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Pseudo Foster Kennedy Syndrome with Parkinson's disease: A Case Report

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Abstract: This case report describes a 70-year-old male patient who reported experiencing sudden blurred vision in his right eye over the past week. The patient has been managing glaucoma for five years, during which he underwent peripheral iridotomy in both eyes, but he has not been on any anti-glaucoma medications. Furthermore, he has been living with diabetes mellitus for 20 years and is also dealing with Parkinson's disease. After many tests, such as a fundus examination, perimetry, OCT, MRI scan, and fundus photography, it was found that the patient had papilledema in the right eye due to ischemic optic neuropathy and primary optic atrophy in the left eye due to Non-Arteritic Anterior Ischemic Optic Neuropathy (NAION). This led to the diagnosis of pseudo-foster Kennedy syndrome (PFKS).

Keywords: Pseudo Foster Kennedy syndrome (PFKS), Glaucoma, Optic atrophy, and papilledema.

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INTRODUCTION

Foster-Kennedy syndrome is a rare clinical condition characterized by the presence of unilateral optic disc swelling along with contralateral optic nerve atrophy. This syndrome typically arises due to an intracranial mass, such as a tumor, which exerts compression on the optic nerve, leading to these distinctive ocular manifestations [1, 2]. However, when similar findings are observed without an identifiable intracranial mass, the condition is classified as Pseudo-Foster-Kennedy syndrome [4]. Pseudo-Foster-Kennedy syndrome can be attributed to an array of conditions that affect the optic nerve and surrounding structures. Among the most common causes are Non-Arteritic Anterior Ischemic Optic Neuropathy (NAION) and Arteritic Anterior Ischemic Optic Neuropathy (AAION)which represent two distinct pathological processes affecting the blood supply to the optic nerve [5,6]. Other notable causes include pachymeningitis, incidental trauma, optic neuritis, infectious processes such as syphilis, and systemic vascular conditions like subdural hematoma and severe arteriosclerosis of the internal carotid arteries [7-10].Nutritional deficiencies, particularly vitamin B12 deficiency and low folate levels, as well as metabolic disorders such as hypertension, diabetic mellitus, hyperhomocysteinemia, and diabetic papillopathy also contribute to the development of this syndrome [11,12]. In this report, we present a unique case of a patient diagnosed with Parkinson's disease and angle closure glaucoma who exhibited manifestations consistent with the pseudo-Foster-Kennedy syndrome. This case highlights the potential for various neurological conditions to produce similar optic nerve findings, emphasizing the need for a thorough clinical evaluation and a differential diagnosis approach when encountering such ocular presentations. We aim to understand the pseudo-Foster-Kennedy syndrome, particularly in neurodegenerative disorders [14] and to underscore the importance of recognizing the interplay between systemic and neurological diseases in ophthalmic presentation.

CASE SUMMARY

A 70-year-old male patient presented with a sudden onset of blurred vision in his right eye (RE) over 1week. The patient had a history of glaucoma for 5 years had undergone peripheral iridotomy in both eyes and had not utilized any anti-glaucoma medication. Additionally, he had a 20-year history of diabetes mellitus (DM) and Parkinson's disease. His uncorrected visual acuity (UCVA) was 6/36 in the right eye and 6/24 in the left eye (LE). In slit lamp examination, the pupil showed a relative afferent pupillary defect in both eyes. Following fundus examination, perimetry, MRI scan, and OCT investigations, the patient received the following diagnoses: 1. age-related nuclear cataract in both eyes (BE), 2. chronic angle-closure glaucoma in both eyes, 3. primary optic atrophy in the left eye

secondary to non-arteritic anterior ischemic optic neuropathy (NAION), and ischemic optic nephropathy (ION) in the right eye secondary to NAION.

