There were 24 (67%) female and 12 (33%) male patients with average age of 79 years (range 62-92 years). Inclusion criteria were typical clinical picture and/or positive biopsy (78%, 18 of 23 performed). Two patients with positive biopsies had on previous presentations negative biopsies. Biopsy was not performed in 13/36 (36%) patients. Reasons included previous diagnosis of GCA, being unable to locate the temporal artery or treatment with anticoagulant therapy. All of them presented with a typical clinical picture (sudden severe loss of vision with combinations of symptoms or signs that included headache, muscle pain, jaw claudication, weight loss, elevated ESR and CRP).

Patients were assigned to four different groups based on their clinical presentation of AION, PION, CRAO and INO. AION was diagnosed based on visual loss in presence of optic disc edema and PION in patients with no optic disc edema and presence of relative afferent pupillary defect.

Visual acuity was measured with Snellen charts. Visual fields were charted by Goldmann perimeter, using isopter II/4 (stimulus size 1mm^2, luminance 1000 apostilbs) and in patients with lower vision also V/4 (stimulus size 64 mm^2, luminance 1000 apostilbs). Color vision was tested with Ishihara plates. Visual fields were first categorized as peripheral defects or scotomas and then further divided into specific categories according to published classification [16]. Mixed defects were classified according to the more prominent defect. Respect for horizontal meridian was noted if the outer isopter formed a horizontal border on the horizontal meridian or near it. Where such assessment was possible, we determined which half of visual field was more affected (superior versus inferior and nasal versus temporal). In evaluation of scotomas we included absolute as well as relative defects.

3. Results

3.1 Clinical presentation:

The most common ischaemic lesions were AION (16 unilateral, 4 bilateral) and PION (5 unilateral, 5 bilateral). Other lesions included CRAO, INO and combinations. There was no significant difference in age among clinical presentations (see Table 1). Loss of vision was more often unilateral (24, 65%) than bilateral (13, 35%). One patient had two separate episodes of unilateral AION on different eyes and was counted as unilateral AION. Among unilateral ischaemic optic neuropathies AION was equally distributed between both eyes whereas PION more often affected the left eye (4 versus 1).

Visual Acuity:

Among all affected eyes, 28/50 (56%) presented with visual acuity of counting fingers to no light perception. In respect to clinical presentation this was found in 50%, 31%, 67% and 0% of AION, PION, CRAO and INO respectively (see Table 2). For 4/36 (11%) patients such visual loss was bilateral. Two patients had no light perception (bilateral AION and AION+CRAO) and two patients had only light perception (bilateral CRAO and AION+PION).

Visual Field:

Visual field examination was possible in 37/50 (74%) of affected eyes. In remaining 13/50 (26%) eyes it was not possible to obtain the fields due to poor vision. Visual fields found in different clinical presentations are shown in Figure 2-5. We observed 11 of 22 different types of visual field defects according to published classification [16] (see Table 3).

AION: visual field examination was possible in 17/26 eyes (Figure 2). We observed three

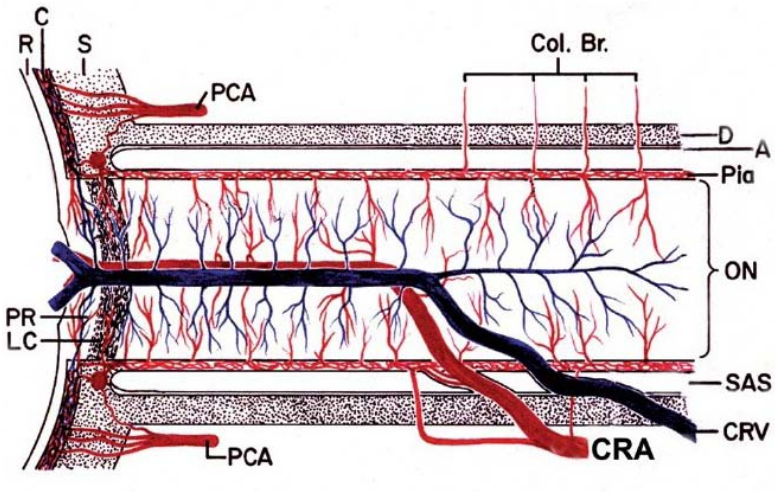

Figure 1. Schematic representation of blood supply of the optic nerve. A = arachnoid; C = choroid; CRA = central retinal artery; Col. Br. = Collateral branches; CRV = central retinal vein; D = dura; LC = lamina cribrosa; ON = optic nerve; PCA = posterior ciliary artery; PR = prelaminar region; R = retina; S = sclera; SAS = subarachnoid space. Reproduced, with permission of the American Academy of Ophthalmology, from Hayreh S. S., Trans. Am. Acad.Ophthalmol. Otolaryngol., Anatomy and physiology of the optic nerve head, 1974, 78, 240-254.

