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PSEUDO-FOSTER-KENNEDY SYNDROME IN A FIFTY-NINE YEARS-OLD MAN WITH UNCONTROLLED HYPERTENSION AND DYSLIPIDEMIA

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ABSTRACT
Pseudo-Foster Kennedy syndrome is characterized ophthalmoscopically by unilateral atrophy with contralateral optic disc edema with anterior ischemic optic neuropathy as the most common etiology. Nonarteritic anterior ischemic optic neuropathy (NAION) is a major nonglaucomatous optic neuropathy, characterized by a sudden, painless, decrease in vision accompanied with a visual field defect and optic disc edema.
To report a case of Pseudo-Foster Kennedy (PFK) syndrome caused by anterior ischemic optic neuropathy (AION) and describe its clinical particularities, as well as the diagnostic difficulties and treatment.
The case of a 59-year-old male patient with painless visual impairment in both eyes especially in the left one, optic nerve atrophy in his left eye, and optic nerve edema in right eye was described. The sign and symptoms from clinical examination, along laboratory evaluation, OCT and HFA examination support the diagnosis of NAION.
The diagnosis in a PFK presentation is essentially one of exclusion. It is generally accepted that optic nerve impairment in NAION is caused by vascular insufficiency in the capillary bed of the optic disc as patients typically have predisposing vascular risk factors. Although there is no current generally accepted treatment for NAION, a correct diagnosis and supportive treatment may contribute to the improvement in visual acuity (VA) that in this case control of control of blood pressure and cholesterol is the key to treatment.
Keywords: Dyslipidemia, hypertension, NAION, Pseudo-Foster Kennedy Syndrome

BACKGROUND

Optic disc swelling predominantly manifests in conditions directly impacting the front section of the optic nerve, but it also arises from elevated intracranial pressure (known as papilledema) and compression of the optic nerve. Various factors lead to optic disc swelling, including inflammatory or ischemic optic neuropathy, central retinal vein occlusion, uveitis, posterior scleritis, and compression of the optic nerve within the eye socket, typically affecting only one eye. Foster Kennedy syndrome involves papilledema in one eye alongside optic atrophy from optic nerve compression in the other, often stemming from a meningioma at the base of the skull. A similar presentation known as pseudo-Foster Kennedy syndrome occurs with ischemic optic neuropathy, where optic disc swelling in one eye due to a recent episode of ischemic optic neuropathy is accompanied by optic atrophy in the other eye due to a prior episode.[1,2]

Non-arteritic anterior ischemic optic neuropathy (NAION) is a major non-glaucomatous optic neuropathy, characterized by a sudden, painless, decrease in vision accompanied with a visual field defect and optic disc edema.[1,2] It is generally accepted that optic nerve

impairment in NAION is caused by vascular insufficiency in the capillary bed of the optic disc as patients typically have predisposing vascular risk factors.[3,4,5] However, the exact pathophysiology of NAION remains unclear.

This condition is the most common cause of acute and sub-acute optic neuropathies in patients over 50 years of age, with an estimated incidence of 2.3 to 10.3 per 100,000 population.[6] While ischemic pathophysiology is the most cited mechanism for NAION, Parsa and Hoyt have recently proposed a non-ischemic pathophysiology for this condition, attributing NAION to shear force injury following vitreous separation from the optic nerve head.[7] There is no proven effective therapy for NAION. Considering the lack of consensus regarding the exact pathophysiology of NAION, most of the current medical therapeutic approaches are empirical including treatments that address the mechanisms of ischemia, such as thrombosis, insufficient blood supply, and inflammation, or regimens with possible neuroprotective effects.[6,7]

Patients with NAION usually experience painless visual loss, visual field defects, relative afferent pupillary defects (RAPD) may develop unless the optic neuropathy becomes bilateral. NAION is always accompanied by optic nerve papilledema of early onset which may precede visual loss involving the anterior portion of the optic nerve supplied by the posterior brevis ciliary artery circulation. The prognosis of visual function in patients with NAION is very poor because it can cause permanent visual damage to blindness if it has manifested as atrophic optic nerve head.[8]

