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Case report

Detection of the progression of retinal nerve fiber layer loss by optical coherence tomography in a patient with glaucomatocyclitic crisis

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ABSTRACT

A 30-year-old man presented with glaucomatocyclitic crisis, also known as Posner–Schlossman syndrome, and acute intraocular pressure elevation. Changes in the longitudinal retinal nerve fiber layer (RNFL) and the cup-to-disc (C/D) ratio of the optic disc were detected by Stratus optical coherence tomography (OCT). The average RNFL thickness in the affected eye was greater than that in the unaffected eye on Day 3 (132.99 μm, C/D ratio: 0.24 vs. 105 μm, C/D ratio: 0.26). However, the RNFL thickness continued to decrease and the C/D ratio progressively increased in the affected eye over 12 months (60 μm, C/D ratio: 0.67), although the intraocular pressure was controlled at <21 mmHg during that period. Glaucomatous visual field defects were also found. A progressive decrease in the thickness of the RNFL was demonstrated by Stratus optical coherence tomography following an elevation in the acute intraocular pressure in the patient. Prompt treatment and longitudinal monitoring are necessary to prevent and detect glaucomatous damage.

1. Introduction

Glaucomatocyclitic crisis, also known as Posner–Schlossman syndrome, is a unilateral, benign ocular disease that is characterized by a mild anterior chamber reaction accompanied by an increase in intraocular pressure (IOP) with open angles. It is often self-limiting and can be recurrent. However, the etiology is controversial.[1,2] Some patients have been known to develop glaucoma-related visual field defects as a result of repeated episodes of glaucomatocyclitic crisis.[3,4]

Using Stratus optical coherence tomography (OCT), we conducted a longitudinal evaluation of the thickness of the retinal nerve fiber layer (RNFL) and the cup-to-disc (C/D) area ratio of the optic disc in a patient with glaucomatocyclitic crisis. We examined the differences in these parameters at the acute and remission stages of the disease.

2. Case report

A 30-year-old man presented with decreased vision in his right eye (OD) for 2 days. He had no history of systemic disease but had experienced a similar episode (OD) 1 year previously. During the first episode, the patient received treatment and had since been in a stable condition. Ocular examination of the OD revealed mild epithelial edema of the cornea, a few fine keratic precipitates, few aqueous cells, and little flare. The IOP was 46 mmHg with a hypopigmented open angle. The vitreous was clear, the retina was normal, and the optic disc exhibited mild hyperemia. The left eye (OS) had no abnormalities, and the IOP was 15 mmHg. The best-corrected visual acuity was 20/20 (OU) with myopia of −0.5 diopters (OU). Color sensation and pupil response were normal (OU).

All laboratory data were normal, including antinuclear antibodies, rheumatoid factor, venereal disease research laboratory test, HLA-B27, and chest radiograph. Therefore, the patient was diagnosed with glaucomatocyclitic crisis.[1–3]

Following treatment with oral acetazolamide, topical 2% carteolol, and 1% prednisolone acetate solution, the IOP decreased to 12 mmHg (OD). The IOP was maintained at <15 mmHg (OU) with topical 2% carteolol solution (OD). Follow up was performed every month over a period of 12 months. The patient did not experience any repeat attacks during the 12-month follow-up period.

The thickness of the peripapillary RNFL and the C/D ratio were automatically calculated using the fast RNFL thickness protocol (3.4), and the optic disc parameters using a Stratus OCT 3000 (Version 4.0.2; Carl Zeiss Meditec, Inc., Dublin, CA, USA). High-quality images with a quality score of signal strength 10 (maximum 10) were obtained on Day 3, and at 2 months, 5 months, and 12 months after the episode of glaucomatocyclitic crisis.[5]

Conflicts of interest: The author has no conflicts of interest relevant to this article.

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The average RNFL thickness in the affected eye (132.99 μm, C/D ratio: 0.24) was greater than that in the unaffected eye (105 μm, C/D ratio: 0.26) on Day 3. However, the RNFL thickness progressively decreased and the C/D ratio progressively increased at 2 months (73 μm, C/D ratio: 0.61), 5 months (67 μm, C/D ratio: 0.63), and 12 months (60 μm, C/D ratio: 0.67) (Figs. 1 and 2). By contrast, there were no obvious changes in the unaffected eye after 12 months (102 μm, C/D ratio: 0.25).