DISCUSSION

The clinical presentation observed in this patient is consistent with Foster-Kennedy syndrome. In Foster-Kennedy Syndrome, one optic nerve is atrophied, and the other optic nerve exhibits disc edema (papilledema) due to an intracranial mass such as a suprasellar mass [3]. In this case, the damage to the optic nerves is caused by chronic angle closure glaucoma and NAION rather than intracranial mass, so the patient has pseudo-foster Kennedy syndrome. Patil *et al*. concluded that "NAION in one eye can cause recurrence in the fellow eye also present as pseudo foster Kennedy syndrome" [4].

The visual field of the patient was assessed in which the Central 24-2 threshold program of each eye was assessed with PI standard. The examination of the GHT reveals outside the normal limit in both eyes. The mean deviation was -22.27DB in the right eye and -30.73 dB in the left eye the pattern deviation was 6.06 dB in the right eye and 4.50 dB in the left eye. These results indicate a reduction in the patient's macular field of vision due to optic atrophy in the left eye.

The central visual field was assessed using the HFA 10-2 program with the SITA standard. The examination of the GHT revealed results outside the normal limit in both eyes. The mean deviation was -26.71 dB in the left eye and -20.83 dB in the right eye. The pattern deviation was 9.21 dB in the left eye and 7.09 dB in the right eye. These results indicate a reduction in the patient's macular field of vision due to optic atrophy in the left eye and papilledema in the right eye.

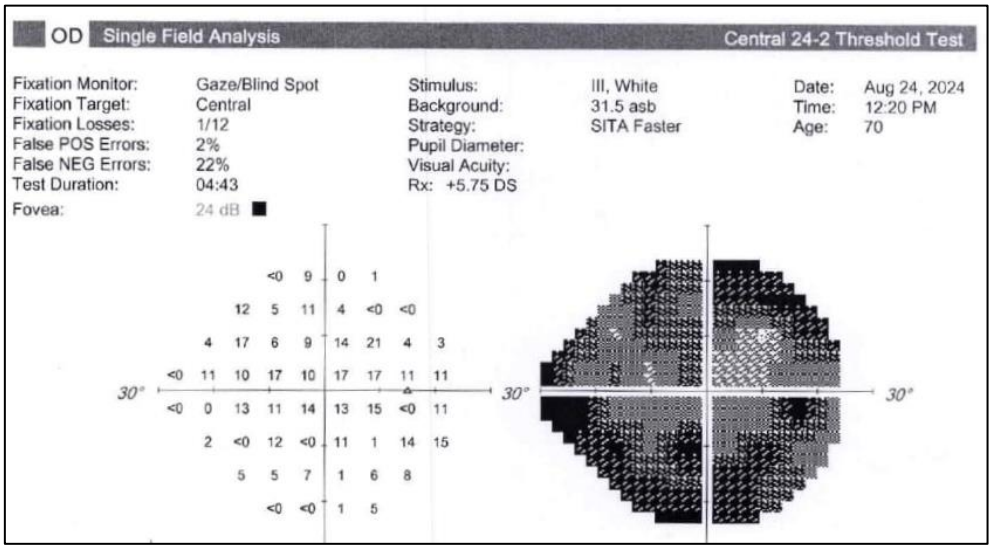

Fig-1: HFA 24-2 OD macular program depicts unreliable visual field loss due to impairment of fixation

