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Delayed Non-arteritic Anterior Ischemic Optic Neuropathy Following Acute Primary Angle Closure

Dear Editor,

Non-arteritic anterior ischemic optic neuropathy (NAION) secondary to acute primary angle closure (APAC) is a rare clinical disorder that leads to significant visual impairment. Although a number of cases of APAC-associated NAION have been reported, most involved NAION concurrent with APAC rather than secondary to it [1-3]. Herein, we report a case of three-day-delayed-onset NAION following APAC. We believe that this case illustrates the predispositional mechanism of post-APAC NAION occurrence.

A 64-year-old woman presented with two-day painful visual loss in the left eye. She had been diagnosed with systemic hypertension on a recent regular check-up. Visual acuity was 6 / 200 in the left eye and 20 / 25 in the right eye. Intraocular pressure (IOP) was 64 mmHg in the left eye and 15 mmHg in the right eye. There were signs of APAC in her left eye, including corneal edema, dilated and nearly-fixed pupil, and shallow anterior chamber. Optic disc exam revealed a hyperemic left optic nerve head (ONH) (Fig. 1A). Optical coherence tomography revealed slight inferior ONH swelling and inferior retinal nerve fiber layer (RNFL) edema (Fig. 1B). There was no relative afferent pupillary defect in either eye on swinging-light test. The patient was administered IOP-lowering and miotic medications, which reduced the left-eye IOP to 16 mmHg. Subsequently, laser iridotomy was performed without complication. The following day, the left eye visual acuity was 16 / 20, and the IOP was 16 mmHg. Coincidentally, the patient was examined using 24-hour ambulatory blood pressure monitoring (already planned by the Internal Medicine physician) one day after APAC relief. The results showed no significant systemic hypotension or nocturnal blood pressure dip. Three days later, however, painless left-eye vision loss was experienced upon waking in the morning. Visual acuity was 4 / 20, and IOP was 15 mmHg. The left pupil showed a relative afferent pupillary defect. The left ONH was diffusely swollen with disc hemorrhage at the temporal margin (Fig. 1C). Optical coherence tomography showed aggravated edema of the inferior RNFL (Fig. 1D). A Humphrey visual-field test showed a superior altitudinal visual-field defect corresponding to the inferior RNFL swelling (Fig. 1E). Based on these findings, left NAION was diagnosed. One year later, the left visual acuity was 16 / 20, and the IOP was 17 mmHg. The optic nerve edema of the left eye had subsided (Fig. 1F), but the RNFL showed atrophy (Fig. 1G). The visual-field defect was incompletely improved (Fig. 1H).

NAION is caused by hypo-perfusion of the ONH. Ocular perfusion pressure is determined according to the difference between mean arterial pressure and IOP. Therefore, NAION can develop in settings of hemodynamic compromise such as systemic hypotension, blood loss, and anemia [4]. However, our patient showed no significant systemic hypotension or nocturnal blood pressure dip. In the present case, a significant increase in IOP reduced the perfusion pressure on the optic nerve due to compression of vessels in the prelaminar region [5].

ONH edema is a sign integral to the diagnosis of NAION. Patients with APAC sometimes also develop mild optic nerve edema that is not associated with NAION. However, these cases can be differentiated from post-APAC NAION by the absence of subsequent optic nerve atrophy and preservation of visual acuity and the visual field [6].

According to our research, there have been no more than several case reports of NAION occurring simultaneously with APAC [1-3]. These reports posited only a possible association between APAC and NAION, and, when considering that hypothesis, could not draw any firm conclusions as to whether APAC is a cause or a result. We believe that APAC is one of the possible causes of NAION. We suggest that slight but critical ONH edema (of the inferior portion in our case) caused by APAC-coincident ischemia initiates the vicious cycle of ONH ischemia, swelling, capillary compression, and additional ischemia, though we do not yet know the critical amount of optic-disc edema causing NAION. Additionally, premonitory ONH swelling is a well-recognized phenomenon in typical NAION [7].

Our present case demonstrates the possibility of the occurrence of NAION despite successful APAC treatment and recovery of normal visual acuity. Therefore, close observation of APAC with ONH edema is recommended.

