



## DENTAL SEPSIS AS A LIKELY CAUSE OF NEURORETINITIS : A RARE CLINICAL SCENARIO

### Ophthalmology

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### ABSTRACT

A 39-year-old male presented to the ophthalmology clinic with progressive painless loss of vision in the left eye for 20 days. There was no history of systemic illness. Vision was 6/6; N6 in the right eye and 6/60 <N36 in the left eye. Anterior segment examination showed grade I relative afferent pupillary defect (RAPD) in the left eye. On fundus examination macular edema, with hard exudates in a macular star configuration was seen on the left eye. Complete blood counts, cerebrospinal fluid analysis and autoimmune workup were normal. **MRI brain showed a post contrast enhancement of optic nerve on T1 images and a mildly thickened, tortuous optic nerve on T2 images. Based on clinical features, a diagnosis of neuro-retinitis was made.** Gingival swab and blood culture showed growth of *Enterococcus faecium*. The patient was started on double dose of antibiotics along with cortico-steroids. The patient improved symptomatically and best corrected visual acuity (BCVA) was 6/9 after 45 days.

### KEYWORDS

Neuro-retinitis, Enterococcus, Dental foci

### INTRODUCTION

Inflammatory disorders of the optic nerve may present as papillitis, retrobulbar neuritis or neuro-retinitis. Neuro-retinitis is a focal inflammatory disorder affecting the optic nerve head and macula. It can occur due to infectious causes such as **cat scratch disease which is the most common etiology**<sup>1</sup>. Other common infectious agents implicated are Herpes Zoster, Hepatitis B, Mumps, HIV, HBV, Toxoplasma, Toxocariasis, Histoplasmosis, Syphilis, Leptospirosis, Lyme disease, and tuberculosis. It could be due to non-infectious aetiology such as immune-mediated neuro-retinitis and Leber's stellate neuro-retinitis or could be idiopathic in nature. It presents with acute unilateral visual loss, accompanied by optic hyperaemia and edema of the optic disc along with macular edema and hard exudates which are characteristically arranged in a star shaped pattern around the fovea which is known as macular star<sup>2</sup>. A relatively rare condition, it is often misdiagnosed as papilledema or demyelinating optic neuritis due to the later onset of macular. The role of corticosteroids in neuro-retinitis is controversial in infective aetiology but can be used under cover of antibiotics in severe inflammation and visual loss before serological tests result have been obtained.

### CASE PRESENTATION

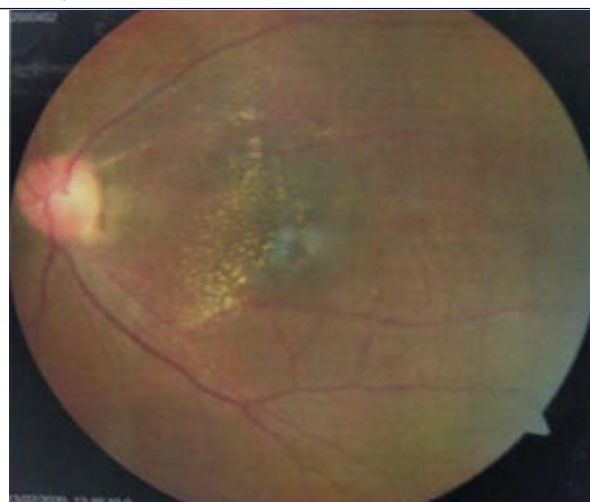
A 39-year-old male presented with painless progressive loss of vision in the left eye for 20 days. He reported painful eye movements at the onset of symptoms which resolved spontaneously. There was no history of systemic illness, ocular redness, discharge, floaters, fever, headache, neck stiffness, malaise, ocular trauma or surgery.

He had a history of smoking for 20 years. He was a non-vegetarian. There was no preauricular or submandibular lymphadenopathy. Dermatological, neurological and general medical examinations were normal.

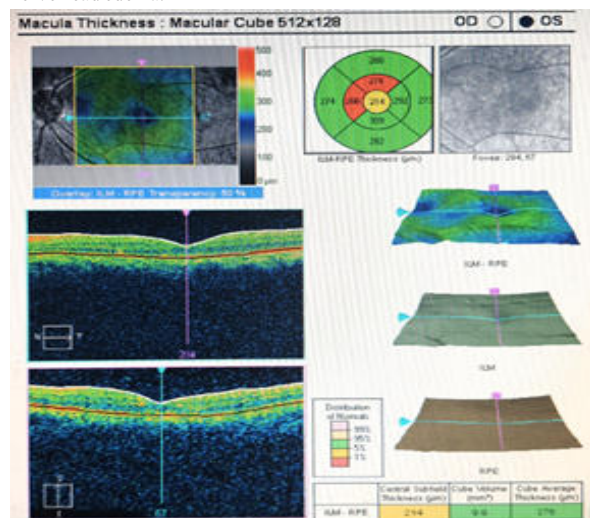
On dental examination, he was found to have gingivitis. On ocular examination, right eye was found to be normal. In the left eye Grade I RAPD was present. On Slit lamp examination showed a quiet anterior segment with no cells or flare.

