A Preliminary Diagnosis Need to be Considered as a Differential Diagnosis of Submacular Fluid: Non-arteritic Ischemic Optic Neuropathy-Case Report

Ayse Yagmur KANRA[1], Hasim USLU[2]

ABSTRACT

Here we present the two cases of non-arteritic ischemic optik neuropathy that demonstrated subretinal fluid on optical coherence tomography. First case was diagnosed as central serous chorioretinopathy in the outer center and treated with oral acetazolamide during 1 week; because of less fluid, she was followed with topical nepafanac in our clinic. As another case had symptoms during three days and significant subretinal fluid, he was treated with oral steroid therapy. Following the week, both of them had visual improvement and subretinal fluid resolution. Their fluorescein angiogram did not show accumulation of dye in the macular region, indicating the fluid did not arise from the retinal vessels or directly from the choroid; early dye leakage and late staining of the optic disc were present. According to age, ischemic optic neuropathy is a differential diagnosis of maculer edema and can affect the therapy options.

Key Words: Optic neuropathy, Subretinal fluid, Steroid therapy.

INTRODUCTION

Anterior ischemic optic neuropathy (AION) is the most common cause of acute optic neuropathy that decreased visual acuity and causes permanent damage in individuals older than 50 years.[1] Although pathogenesis hasn't been fully understood, it is generally attributed to ischemia at optic nerve head resulting from combination of different factors such as age, anatomy, vascular support, compartment syndrome and nocturnal hypoxemia.[2] Although several treatment modalities such as optic nerve sheath decompression, levo L-3,4-dihydroxyphenylalanine (DOPA), aspirin, intravitreal steroids and anti-vascular endothelial growth factor have been used in the treatment, they have limited effectiveness.[3] However, in a large, prospective study, Hayreh et al. found promising outcomes regarding benefits of systemic steroids.[4]

It was recognized that macular involvement can also be present in optic nerve disorders such as AION and pupil edema by advances in imaging studies in recent years.[5] Subretinal fluid secondary to AION exacerbates visula loss. We present 2 cases to emphasize association of AION with subretinal fluid which should be considered in differential diagnosis of macular edema and may affect on treatment modalities.

CASE REPORT

Case 1

A 53-years old woman had been diagnosed as central serous chorioretinopathy by optic coherence tomography (OCT) in another facility and used acetazolamide tablet over a week. In the ophthalmological examination in our clinic, visual acuity was 20/50 with hypermetropia correction (+2.00) in left eye while there was no abnormal finding in anterior segment. In fundus examination, right eye was normal while optic nerve margins were subtle. There was subretinal fluid on macular OCT obtained with initial diagnosis of ischemic neuropathy. (Picture 1A) On fundus fluorescein angiography, (FFA), no staining or leakage was observed on macular region while early leakage with delayed staining was marked at optic disc. While relative afferent pupil defect was not prominent, color vision was

1- Ophthalmologist. Ophthalmology Department of Sultanbeyli Dunyagoz Hospital, Istanbul, Turkey

2- Assistant Prof. MD., Ophthalmology Department of Hisar Hospital, Istanbul, Turkey

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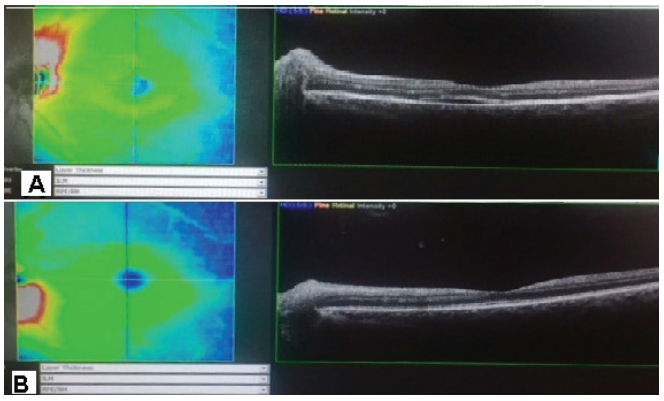
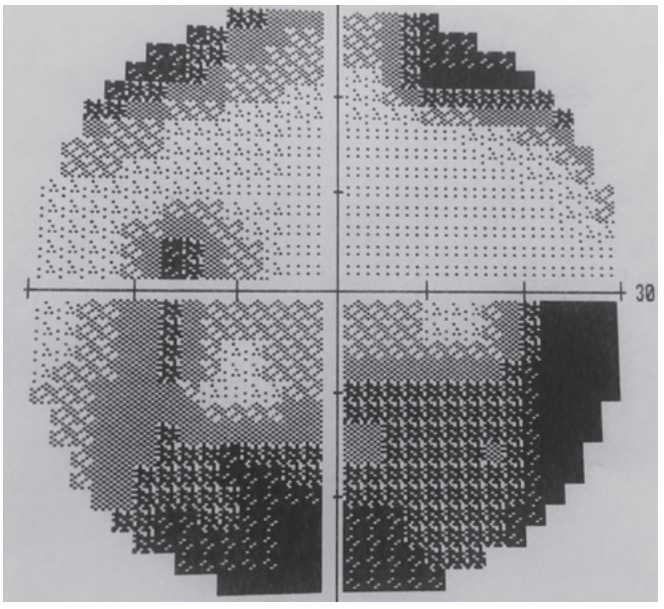
Correspondence Adress:
Ayse Yagmur KANRA
Sultanbeyli Dunyagoz Hospital, Ophthalmology Department, Istanbul, Turkey