The incidence of AION is 0.36 per 100,000 whereas the incidence of NAION is 2.3-10.3 per 100,000 in the United States. Almost similar data are also presented in the AAO (American Academy of Ophthalmology) textbook, namely the annual incidence is around 3.6-10.2 per 100,000.[9]

Many studies have shown that disturbances in the balance of serum lipid profiles are closely related to several cardiovascular diseases. Arteriosclerosis induced by dyslipidemia can also affect ocular vasculature and is the most common cause of NAION. NAION is of interest in the field of ophthalmology because of its poor prognosis.[2,8]

CASE PRESENTATION

A fifty-nine years old man came to outpatient clinic with chief complaint blurred vision on both eyes since 1 year ago. Complaints of blurring were more severe in the left eye. A year ago, left eye suddenly felt blurry. A history of eye pain, diplopia, and red eye was denied. The patient underwent cataract surgery 1 year ago, blurred vision in the left eye was getting worse after surgery. The patient has a history of hypertension and cardiomegaly due to hypertension in the last 1 year, but not taking any medication because he assumed that this condition has been under control. History of other systemic disease, wearing glasses, and trauma was denied.

From the general examination, patient revealed normal vital sign. There were no enlarged lymph nodes, myalgia, stiff neck, painful swallowing or abdominal pain.

During his first visit examination revealed his visual acuity of right eye was 5/40, and left eye 2/60 with best corrected visual acuity of right eye with sphere -2.25 was 5/15 and left eye still 2/60. Color vision examination with Ishihara was 38/38 in right eye and 1/38 in left eye. Intraocular pressure of the right eye was 17.3 mmHg, left eye 14,6 mmHg. Anterior segment (Fig.1) showed rounded pupils, isochoric 3

mm diameter, normal light reflex, and left eye relative afferent pupillary defect. The patient was pseudophakia in left eye and has cataract in the right eye (LOCS NO2NC2C1P4). Ocular motility on the both eyes was normal with no pain within movement. Posterior segment of left eye showed pale and blurred margin optic disc (Fig.3). Right eye posterior segment showed blurred margin of optic disc in the superior area with slightly hyperemic color. In retina, there were AV crossing nipping, copper wire with AV ratio 1:3 on both eyes which were signs of hypertensive retinopathy.

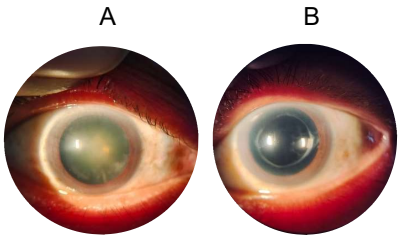

Figure 1. (A) Right eye showed cataract LOCS NO2NC2C1P4 (B) Left eye pseudophakia (Picture taken with patient's consent. Courtesy of RSUD Dr Soetomo)

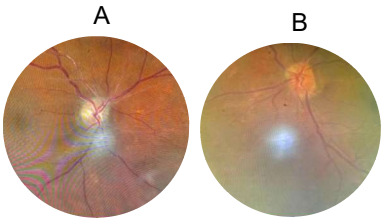

Figure 2. (A) Fundus photograph of right eye, visualization was not proper due to cataract, the optic nerve color was slightly hyperemic with blurred margin. (B) Fundus photograph of left eye showed pale and blurred margin optic disc. (Courtesy of RSUD Dr Soetomo)

Optical coherence tomography revealed a decreased of RNFL thickness in superior area in both eyes. Ganglion cell analysis revealed thinning in superotemporal area on right eye and generalized thinning on left eye. On examination of the visual field with Humphrey visual field analyzer (figure 3), an altitudinal defect was found in both eyes, with the left eye defect being wider than the right eye.

From systemic examination, Stage II hypertension was found in this patient, there were no jaw or tongue claudication, scalp tenderness, malaise and loss of weight. Blood investigation was done to rule out other predilecting factor. His investigations showed normal full blood count, normal liver, slightly increased renal function test, and dyslipidemia with total cholesterol, HDL, LDL, Triglyseride count are 243, 65, 151, 240, respectively.