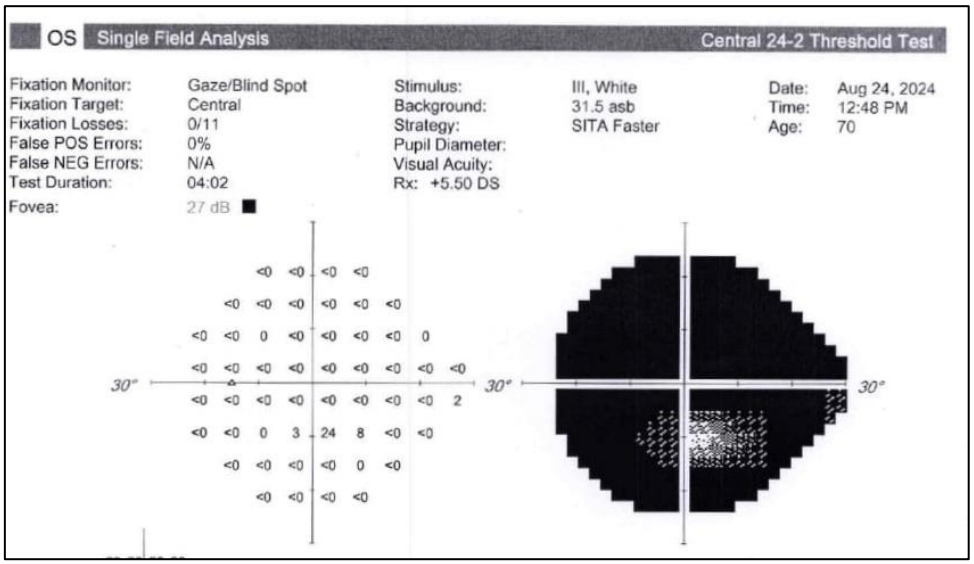

Fig-2: HFA 24-2 OS macular program depicts unreliable visual field loss due to impairment of fixation

Visa Rene *et al*., in their studies, concluded that visual field loss is present in the eye with optic atrophy, and the other eye is normal despite optic disc edema[13]. However, in our case, due to the severity of Parkinson's disease, the patient was unable to do the visual field assessment, which resulted in unreliability.

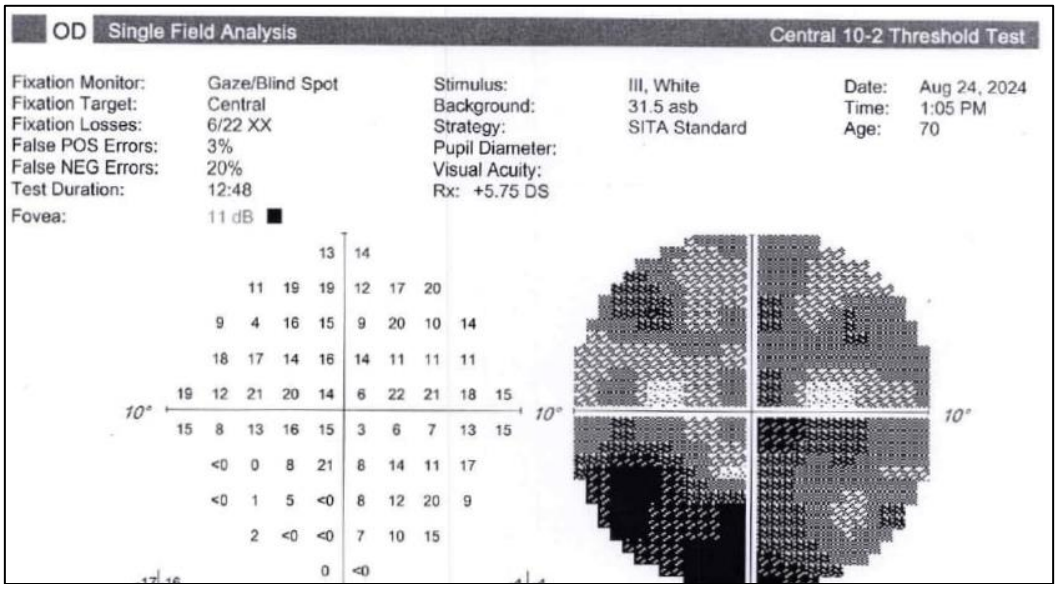

Fig-3: HFA 10-2 -OD depictsunreliable visual field loss due to impairment of fixation