Table 1. Clinical presentation and average age of patients with giant cell arteritis. AION = Anterior ischaemic optic neuropathy; PION = Posterior ischaemic optic neuropathy; CRAO = Central retinal artery occlusion; INO = Internuclear ophthalmoplegia. *One patient is counted twice for two separate episodes on different eyes.

Clinical Presentation	Number of eyes (%)	Average age (years)
Unilateral AION	16* (34,2) (13 right, 13 left)	79
Unilateral PION	5 (13,5) (1 right, 4 left)	80
Bilateral PION	5 (13,5)	80
Bilateral AION	4 (10,8)	77
Unilateral CRAO	3 (8,1)	72
Bilateral CRAO	1 (2,7)	78
Bilateral AION and unilateral CRAO	1 (2,7)	89
Unilateral AION and unilateral PION	1 (2,7)	84
Internuclear ophthalmoplegia (INO)	1 (2,7)	73

Table 2. Visual acuity in giant cell arteritis. AION = Anterior ischaemic optic neuropathy; PION = Posterior ischaemic optic neuropathy; CRAO = Central retinal artery occlusion; INO = Internuclear ophthalmoplegia.

Visual Acuity	**Number of eyes (%)**				
	TOTAL (50 eyes)	**AION (26 eyes)**	**PION (16 eyes)**	**CRAO (6 eyes)**	**INO (2 eyes)**
20/40 (0,5) or better	10 (20,0)	3(11,5)	6 (37,5)	0	1 (50)
20/50 – 20/400 (0,4 – 0,05)	13 (26,0)	5(19,2)	5 (31,3)	1 (16,7)	1 (50)
Counting fingers to light perception	22 (44,0)	13(50)	5 (31,3)	4 (66,7)	0
No light perception	6 (12,0)	5(19,2)	0	1 (16,7)	0

Table 3. Visual field defects in giant cell arteritis. Combined defects were designated in regard to more prominent defect. Numbers in brackets represent eyes with respect for horizontal meridian. AION = Anterior ischaemic optic neuropathy; PION = Posterior ischaemic optic neuropathy; CRAO = Central retinal artery occlusion; INO = Internuclear ophthalmoplegia.

	Number of eyes			
Visual Field Defects	**AION**	**PION**	**CRAO**	**INO**
Normal Visual Field				**2**
Peripheral Defects				
Sector defect	6(6)	3(1)		
Inferior altitudinal defect	3(3)	1(1)		
Superior altitudinal defect	1(1)			
Vertical defect	1			
Only peripheral island of field present	6(1)			
Only central island of field present		2	3	
Generalized constriction		1		
Scotomas				
Central scotoma		1		
Paracentral scotoma		3		
Centrocecal scotoma		2		
Enlarged blind spot		2		
Examination not possible	9	1	3	

main categories: sector defects (6, 35%), half-field defects (5, 29%; 4 altitudinal, 1 vertical) and peripheral islands (6, 35%). The most common sector defect was inferior nasal (3 eyes), which represented 18% (3/17) of eyes with AION, where examination was possible. Respect for horizontal meridian was seen in 11/17 (65%) eyes. It was not seen in five eyes with peripheral islands and one eye with vertical defect. Where it was possible, we determined which half of visual field was more affected. In regards to nasal versus temporal, 11/13 (85%) of visual fields were more affected in the nasal half. In regards to superior versus inferior, 8/11 (73%) were more affected in the inferior half.

PION: visual field examination was possible in 15/16 eyes (Figure 3). There were 8/15 (53%) eyes with various scotomas (3 absolute and 3 relative) or enlarged blind spots (2). Remaining eyes (7/15, 47%) presented with more prominent peripheral defects. Out of those, four (three sector and one altitudinal) were combined with scotomas and three were not (one generalized constriction and two central islands). Two eyes showed some respect for horizontal meridian, however, the border was less sharp than in AION.