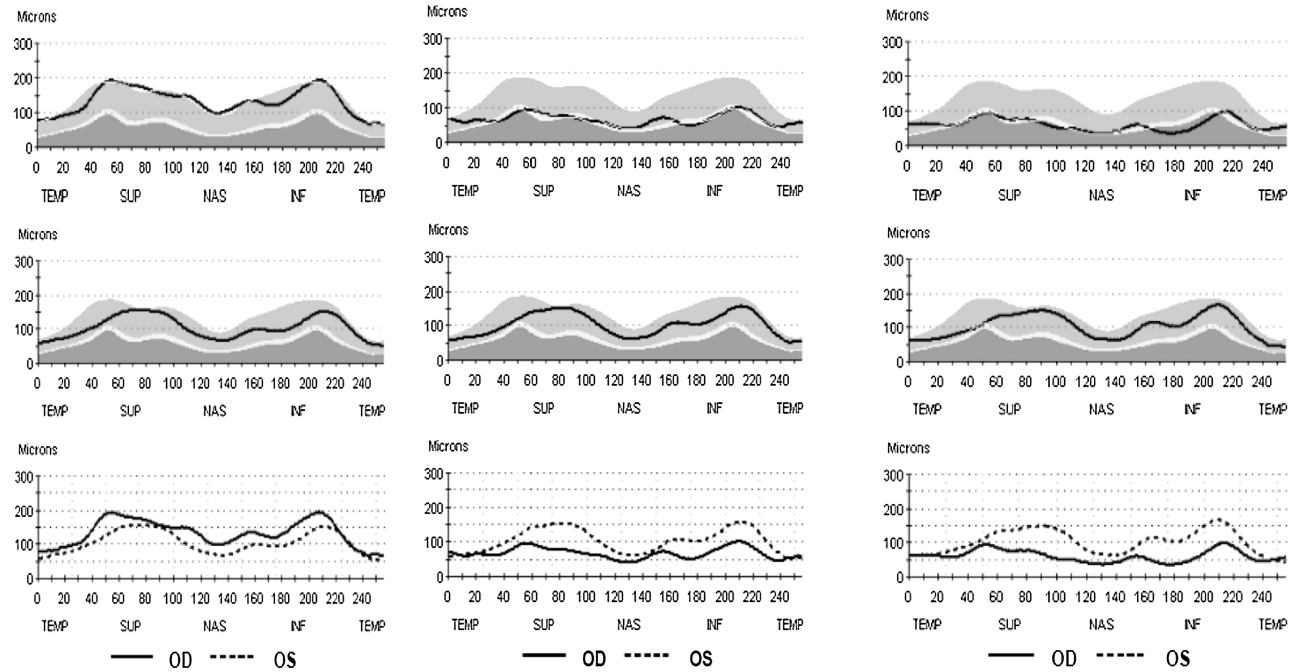
An automated perimetry test was conducted using the Humphrey 30-2 threshold program. After an episode of glaucomatocyclitic crisis in the affected eye, a glaucomatous superior arcuate visual field defect was found at initial examination and at 1 year (mean deviation: −7.22 dB, pattern standard deviation: 5.23 dB, left, vs. mean deviation: −7.28 dB, pattern standard deviation: 8.02 dB, right; Fig. 3).

3. Discussion

Investigations into the "normal" RNFL thickness, using Stratus OCT, revealed that between the ages of 18 years and 85 years, Caucasians have an average RNFL thickness of 98.1 ± 10.9 μm, Hispanics 103.7 ± 11.6 μm, and Asians 105.8 ± 9.2 μm. The mean RNFL thickness of the inferior quadrant is 126.1 ± 17.8 μm, superior quadrant 124.2 ± 17.9 μm, nasal quadrant 80.9 ± 18.1 μm, and temporal quadrant 69.0 ± 12.7 μm. The inferior and superior quadrants are thicker than the nasal and temporal quadrants.[6] Decreases in the RNFL thickness of the inferior and superior quadrants are more sensitive for the detection of glaucoma.[7]

A previous study demonstrated that after a single episode of acute primary angle closure with a high IOP (>50 mmHg), there was an initial increase, followed by a decrease, in the diffuse RNFL thickness. The average decrease in the RNFL thickness correlated with the interval of follow-up, and gradually stabilized at 3 months. The overall reduction in RNFL thickness correlated with the duration of acute IOP elevation.[8]

