CASE REPORT

Lack of Correspondence between Early Optic Disc Oedema and Macular Ganglion Cell Loss in Non-arteritic Anterior Ischaemic Optic Neuropathy

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ABSTRACT

A 73-year-old Asian male presented with a 2-week history of acute visual disturbance in his right eye. Visual acuity in his right eye was hand movement and fundus examination showed right optic disc oedema and splinter haemorrhages on the disc margin. Each time the patient visited our clinic, spectral domain optical coherence tomography (OCT) was performed. He was diagnosed to have non-arteritic anterior ischaemic optic neuropathy. Optical coherence tomography showed disagreement between the areas of early peripapillary retinal nerve fibre layer thickening and macular ganglion cell layer thickness reduction.

Keywords: Ganglion cell layer, NAION, optical coherence tomography

INTRODUCTION

Non-arteritic anterior ischaemic optic neuropathy (NAION) is a well-known disorder causing sudden onset of painless visual loss with optic disc oedema, with or without flame-shaped haemorrhages present at or near the disc margin. It has been proposed that in NAION retinal ganglion cell losses result from obstruction of axoplasmic flow and axonal swelling due to minimal microvascular ischaemia.¹ Therefore, there is consistent correlation between the sector of the optic disc showing the earliest oedema and the visual field defect. In other words, the area of macular ganglion cell loss should correspond to the area of early optic disc oedema.²

Optical coherence tomography (OCT) is proving a useful tool in neuro-ophthalmology. Recently, studies on peripapillary retinal nerve fibre layer (RNFL) and macular ganglion cell layer (GCL) complex in NAION have been reported, in which it was suggested that OCT can be used to demonstrate optic disc oedema and monitor RNFL loss over time. In these studies, RNFL and macular GCL losses correlated well with

visual field (VF) losses in both magnitude and location.^{3,4}

Using spectral domain OCT, we report a patient with NAION who showed disagreement between the area of early optic disc oedema and macular ganglion cell loss.

CASE REPORT

A 73-year-old Asian male presented with a 2-week history of acute visual disturbance in his right eye, which worsened gradually over the next 5 days. No associated ocular pain and systemic symptoms were observed. The patient underwent aortic valve replacement 16 months ago and had been taking warfarin. He had no history of systemic hypertension, diabetes, nor hyperlipidaemia. Laboratory testing showed normal erythrocyte sedimentation rate and C-reactive protein. Full blood count values were normal. Prothrombin time and activated partial thromboplastin time were prolonged due to warfarin.

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Visual acuity (VA) was hand movements OD and 20/20 OS, A relative afferent pupillary defect OD was observed. Perimetry and colour vision testing for the right eye could not be performed due to the patient's low vision. Dilated funduscopic examination showed optic disc oedema and splinter haemorrhages on the optic disc margin OD and a slightly small optic disc with a cup disc ratio of 0.3 OS. Fluorescein angiography showed only dye leaking from the capillaries in the optic nerve head and late staining. On the basis of the history, examination and laboratory evaluation a diagnosis of NAION was made. No treatment for NAION was given. Each time the patient visited our clinic, an OCT evaluation was performed using the Cirrus HD-OCT machine (Carl Zeiss Meditec) software version 6.0. Peripapillary retinal nerve fibre layer (pRNFL) thickness, ganglion cell layer

(GCL) + inner plexiform layer (IPL) thickness, and macular thickness were analyzed.⁵

Figure 1 shows the serial results of the fundus photograph, and pRNFL, and macular GCL + IPL, and macular thickness OD using OCT. The results showed early superior optic disc oedema and increased superior pRNFL thickness, but inferior macular GCL + IPL thickness began to decrease prior to a decrease in superior macular GCL + IPL thickness. His visual acuity was not improved at the most recent follow-up, remaining at hand movements.