CRAO: visual field examination was possible in 3/6 eyes. All three eyes showed profound visual loss with only central island of field left. Figure 4 shows one example.

INO: Both eyes in patient with internuclear ophthalmoplegia showed normal visual field (Figure 5).

Color vision data was available for 42/50 (84%) eyes. Out of those, 28 (56%) eyes could not read the first plate (vision was too low to perform the examination), 3 eyes (6%) could read only the first plate (severely affected color vision), four eyes (8%) could read 2-6 plates and seven eyes (14%) could read 10 plates or more (normal or moderately affected color vision). All of the latter were affected with PION or INO. There were no eyes with AION that could see 10 or more plates. Color vision of individual eyes is shown on Figures 2-5.

Visual acuity in respect to various visual field defects is shown in Figure 6. Visual acuity was better in eyes with enlarged blind spots and sector defects and worse in eyes with altitudinal defects, central scotomas and only islands of visual field remaining.

4. Discussion

The purpose of this study was to investigate visual field defects in different presentations of giant cell arteritis. We included 36 consecutive patients (50 eyes) with a typical clinical picture and, in most cases positive biopsy. Patients with negative biopsy or without biopsy were included only if the clinical picture was typical. Negative biopsy in GCA is possible as a result of "skip lesions" in the temporal artery [25,26]. We also showed this in our study as in two patients with multiple episodes of disease, biopsies were first negative and later positive.

Average age (79 years) and distribution among men and women (two thirds of patients were women) was in concordance with published studies [2,5]. All patients were older than 62 years.

Patients presented with different clinical pictures depending on the site of ischaemic lesion. AION was most common and was found in 22/36 (61%) patients or 27/50 (54%) eyes. It presented as unilateral (16), bilateral (4) or in combination with PION (1) or CRAO (1). The incidence for A-AION in our study was 0.3/100.000 for persons older than 60 years of age. The second most common clinical presentation was PION, which presented as unilateral (5), bilateral (5) or in combination with AION (1). In total, it affected 16/50 (32%) eyes or 11/36 (31%) patients. Our proportion of patients with PION is high in comparison to other studies [5,22]. In a study by Hayreh, from 85 biopsy-confirmed patients there were only six (7%) patients with PION [5]. Even If we were to include only patients with positive biopsies, the proportion of PION would remain around 30% (5/18, 28%). PION was only recently described as clinical entity [27]. In the literature, information on the clinical features of PION consists mostly of three retrospective studies: by Isayama et al. in 14 patients [28], Sadda et al. in 72 patients [29] and Hayreh in 42 patients [22]. All cases in the Isayama et al. series had nonarteritic PION, among the 72 patients in the Sadda et al. 53% had nonarteritic, 8% arteritic, and 39% surgical PION. In the study of Hayreh of 43 patients in the course of 30 years, 65% had nonarteritic, 28% arteritic, and 7% surgical PION. Low number of reports of PION in literature in comparison to our numbers might be due to the fact that PION is a new clinical entity

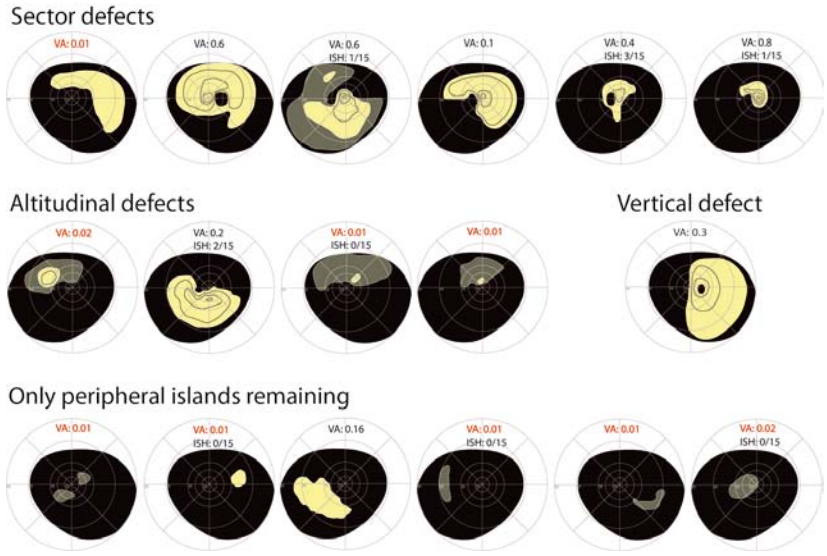

Figure 2. Visual fields in arteritic AION. AION = Anterior ischaemic optic neuropathy, VA = Visual acuity, ISH = Ishihara color vision test.