Phone: +90 554 588 4945
E-mail: ygurturk@yahoo.com



Picture 1A. *OCT image of first case at presentation. Optic disc edema and submacular fluid are seen.*

Picture 1B. *OCT image of first case at week one. It is seen that submacular fluid was completely resolved and macular was relieved.*



Picture 2. *Visual field of 30-2 of first case at admission. Altudinal defect compatible with ischemic neuropathy is observed in the patient.*

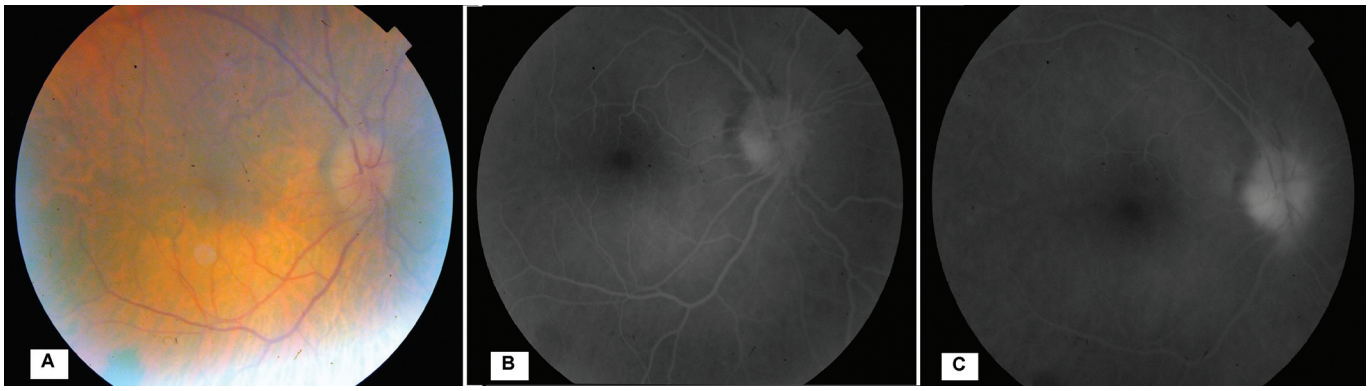
disrupted. In visual field assessment, altudinal defect was observed (Picture 2). No abnormal finding was observed in blood tests regarding systemic disorders and on contrast enhanced orbital and cranial MR imaging studies. The erythrocyte sedimentation rate and CRP levels were normal. Based on these findings, the patient was diagnosed as non-arteritic AION and concomitant subretinal fluid. Nepafanac eye drop was given to maintain therapy since the patient declined oral therapy. After one week, subretnal fluid was completely resolved and visual acuity was improved to 20/32 (Picture 1B)

Case 2

A 67-years old man presented to our clinic with blurry vision without pain over 3 years. In ophthalmological examination, visual acuity was 20/63 in left eye and 20/32 in the right eye with hypermetropia correction (+2.00) while there was no abnormal finding in anterior segment.