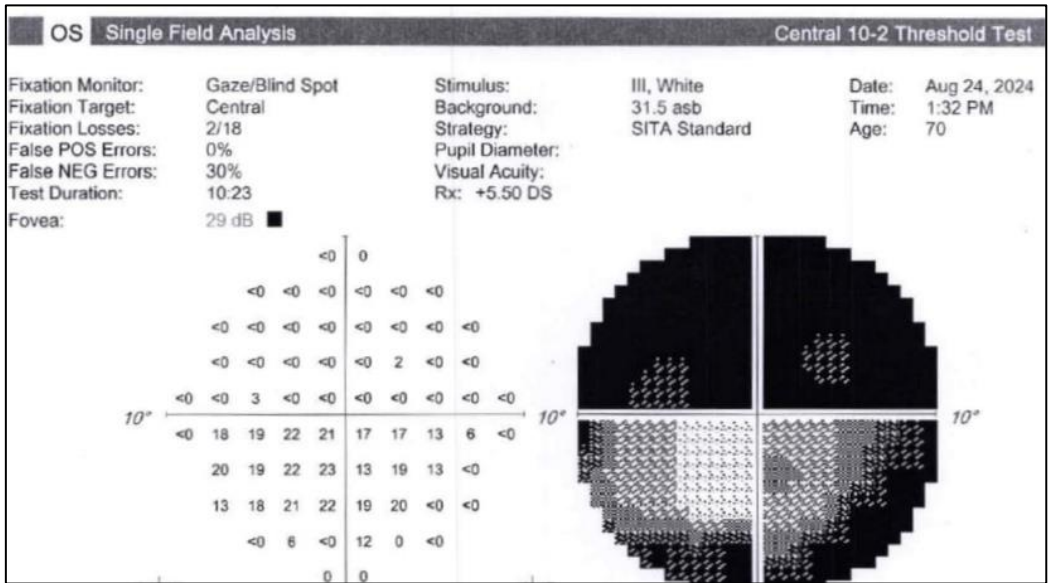

Fig-4: HFA 10-2 -OS depicts altitudinal field loss

In addition to that, the patient in our case has Parkinson's disease. Patients with Parkinson's disease have the following ophthalmic features, such as ocular surface irritation, altered tear film, visual hallucinations, blepharospasm, decreased blink rate, and decreased convergence amplitudes which were more common in Parkinson's disease patients [14]. Parkinson's patients also have retinal disorders such as retinal degeneration, retinal layers thinning, and macula used to have reduced thickness [15,16]

Verghese *et al*. concluded in their case-control study that" macular volume has significant thinning" [17,18]in Parkinson's disease. Our study also showed that the OCT macula scan of the left eye, the macular thickness analysis program, indicated a normal central subfield thickness of 251 µm, a normal outer temporal quadrant (250 µm), decreased thickness in the inner temporal quadrant (278 µm), the inner superior quadrant (289 µm), the inner nasal quadrant (284 µm), the inner inferior quadrant (284 µm), the outer superior quadrant (248 µm), the outer nasal quadrant (257 µm), and the outer inferior quadrant (243 µm**)**.These values showed thinning in macular thickness in the patient's left eye.

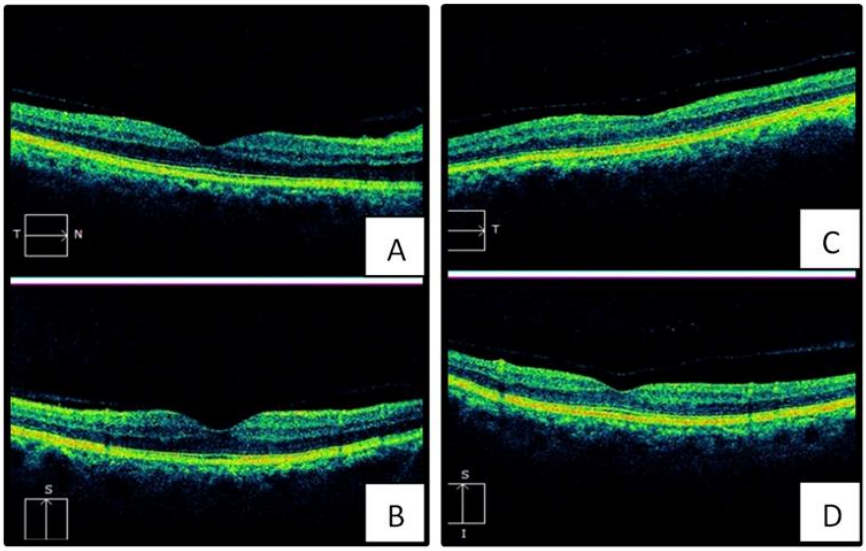

Fig-5: OD (A and B): OCT MaculaFig .6:OS (C and D): OCT Macula depicts Papilledema depicts optic atrophy