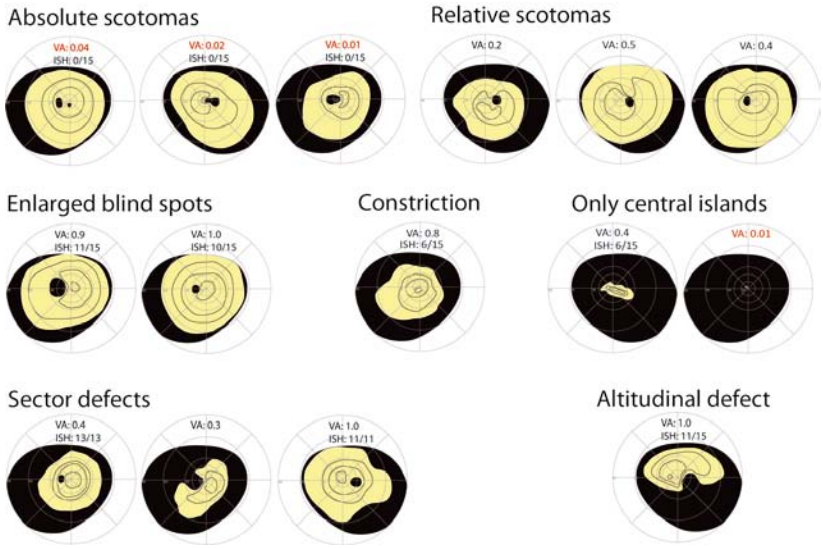

Figure 3. Visual fields in arteritic PION. PION = Posterior ischaemic optic neuropathy, VA = Visual acuity, ISH = Ishihara color vision test.

Figure 4. Visual fields in arteritic CRAO. CRAO = Central retinal artery occlusion, VA = Visual acuity, ISH = Ishihara color vision test.

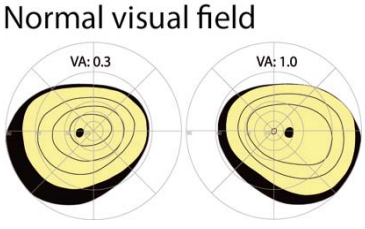

Figure 5. Visual fields in arteritic INO. INO = Internuclear ophthalmoplegia, VA = Visual acuity, ISH = Ishihara color vision test.

and can be underdiagnosed, especially because there is no patognomonic sign such as optic edema in AION. All our patients with PION had typical clinical signs of GCA, visual loss and no optic disc edema. Our higher numbers of PION might also be due to the fact that we included in diagnosis all symptomatic disturbances of vision and visual field, including enlarged blind spot, relative scotoma and peripheral constriction. There were four eyes with such presentations. Two patients had unilateral PION with confirmed biopsy and two patients had bilateral PION without performed biopsy. From five patients with PION with positive biopsies there were two patients with mild presentation.

Unilateral AION was equally distributed between both eyes whereas unilateral PION was more often found in the left eye (4 versus 1). This number is too low for any statistical analysis but it is interesting to note that same distribution was found in a study by Hayreh (5 versus 0) [5].

More than half of the GCA patients (28/50, 56%) in our study presented with visual acuity of counting fingers to no light perception. With respect to clinical presentation, this was found in 50%, 31%, 67% and 0% in AION, PION, CRAO and INO respectively. For 4/36 (11%) patients such visual loss was bilateral. Our data is similar to previously published study [5] (Table 4). Color vision was reduced in most patients and it was worse in AION than in PION.

Visual field examination was possible in 37/50 (74%) of affected eyes.