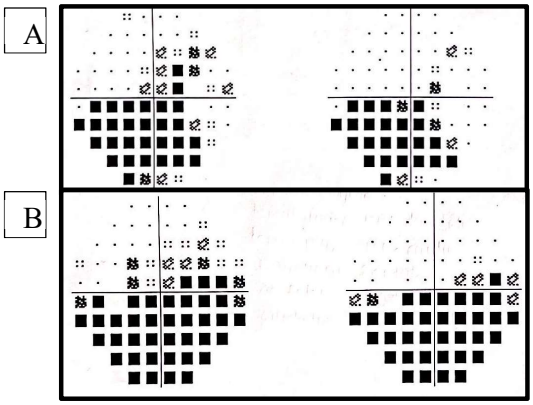

Figure 3 Humphrey Visual Field Analyzer Result on right eye (A) showed altitudinal defect and left eye (B) showed full blown altitudinal defect. (Courtesy of RSUD Dr Soetomo)

On the basis of clinical findings and laboratory result, patient was diagnosed as pseudo-foster kennedy syndrome caused by Nonarteritic Anterior Ischemic Optic Neuropathy on both eyes, with left eye was more severe than right eye. Patient was later treated with 50 mg vitamin B1 every 12 hours, 10 mg vitamin B6 twice a day, vitamin B12 50 mcg every 12 hours, folic acid 1 mg once a day, and cithicolin 500 mg twice a day. Consultation to the department of internal medicine and cardiology was ordered to treat the patient's risk factors.

In response to the consultation from the cardiology department, the patient was assessed for Grade 3 hypertension with hypertensive heard disease NYHA FC1. The patient was treated with adalat oros 1x30 mg, lisinopril 1x10 mg, and concor 1x2.5 mg. From the results of consultation with the department of internal medicine, the patient was diagnosed with dyslipidemia, hyperuricemia, acute kidney injury without emergency signs that could be related to hypertensive heart disease, and JNC VII stage II hypertension. The patient was treated with simvastatin 1x20 mg and allopurinol 1x100mg.

The patient then further underwent cataract extraction with phacoemulsification technique. Complaints of blurring in the right eye felt reduced. Postoperative visual acuity in right eye 5/20 with spherical correction -1.00D improved to 5/6. On examination, the posterior segment of the right eye revealed a normal color optic nerve head with blurred margin. Patient was educated to control routinely and use medication as suggested to control the risk factors for NAION.

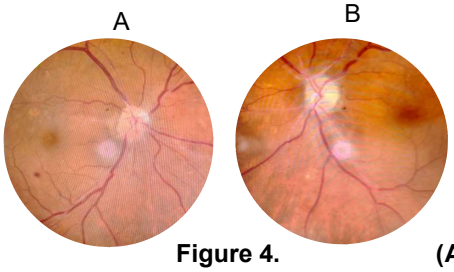

Figure 4. (A) Postoperative fundus photograph of right eye, the optic nerve color was normal with blurred margin. (B) Fundus photograph of left eye showed pale and blurred margin optic nerve head. (Courtesy of RSUD Dr Soetomo)

DISCUSSION

A fifty-nine years old man came to outpatient clinic with chief complaint blurred vision on both eyes since 1 year ago, with left eye felt more blurred than the right one. From the physical examination, RAPD on left eye was found. Posterior segment examination revealed pale and blurred margin optic disc on left eye and normal color and blurred margin optic disc on the right eye. The patient had uncontrolled hypertension and dyslipidemia. Humphrey visual field analysis result showed altitudinal defect on both eyes, with left eye was more severe than right eye. Macula and ONH OCT revealed thinning in some areas. In ganglion cell analysis result, thinning in superotemporal sector on right eye and generalized thinning on the left eye were found.