OCT macula scan of the right eye, the macular thickness analysis program revealed a normal central subfield thickness of 285µmandnormal thickness in the inner temporal quadrant (327µm), inner superior quadrant (341µm), inner nasal quadrant (347µm), inner inferior quadrant (313µm), outer temporal quadrant (266µm), outer superior quadrant (283µm), normal outer inferior quadrant (254µm), and increased outer nasal quadrant (337µm).These values showed the thickening of the outer nasal quadrant of the macula in the right eye.

A comparison of the right and left eye OCT macula scans showed that macular thinning was more pronounced in the left eye, with only the central subfield and outer temporal quadrants exhibiting normal thickness. This suggests reduced macular thickness in the left eye due to NAION. In addition to that, the patient has Parkinson's disease and increased outer nasal quadrant thickness in the right eye due to ischemic neuropathy [19].

Orbit screening of our studies showed altered hyper-intense signal change involving the left retro bulbar optic nerve near the orbital apex in T2W and STIR images, which are suggestive of acute/subacute left optic neuritis causing optic atrophy in the left eye. Also in the studies of Desai *et al.***,** orbital and brain MRI reveals left lateral ventriculomegaly with downward displacement of the gyrus rectus, resulting in left optic nerve compression causing optic atrophy in the left eye showing the absence of intracranial lesions [20]

The B-scan report shows a normal posterior segment in both eyes. Semeraro *et al*., in their studies, showed normal B-scan reports in PFKS [21].

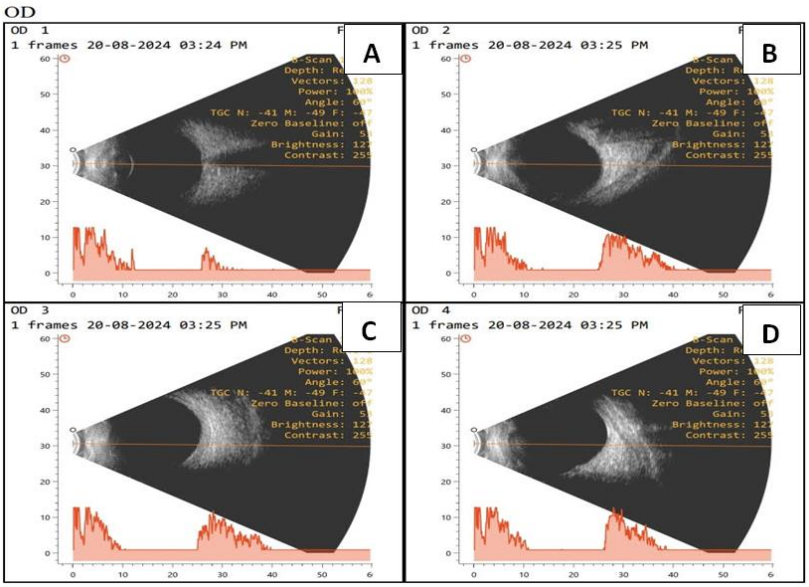

Fig. 7: OD (A, B, C, D) depicts a normal B-scan ultrasound report.