In this case, most reduction in the thickness of the RNFL occurred within the first 2 months, with only a small reduction occurring after 2 months. This result is consistent with the findings of previous reports.[8,9] A possible explanation for this occurrence has been proposed by Yoles and Schwart.[10] These authors proposed a mechanism, termed secondary degeneration, whereby glaucomatous neuropathy continues to progress even after the reduction of



	OD (N=3)	OS (N=3)	OD-OS
Imax/Smax	1.01	0.97	0.04
Smax/Imax	0.99	1.03	-0.04
Smax/Tavg	2.26	2.11	0.15
Imax/Tavg	2.28	2.04	0.23
Smax/Navg	1.53	1.79	-0.26
Max-Min	128.00	105.00	23.00
Smax	192.00	156.00	36.00
Imax	193.00	151.00	42.00
Savg	168.00	137.00	31.00
Iavg	154.00	120.00	34.00
Avg.Thick	132.99	104.53	28.46

	OD (N=3)	OS (N=3)	OD-OS
Imax/Smax	1.06	1.04	0.03
Smax/Imax	0.94	0.97	-0.02
Smax/Tavg	1.62	2.17	-0.55
Imax/Tavg	1.72	2.25	-0.52
Smax/Navg	1.70	1.80	-0.10
Max-Min	60.00	106.00	-46.00
Smax	95.00	152.00	-57.00
Imax	101.00	157.00	-56.00
Savg	79.00	132.00	-53.00
Iavg	75.00	126.00	-51.00
Avg.Thick	67.30	103.21	-35.91

	OD (N=3)	OS (N=3)	OD-OS
Imax/Smax	1.05	1.12	-0.07
Smax/Imax	0.95	0.89	0.06
Smax/Tavg	1.67	2.26	-0.60
Imax/Tavg	1.75	2.54	-0.80
Smax/Navg	2.02	1.66	0.36
Max-Min	64.00	124.00	-60.00
Smax	94.00	149.00	-55.00
Imax	99.00	167.00	-68.00
Savg	76.00	126.00	-50.00
Iavg	61.00	130.00	-69.00
Avg.Thick	60.33	102.97	-42.64

Fig. 1. RNFL thickness, measured using Stratus optical coherence tomography, after an episode of glaucomatocyclitic crisis. The Avg.Thick, Savg, and Iavg were, respectively, 132.99 μm, 168 μm, and 154 μm in the affected eye (OD) on Day 3 (left); 67.3 μm, 79 μm, and 75 μm (OD) at 5 months (middle); and 60.33 μm, 76 μm, and 61 μm at 12 months (right). Avg.Thick = average retinal nerve fiber layer thickness; Iavg = retinal nerve fiber layer thickness of the inferior quadrant; INF = inferior quadrant; NAS = nasal quadrant; RNFL = retinal nerve fiber layer; Savg = retinal nerve fiber layer thickness of the superior quadrant; SUP = superior quadrant; TEMP = temporal quadrant.

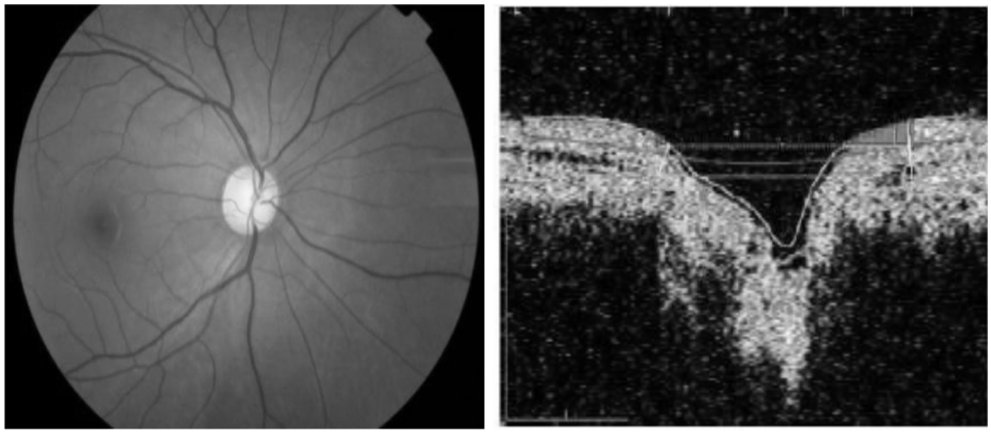


Fig. 2. Optic disc and retinal photographs at 12 months, after an episode of glaucomatocyclitic crisis in the affected eye. The optic disc cup/disc area ratio measured using Stratus optical coherence tomography was 0.67.

the high IOP. Furthermore, there may be an effect on the healthy neurons that were not part of the primary injury but were adjacent to the injured neurons, as they have been exposed to the degenerative environment.