Optic nerve ischemic syndrome (Ischemic Optic Neuropathy, ION) is classified according to the location of the ischemic damage and the causes of the ischemia. Anterior ischemic optic neuropathy (AION) is a syndrome involving ONH with optic nerve papilledema seen on posterior segment examination. Posterior ischemic optic neuropathy (PION) includes conditions involving the intraorbital, intracanalicular, or intracranial regions of the optic nerve without visible optic nerve papilledema. Based on the etiology, ION is divided into arteritic and non-arteritic.[10]

The diagnosis of optic neuropathy is usually considered under two circumstances: (1) when visual loss is associated with an anomalous, swollen, or pale optic disc or (2) when the fundus examination is normal but deficits in acuity, color, and visual field are accompanied by an afferent pupillary defect. When the history and examination are typical of optic neuropathy (visual acuity and color vision loss, decreased contrast sensitivity, afferent pupillary defect, and typical visual field defect), four different diagnostic groups should be considered, based upon the ophthalmoscopic appearance of the disc: anomalous, swollen, normal, and pale. In summary, most cases of suspected

optic neuropathy, careful consideration of historical details, general examination, and the ophthalmoscopic appearance of the optic nerve head will lead to the correct diagnosis long before any ancillary tests are ordered. [11]

Ischemic optic neuropathy is caused by infarction of the optic nerve. Anterior ischemic optic neuropathy is caused by infarction of the retrolaminar optic nerve (the region just posterior to the lamina cribrosa) from occlusion (eg, giant cell arteritis), thrombosis, or more commonly, decreased perfusion (eg, non-arteritic type) of the short posterior ciliary arteries. It causes acute loss of vision with optic disk swelling in all cases. In the rare posterior ischemic optic neuropathy due to infarction of the retrobulbar optic nerve, there are no optic disk changes in the acute stage. Optic atrophy develops after both anterior and posterior ischemic optic neuropathy.[12]

Ischemic optic neuropathy (ION) describes a state of hypoxic injury of the optic nerve. Clinically, ION is divided into anterior and posterior forms defined by the presence or absence of optic disc swelling, respectively. The site of vascular occlusion for anterior ION from giant cell arteritis is the short posterior ciliary arteries, but mechanical vascular obstruction does not play a role in most non - arteritic cases. Histologically, ION is characterized by axon and glial necrosis, edema, and a sparse mononuclear response.[13]

The optic nerve may be damaged by ischemia anywhere from its visible portion at the back of the eye to its intracranial transition into the optic chiasm. Of conditions classified as ischemic optic neuropathy, involvement at the optic nerve head (anterior ischemic optic neuropathy [also known as AION]) is far more common than involvement behind the eye (posterior ischemic optic neuropathy [also known as PION]). Anterior ischemic optic neuropathy is traditionally divided into those cases that are associated with vasculitis, usually giant cell arteritis (GCA), and those that are not.[14]

Anterior ischemic optic neuropathy is the most common cause of acute opticnerve damage in persons older than 50 years of age. It often leads to significant permanent visual impairment in affected eyes and whether associated with arteritis or not, anterior ischemic optic neuropathy is typically painless. Visual field impairment is usually altitudinal, with the deficit usually worse below the visual horizon, while visual acuity is variably affected and generally worse in arteritic anterior ischemic optic neuropathy than in non - arteritic anterior ischemic optic neuropathy. Nearly all cases of arteritic AION are due to giant cell arteritis, while the pathogenesis of non - arteritic AION is unsettled, but the clinical diagnosis encompasses several distinct, albeit uncommon, sources of injury including thromboembolism, systemic hypotension, and atherosclerotic vascular occlusion. AION begins suddenly, sometimes first noted upon awakening, but may slowly progress to peak visual loss over a week or more.[13,14]

More common differential diagnosis for ischemic optic neuropathy are other vascular phenomena, such as retinal artery and vein occlusions, optic neuritis, and acute optic nerve compression. These conditions can almost always be distinguished by a thorough history and an examination that includes visual and pupillary function and funduscopy. Given the high risk of second eye involvement in arteritic anterior ION and the effectiveness of systemic corticosteroids in preventing it, an expedited evaluation, including erythrocyte sedimentation rate, C-reactive protein, platelet count, and temporal artery biopsy, is indicated. Posterior ION results in similar symptoms and clinical findings as

anterior ION with the exception of disc edema.[13,14]