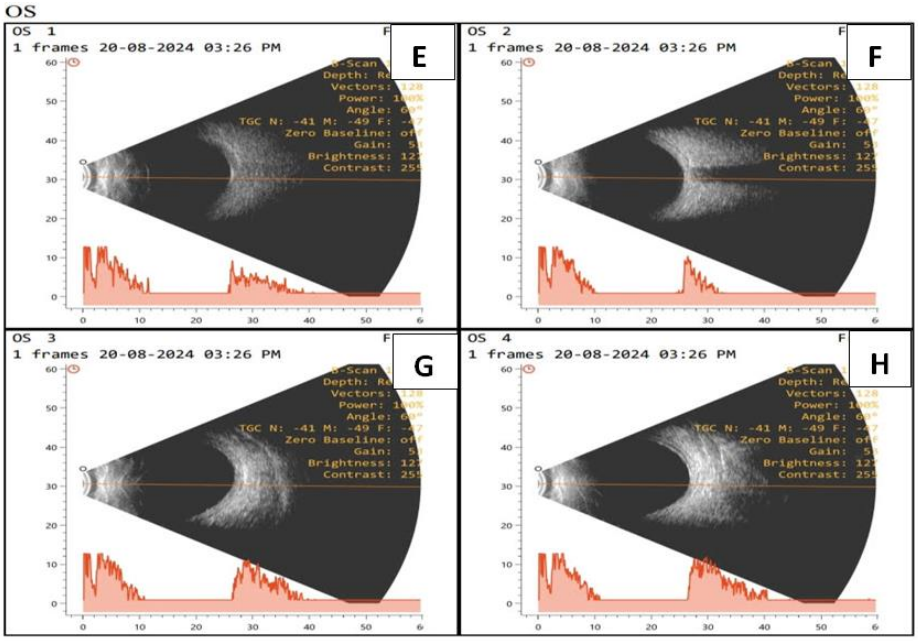

Fig. 8: OS (E,F,G,H) depicts a normal B-scan ultrasound report

In the OCT retinal nerve fiber layer (RNFL) scan of the right eye, the average RNFL thickness was normal at 80 µm, with reduced RNFL symmetry at 51%. The rim area and disc area were both normal, with an average cup-to-disc (CD) ratio of 0.56 mm and a normal cup volume.

In the OCT RNFL scan of the left eye, the average RNFL thickness was reduced to 63 µm, with reduced RNFL symmetry at 51% and a reduced rim area of 0.81 mm^2. The average CD BEratio was reduced to 0.77 mm, the vertical CD ratio was reduced to 0.74mm, and the cup volume was normal.

The OCT RNFL results indicated that the reduced RNFL symmetry in the right eye was due to ischemic optic neuropathy and chronic angle closure glaucoma, while the reduced RNFL thickness in the left eye was attributed to optic atrophy and chronic angle closure glaucoma. Bansal *et al*. also concluded that RNFL thinning is present in optic atrophied eyes and relatively normal RNFL thickness in another eye [22]. In our study, the RNFL thickness of the right eye was relatively normal except for RNFL symmetry thinning due to the patient having Parkinson's disease. Elan War *et al*. concluded that in Parkinson's disease, the thickness of RNFL, the ganglion cell layer, and the peripapillary thickness reduced [23].