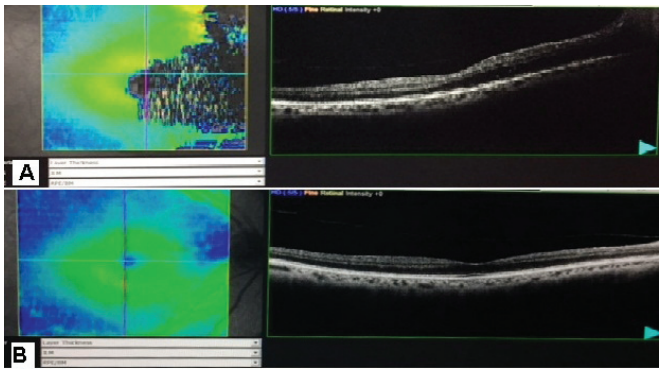
In fundus examination, optic disc was edematous in right eye with subtle margins (Picture 3A) while optic nerve was faded at temporal in left eye. There was subretinal fluid along papillomacular bundle on macular OCT obtained by initial diagnosis of ischemic optic neuropathy (Picture 4A). On fundus fluorescein angiography (FFA) no staining or leakage was observed on macular region while there was early leakage with delayed staining in right optic disc (Picture 3B-C) and staining compatible with previous AION. There was relative afferent pupil defect and impaired color vision.

No interpretable visual field assessment could be obtained. No abnormal finding was observed on contrast enhanced orbital and cranial MR imaging studies while impaired



Picture 3A: *Fundus image of second case presentation. Marked optic disc edema and subtle margins in acute phase of ischemic optic neuropathy.*

Picture 3B-C: *FFA images of second case at presentation. No leakage was observed at macula while early leakage with delayed, marked staining is observed on FFA.*



Picture **4A:** *OCT image of second patient at presentation. Marked edema and fluid along papillomacular bundle is seen.*

Picture 4B: *OCT image of first case on day 10. Complete resolution of subretinal fluid and complete alleviation of macula are seen.*

glucose tolerance was detected in blood tests regarding systemic disorders. The erythrocyte sedimentation rate and CRP levels were normal. Based on these findings, the patient was diagnosed as non-arteritic AION and concomitant subretinal fluid. Oral steroid therapy was initiated according to protocol described by Hayreh et al.3 After 10 days, subretinal fluid was completely resolved while optic disc edema was decreased and visual acuity was improved to 20/20 (Picture 4B).

DISCUSSION

In recent years, structural abnormalities such as subretinal fluid, vitreopapillary traction and micro-cystic retinal changes accompanying to optic nerve disorders have been increasingly recognized by advances in OCT technology.6-8 In a comprehensive review on OCT findings in optic nerve disorders, Tawse et al. emphasized that subretinal fluid secondary to AION was present in 15% of acute cases and discussed origin of subretinal fluid.6 According to authors, subretinal fluid does not directly originate from choroid or retinal vasculature since no marked leakage is observed on FFA. As similar to pupil edema, it is thought that there is a distortion in glial tissue including Kuhnt intermediary tissue acting as a barrier between optic nerve and retina. As such, the fluid seems to pass peripapillary choroid to subretinal space, which, in turn, extends to macular region. Thus, treatment of optic disc pathology will facilitate resolution of subretinal fluid that exacerbates visual symptoms. Thus, we think that FFA imaging is essential in order to exclude other inflammatory pathologies in atypical AION with subretinal fluid..

The compartment syndrome seen in ischemic neuropathy leads release of cytotoxic factors and increase in axonal injury and nerve edema by exacerbating ischemia at optic nerve head.9 As similar to our cases, compartment syndrome may become more prominent in compressed disc due to hypermetropia. It was found that oral prednisolone (80 mg daily) during acute phase provided more rapid resolution of optic disc edema; increasing likelihood of recovery in visual field and acuity compared to follow-up without treatment.6 This results from relief of compartment syndrome and increased blood flow by decompressing capillary at optic nerve head. Thus, we initiated oral prednisolone therapy (80mg daily) in case 2 diagnosed during acute phase and observed improvement in both optic nerve edema and subretinal fluid. This finding support Hayreh et al. reported rapid recovery within 2 weeks in patients treated when compared to those followed without treatment. Although there are large series suggesting that improvement in visual acuity may be spontaneous 10 we think that rapid resolution of subretinal fluid by systemic steroids in eligible cases will be beneficial. First case presented to our clinic beyond acute period; thus, treatment was maintained by topical nepafanac until regression of subretinal fluid since amount of fluid was small and compartment syndrome was not prominent. We think that ischemic neuropathy in addition to age-related macular degeneration, central serous chorioretinopathy and choroidal metastasis should be considered when evaluation etiology of subretinal fluid in patients aged≥50 years, since it may affect treatment modality.

CONCLUSION

Subretinal fluid may arise in patients with non-arteritic AION and may have negative influence on visual acuity. We think that OCT is indicated to show macular involvement and treatment response.

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