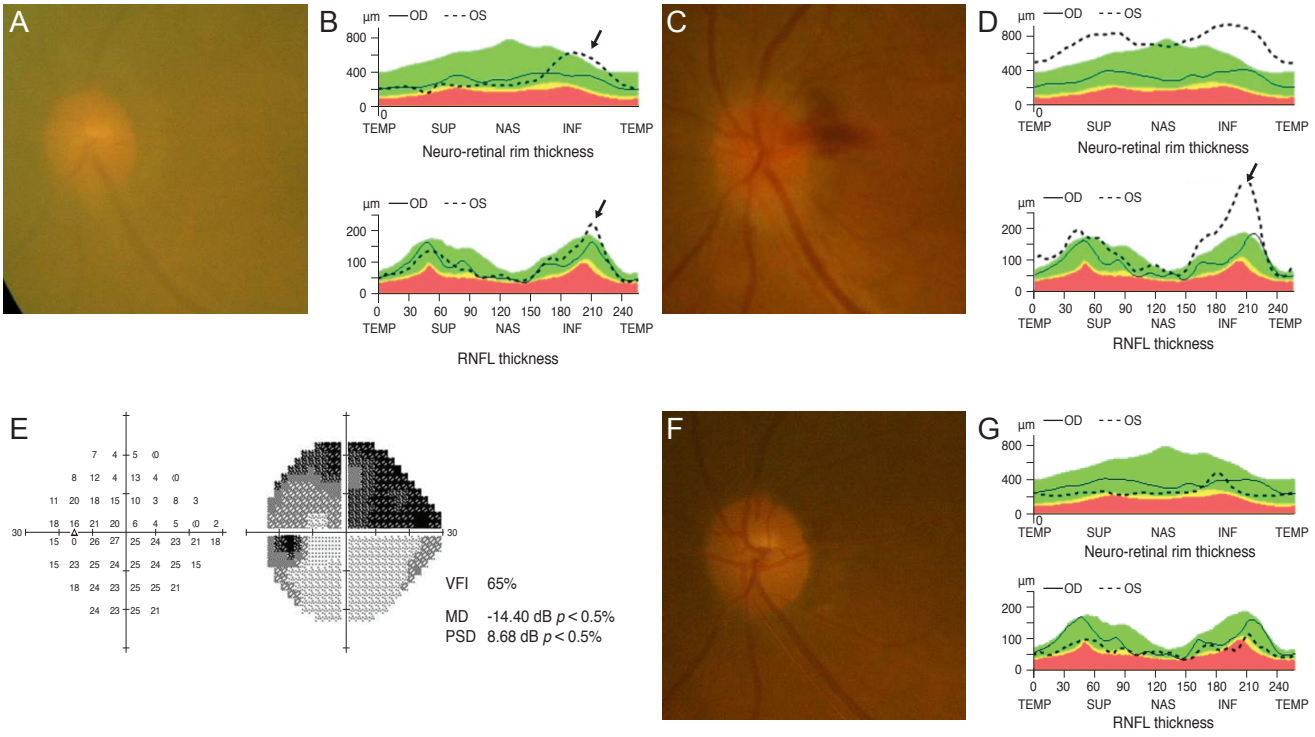

Fig. 1. Disc photographs and printout of optical coherence tomography (OCT) taken at the time of left acute primary angle closure show a hyperemic and slightly edematous left optic nerve head (A) and a slight inferior neuro-retinal rim edema and inferior retinal nerve fiber layer (RNFL) edema (B, black arrows). Three days after angle closure relief (C,D,E), disc photograph shows a diffusely swollen optic nerve head with disc hemorrhage at the temporal margin. The inferior pole is more swollen (C). OCT shows a diffusely, especially inferiorly, swollen RNFL (D, black arrow). Humphrey visual field test demonstrates a superior altitudinal visual-field defect corresponding to inferior RNFL swelling (E). One year later, optic nerve head edema of the left eye subside (F). OCT shows RNFL thinning (G). The visual-field defect is only incompletely improved (H). OD = right eye; OS = left eye; TEMP = temporal; SUP = superior; NAS = nasal; INF = inferior; VFI = visual field index; MD = mean deviation; PSD = pattern standard deviation.

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Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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