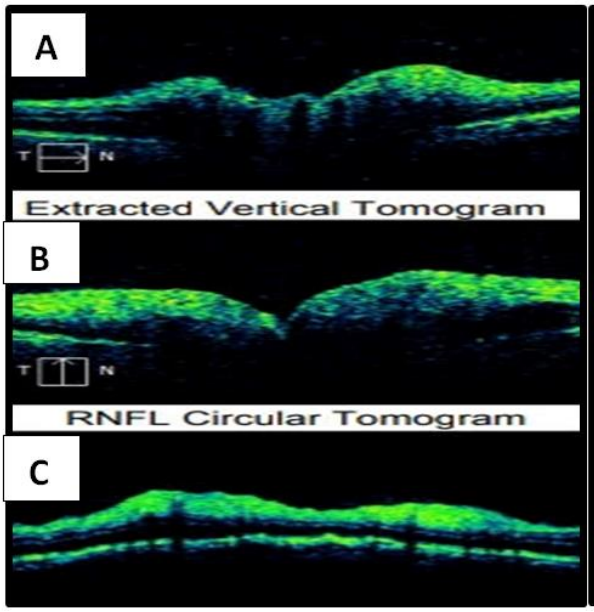

Fig.9: OD(A, B, C) OCT RNFL depicts Papilledema

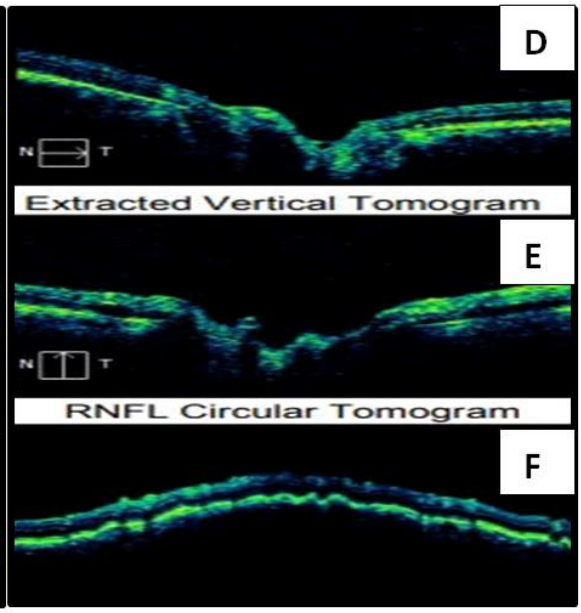

Fig.10: OS (D, E, F) OCT RNFL depicts optic atrophy

The fundus examination identified optic disc edema and sphincter hemorrhages in the RE NAION and consecutive optic atrophy in the LE due to NAION resulting from prior primary angle-closure glaucoma (PACG) and age-related nuclear cataract in both eyes (BE).

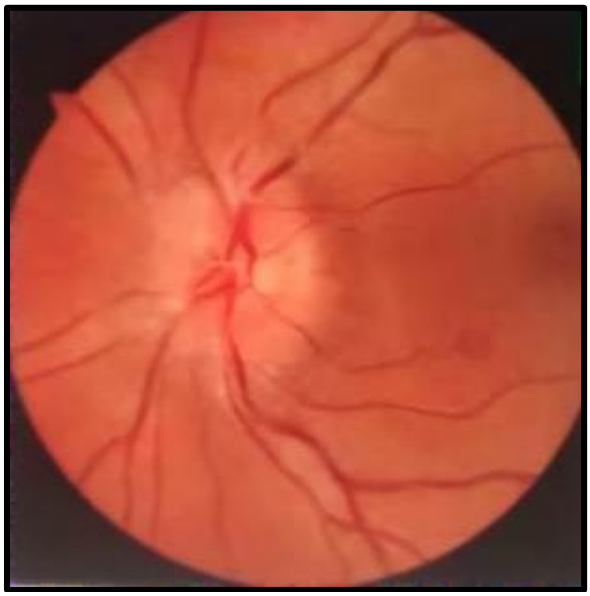

Fig-11: RE Papilledema

Fig-12: LE Optic atrophy

David *et al*., in their studies, revealed that a common cause for PFKS was due to NAION. There is no generally accepted treatment for NAION, but oral corticosteroids have shown improvements in visual acuity and visual fields in treated and untreated cases [24].

CONCLUSION

The patient with NAION was closely monitored, and no relapses have been observed. As of now, there is still no universally accepted treatment for this condition. After the intravenous prednisolonecourse and tablet Wysolone 20 mg and under anti-glaucoma medication, the patient's UCVA was increased to 6/24 and 6/18. After 3 weeks of follow-up, the patient's UCVA was increased to 6/18 in both eyes.

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ACRONYMS

- AAION -Arteritic Anterior Ischemic Optic Neuropathy
- DM - Diabetes Mellitus
- GHT - Glaucoma Hemi field Test
- HFA- Humphrey Field Analyser
- NAION - Non-Arteritic Anterior Ischemic Optic Neuropathy
- OCT - Optical Coherence Tomography
- PACG - Primary Angle Closure Glaucoma
- PFKS - Pseudo Foster Kennedy Syndrome
- PI - Peripheral Iridotomy
- RNFL - Retinal Nerve Fiber layer
- SITA - Swedish Interactive Threshold Algorithm
- STIR - Short TI (Tau) Inversion Recovery

UCVA - Uncorrected Visual Acuity