The patient in our case had a relatively greater average RNFL thickness in the affected eye than in the unaffected eye on Day 3. The optic nerve head appeared slightly edematous after the high IOP persisted for up to 2 days prior to treatment, even after there was a reduction of the IOP in the acute stage. However, the visual field examination may be unreliable in the acute IOP elevation stage. After remission, the visual field defects vary in severity.[11] This may explain why the visual field defect and the reduction of the RNFL thickness were not the same in the acute stage. Fang et al[9] reported that most eyes affected by acute primary angle closure demonstrated an increase in the RNFL thickness within 2 weeks after the episode, except in one patient who had previously suffered acute attacks. The authors suggested that if the increase of the RNFL thickness due to edema is less than the decrease caused by glaucoma, RNFL atrophy may not be evident. Thus, the RNFL thickness may be in the normal range in the early period after an acute episode, especially in patients who experienced elevated IOP with ongoing RNFL damage prior to the acute attack.

After the episode, the decrease in thickness of the RNFL was generalized. However, the visual field defect, which occurred predominantly in the superior hemifield, existed prior to the episode, with only focal deepening after the episode. The possible explanation for the structure–function discrepancy is that there was pre-existing RNFL thinning in the right eye prior to the episode, which was masked by the RNFL swelling that was caused by the attack.[9]

Jap et al[3] reported that 26.4% of patients developed glaucoma as a result of repeated attacks of glaucomatocyclitic crisis. Patients who experienced glaucomatocyclitic crisis for 10 years or more had a 2.8 times higher risk of developing glaucoma than those who had experienced the disease for less than 10 years.

Although glaucomatocyclitic crisis is a relatively benign and often self-limiting disease, progressive RNFL loss and glaucomatous damage can occur as a result of repeated attacks and prolonged IOP elevation, if there is no immediate treatment.

In conclusion, Stratus OCT demonstrated progression of RNFL thickness loss and C/D ratio enlargement after acute IOP elevation in a patient with glaucomatocyclitic crisis. Therefore, urgent treatment and longitudinal monitoring are necessary to prevent and detect glaucomatous damage in glaucomatocyclitic crisis.

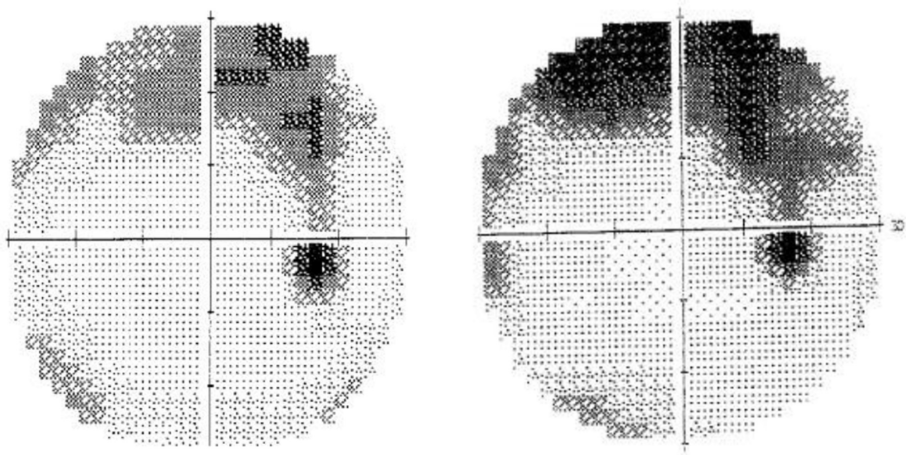


Fig. 3. Superior arcuate visual field defect, initially (mean deviation: −7.22 dB, PSD: 5.23 dB, left) and at 1 year (mean deviation: −7.28 dB, PSD: 8.02 dB, right) after an episode of glaucomatocyclitic crisis in the affected eye.

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