AION: In more than half of our patients (15/26, 58%), vision was too low to perform examination. We observed three main categories: sector defects (6, 35%), half field defects (5, 29%; 4 altitudinal, 1 vertical) and fields with only peripheral islands remaining (6, 35%). Respect for horizontal meridian was found in 11/17 (65%) eyes. It was not found in 5 eyes with only peripheral islands (there was not enough visual field left) and one eye with vertical defect. Horizontal border of visual fields was not always very sharp and directly on the mid horizontal line. In some eyes it was a little off the centre and in other it was rounded (see Figure 2). This could be an effect of poor fixation or examination error. Where such assessment was possible, we found that nasal and inferior halves of fields were most often affected. Literature on visual fields in arteritic AION reports that the most common defects in arteritic AION are inferior altitudinal [23] and altitudinal and arcuate patterns [24]. We found that the most common were peripheral islands (6/17, 35%) and sector defect (6/17, 35%). Only 3/17 (18%) patients presented with inferior altitudinal defect and 1/17 (6%) with superior altitudinal defect. In Figure 2 we present all visual fields in our patients; it can be seen that peripheral islands and sector defects are most common whereas altitudinal defects present minority of cases. In comparison to nonarteritic AION there were also some differences. Inferior nasal defect, which is most common in nonarteritic AION (36%) [16], occurred only in 3/17 (18%) patients.

PION: There were 8/15 (53%) eyes with various scotomas or enlarged blind spot, and 7/15 (47%) eyes with more prominent peripheral defects. Out of those, three were combined with scotomas. This is in agreement with published data [22]. Other: All three eyes with CRAO in which examination was possible showed profound visual loss leaving only central island of visual field, while patient with INO presented normal visual field.

In conclusion, our study presents systematic evaluation of visual fields in patients with giant cell arteritis. In more than half of the GCA patients with visual loss, visual acuity was reduced to counting fingers or less. AION presented most commonly with peripheral islands (35%) and sector defects (35%). Inferior altitudinal defects occurred less commonly than expected (18%). Visual field defects in AION almost universally showed respect for horizontal meridian, except where only small residual islands remained. There was a tendency to affect nasal and inferior halves of visual fields. PION was more numerous in our study as that reported by others (31%) and most often presented with scotoma with or without peripheral defect that were most often associated with left eyes of the patients, as also reported by Hayreh [5].

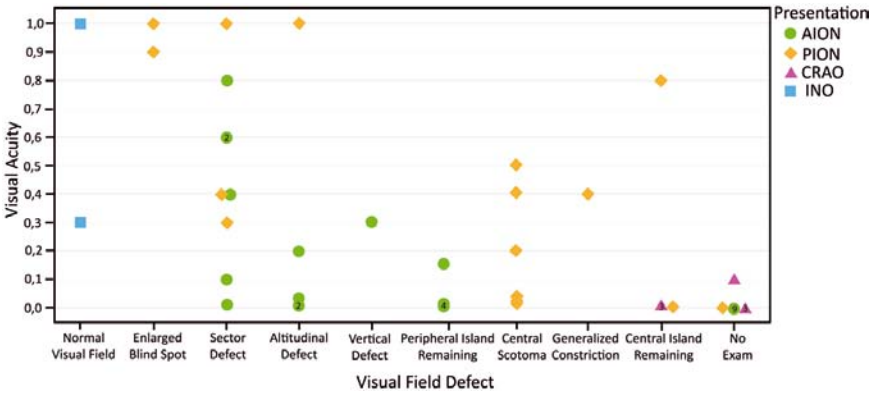

Figure 6. Visual acuity in specific visual field defects. AION = Anterior ischaemic optic neuropathy, PION = Posterior ischaemic optic neuropathy, CRAO = Central retinal artery occlusion, INO = Internuclear ophthalmoplegia.

Table 4. Visual acuity in giant cell arteritis, our study compared to published data [5]. AION = Anterior ischaemic optic neuropathy; PION = Posterior ischaemic optic neuropathy; CRAO = Central retinal artery occlusion; INO = Internuclear ophthalmoplegia.

Initial Visual Acuity	Percentage of eyes (number of eyes)	
	Our study (50 eyes)	Hayreh et al. 1998 (123 eyes)
20/40 (0,5) or better	20 (10)	21 (26)
20/50 – 20/400 (0,4 – 0,05)	26 (13)	25 (31)
Counting fingers to light perception	44 (22)	38 (47)
No light perception	12 (6)	15 (19)

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