NAION is the most common cause of optic neuropathy in adults over 50 years of age. The prevalence of NAION in the US has been estimated to be anywhere between 2.3 to 10.2 per 100.000. It is less common in blacks and is most common in Caucasians presumably because blacks tend to have a larger cup to disc ratio and are thus less likely to have small optic nerve cups, which is the biggest risk factor for developing NAION. Complete neuro-ophthalmological history should be obtained emphasizing the onset of visual loss (typically sudden in NAION and semi-acute in optic neuritis) and any other associated symptoms (about 10-15% of patients with NAION experience pain in and around the eye but not with eye movements as is typical in optic neuritis), there usually aren't any other accompanying neurological symptoms.[16,17]

NAION typically presents with acute unilateral painless vision loss accompanied by sector or diffuse optic nerve edema. It generally affects people over 50 years of age, with a mean onset between 57 and 65 years of age, however, it has also been reported in patients <40 years old both with and without vasculopathic risk factors. Visual field (VF) defects following a nerve fiber layer distribution are typical VF findings, with inferior altitudinal and arcuate defects being the most common.[18]

The chief complains of patients with NAION usually is acute painless unilateral or sometimes bilateral visual loss that is often described as blurring or clouding of vision, often inferiorly, sometimes even more widely. Although most patients have no accompanying pain, headache or periocular pain is reported in 8-12% of patients which can be difficult to distinguish from optic neuritis. Two-thirds of NAION patients report loss of vision upon awakening. Vision loss was often reported upon awakening, and some authors believe that the etiology for NAION is nocturnal hypotension. Patients may describe the vision loss as a “dim or blur”, especially in the area of the field loss, and it is variable but typically less severe than the loss from arteritic anterior ischemic optic neuropathy (AAION). [19]

The occurrence of NAION in both eyes resulted in the clinical manifestation of the pseudo-Foster-Kennedy syndrome, in which the previously affected ONH was atrophic and now the involved ONH was edematous. The diagnosis of NAION is clinical. Patients who present with a typical history of acute, painless, unilateral vision loss, and who have classic findings on examination with risk factors finding can be managed according to the treatment of this condition.[17,18]

NAION should be differentiated from optic neuritis, especially in younger patients. In unclear cases, contrast MRI of the orbit (with fat suppression) can help differentiate. The affected optic nerve appears normal in NAION (95% of cases) but has high contrast in optic neuritis (90% of cases). Nervous system imaging should also be performed in any patient presenting with atypical symptoms including those with prolonged optic disc edema or progressive and/or recurrent visual loss more than two months after initial presentation to exclude inflammatory or compressive lesions. OCTA may be useful in diagnosing and predicting the severity of NAION based on blood flow to the optic disc. In this case, MRI was not performed because clinical findings and other diagnostic tests have led to establishment of NAION.[10,11]

Patients with NAION might have a variety of visual field defects although many will have an altitudinal defect supporting the notion that there are two

separate semicircles of short posterior ciliary arteries supplying the superior and inferior portions of the optic nerve. Visual acuity can vary from 20/20 to no light perception although very poor visual acuity is uncommon and should raise the suspicion for giant cell arteritis. While most patients with NAION are over 50 years of age, many are younger and young age at presentation does not exclude the diagnosis. If the differentiation between NAION and other optic neuropathies is difficult, MRI of the orbits with gadolinium can be useful as it should be normal in all patients with NAION. [16,17]

Although the exact pathogenesis of NAION remains unproven, it appears to be a multifactorial disease. Recent studies of the optic nerve head utilizing optical coherence tomography (OCT) have demonstrated thicker prelaminar tissue in NAION patients. Systemic diseases that may cause decreased perfusion to the optic nerve head secondary to microvascular compromise might increase the patient's risk of NAION, these include hypertension, diabetes, and hypercholesterolemia. Other risk factors noted in the literature are nocturnal hypotension, smoking, obstructive sleep apnea, anemia, hypercoagulable states, disc drusen, ocular and nonocular surgery, and migraines.[15,18] In this patient, the risk factors that found are hypertension and dyslipidemia.