DISCUSSION

Optic disc oedema is a classical initial sign of acute NAION. It is by definition always present at the onset

FIGURE 1 Serial results of fundus photographic imaging of the optic nerve head, and OCT imaging of pRNFL, macular GCL + IPL, and macular thicknesses. On the patient's first visit (2 weeks after the onset of visual loss), the superior pRNFL thickness was increased and the macular GCL + IPL thickness was within the reference range. Five weeks after the onset of visual loss, a new splinter haemorrhage on the inferotemporal optic disc rim was found and the inferior pRNFL thickness that was mildly increased on the first visit was further increased. Macular GCL + IPL thickness showed greater thinning in the inferior half than in the superior half. At 8 weeks, severe swelling and haemorrhage of the inferior optic disc margin had developed and inferior pRNFL thickness had significantly increased. The inferior and superior macular GCL + IPL thicknesses had decreased and this was prominent in the inferior half. At 10 weeks, the superior and inferior pRNFL thicknesses had decreased and the inferior pRNFL thickness had nearly normalized. At the final follow-up of 28 weeks, the temporal optic disc was pale. pRNFL and macular GCL + IPL thicknesses were very low in the entire area; macular thickness was within a normal range until the final follow-up. At the final follow-up, macular GCL + IPL thickness in all areas had decreased evenly.

of visual loss. Optic disc oedema in NAION is due to axoplasmic flow stasis in the ischaemic axons in the optic nerve head. Hayreh and Zimmerman⁶ showed that optic disc changes in NAION undergo a characteristic evolutionary process during the first 2–3 months. They demonstrated that initially the involved part of the disc shows oedema, with the remaining area of the disc normal or showing considerably less oedema; after several days, the entire disc can show generalized oedema. Subsequently, the oedema gradually starts to regress in the originally involved part, so that the uninvolved part can show more oedema than the ischaemic part. The involved part then shows pallor, whereas the remaining part of the disc can show mild oedema and even some pallor. They also showed that the overall median time required for the resolution of optic disc oedema from the onset of visual loss is 7.9 (5.8–11.4) weeks. In our case, the fundus photograph showed static disc oedema up to 2 weeks and increased oedema at 8 weeks. Thereafter, a pale disc was observed.

OCT could also prove to be a useful tool in neuro-ophthalmology. Recently, studies on pRNFL thickness and macular GCL complex in NAION have been reported.^{3,4,7} Contreras *et al.*³ showed that OCT could be used to diagnose optic disc oedema and monitor pRNFL loss over time and that pRNFL thickness decreased to values within normal limits at the 1.5-month visit. Papchenko *et al.*⁷ showed that both macular thickness and volume parameters are correlated with visual sensitivity, using Stratus OCT and Humphrey automated perimetry in NAION. Aggarwal *et al.*⁴ stated that pRNFL and macular GCL losses correlated well with VF losses in both magnitude and location.

In our case, the evolution of pRNFL thickness and macular GCL + IPL thickness were clearly shown. Interestingly, superior pRNFL thickness increased more than inferior pRNFL thickness at the early stage, but macular GCL + IPL thickness in the inferior half sector began decreasing prior to the superior half sector. This is different from the previous studies.^{4,7} Generally, early superior disc oedema accompanies inferior field defect with decreased superior GCL + IPL thickness. In our case, a disagreement between the area of early increased disc oedema and GCL + IPL thickness decrease was observed. Although this disagreement is not clearly understood, we postulated that the axons of the inferior optic disc were more susceptible to compression and ischaemic damage than the superior optic disc in this patient. Therefore, although less swelling was shown at the inferior optic disc than at the superior optic disc, inferior macular ganglion cell loss was more prominent than superior macular ganglion cell loss at 2 weeks after the onset of visual loss. The inferior optic

disc swelling then progressed up to 8 weeks with ongoing ganglion cell loss.

This case demonstrates some significant points. Firstly, the area of early increased disc oedema and the macular ganglion cell loss can be at variance in NAION. We have collected over 50 other cases (over 50 cases) of NAION who have been studied in this way but in only the present case was there a discrepancy between the area of early increased disc oedema and GCL + IPL thickness decrease. We therefore propose this to be a rare occurrence. Secondly, OCT is helpful for detecting progression or regression of optic disc swelling using the measurement of pRNFL, whereas the measurement of GCL + IPL thickness is useful to determine the macular ganglion cell condition, particularly when we cannot perform a visual field test.

In summary, this is an interesting case of NAION that shows incongruity between the area of early increased disc oedema and macular ganglion cell loss and the potential usefulness of OCT in NAION.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Note: Figure 1 of this article is available in colour online at www.informahealthcare.com/oph.

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