Nonarteritic anterior ischemic optic neuropathy (NAION) is a potential cause of irreversible vision loss. Usually at presentation one is affected, however subsequent development of the same condition in the fellow eye is not uncommon; occurring in approximately 15% of patients within a 5-year period. Presumably, having similar optic disk anatomy in both eyes and exposure to the same vasculopathic risk may result in bilateral involvement of both optic nerves. Diabetics and patients who suffered significant visual loss following the first event are at increased risk for bilateral sequential involvement. Thus, patients suffering from unilateral NAION are naturally concerned about the imminent possibility of losing vision in their other eye, and are often interested to know if it is possible to predict the visual outcome following the second event, should it occur, based on the initial presentation.[20]

Although a progressive form of NAION has been reported, the course of NAION typically stabilizes within 2–3 months. The visual acuity may improve by up to three lines in 43% of patients, while VF defects are less likely to improve. There is <5% chance of recurrence in the same eye. The initial disc edema usually resolves and sector or diffuse atrophy ensues typically within the next 6–11 weeks. There is up to a 15% chance of fellow eye involvement at 5 years. The VA and VF defect in a patient with prior NAION cannot directly predict the prognosis of visual impairment if there is second eye involvement. While many medications and treatment strategies have been tried over the years, none have been proven to be effective. One of the very few randomized controlled clinical trials in neuro-ophtalmology was done evaluating whether patients with NAION will benefit from optic nerve head decompression via vitrectomy and demonstrated that surgery was not beneficial and potentially harmful. Intravitreal injections of bevacizumab, as well as triamcinolone, have been tried with disappointing results as well.[16,18]

The use of neuroprotection agents in ischemic stroke and various types of optic neuropathies has been considered from time to time. In optic neuropathies, axonal injury and degeneration result in secondary retinal ganglion cell (RGC) death. So far, in experimental studies, neuroprotectors

have been shown to protect and improve survival of the RGCs. In optic neuropathies, since the primary lesion is injury and degeneration of the axons, resulting in secondary damage of the RGC bodies, simply preserving the RGC bodies via neuroprotection cannot improve function if the axon is damaged, because the cell body is still disconnected from its target in the brain. Moreover, in NAION, 100% of the axons comprising the optic nerve do not die, only a portion of optic nerve fibers are damaged; the remaining surviving, viable axons are responsible for the residual vision and any subsequent improvement that may take place.[3]

The rationale for the use of steroids in NAION is based on a study published in the late 1960s in which several case series demonstrated improved visual outcomes in patients with NAION treated with steroid therapy. Theoretically, the effect of steroid therapy could be attributed to decreased compression of capillaries in the optic nerve head as a result of decreased edema and increased blood flow to the optic nerve head. Based on these results, steroids are selected as a treatment option for NAION by many physicians, although conflicting results regarding its benefit have been reported over the past 2 to 3 decades. Newest systemic review and meta-analysis result revealed that there was no significant change in BCVA after steroid administration.[3] Steroid therapy in this patient was not given because it has been proven not improved symptoms and visual acuity.

Untreated NAION generally remains stable after visual function reaches a low point, but one study showed that 43% of patients with visual acuity worse than 20/64 at initial presentation regained at least three lines of visual acuity on the Snellen chart within six months. Repeated episodes of vision loss in the same eye after three months are not uncommon in NAION (up to 6.4%) and a more holistic evaluation of the underlying systemic disorder or differential diagnosis of optic neuropathy should be considered.[15]

CONCLUSION

Pseudo-Foster Kennedy (PFK) syndrome refers to a constellation of unilateral optic disc edema and contralateral optic atrophy in the absence of any compressive optic nerve lesion. The diagnosis in a PFK presentation is essentially one of exclusion. Patients with NAION, such as suspected in this case report, are at risk for recurrence in fellow eye, thereby presenting as PFK syndrome. NAION should be considered as a differential diagnosis especially when imaging and other laboratory investigations are not suggestive of any compressive lesion.

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