IOP measured by Goldman Applanation Tonometry was 18 mm Hg in right eye and 16 mm Hg in left eye. Best corrected visual acuity was 6/6 N6 in the right eye and 6/60, < N36 in the left eye. Colour vision was 16/16 and 3/16 with Ishihara colour plates.

Fundus examination of the left eye showed disc edema, macular edema and macular hard exudates arranged in a star shaped configuration [Fig.1].



**Fig.1** – Fundus photo showing hard exudates around macula with optic nerve head edema.



**Fig.2** – OCT macula during the initial presentation.

Optical Coherence Tomography (OCT) showed vitreous cells and mild thinning in the superior and temporal parafoveal quadrants in the left eye [Fig.2]. The hyperreflectivity was indication of hard exudates in the retina and vitreous cells over the area of inflammation. These infiltrates in the vitreous may indicate the involvement of the macula.

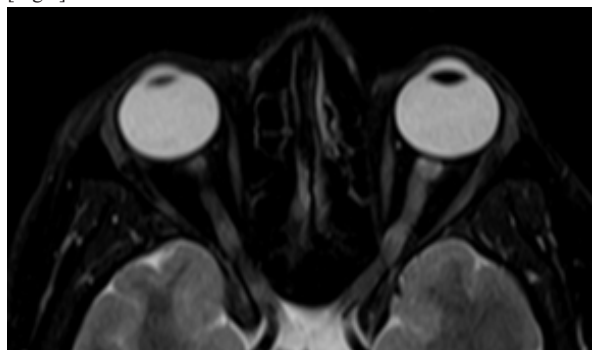
Full blood counts, chest X ray, CSF profile and ANA profile, c ANCA, p ANCA and TB panel were normal. He was referred to the dental outpatient department (OPD) to rule out focal sepsis. A diagnosis of chronic gingivitis was made. A gingival swab for culture and sensitivity showed the growth of *Enterococcus faecium*. [Fig.3.]

Blood cultures also showed the same organism.

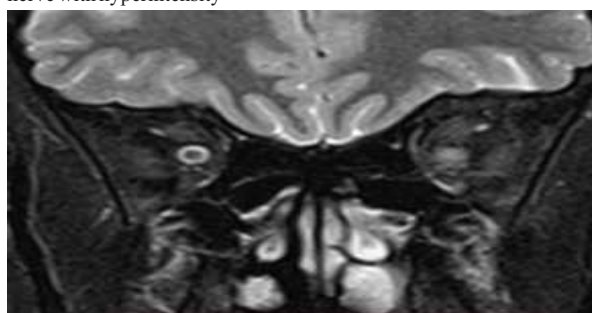


**Fig.3**– Culture plate showing growth of *Enterococcus faecium*

The patient was started on antibiotics, Tab. Ciprofloxacin 500mg twice daily for 5 days and Tab. Amoxicillin clavulanate 625 mg twice daily for 7 days based on his culture and sensitivity reports. Along with antibiotics, a tapering course corticosteroid for 24 days was given. The initial dose was Tab. Prednisolone 60 mg once daily for 4 days. This was followed by reduction and tapering of Tab. Prednisolone by 10 mg each reduction up to 4 days. MRI brain was done to rule out demyelinating disease. It showed a post contrast enhancement of optic nerve on T1 images and a mildly thickened, tortuous optic nerve on T2 images [Fig.4], loss of cerebrospinal fluid intensity around the optic nerve on T2 fat saturation coronal section [Fig.5] and mild diffusion restriction.



**Fig.4 – T2 fat saturation showing mildly thickened tortuous left optic nerve with hyperintensity**



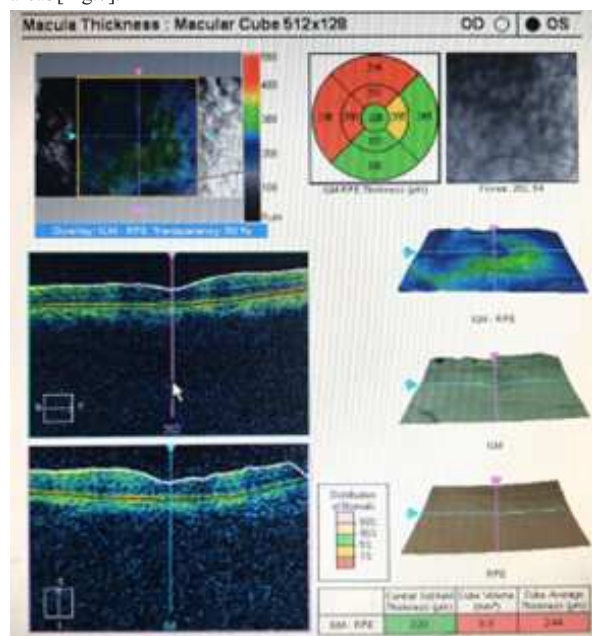
**Fig.5** – T2 fat saturation coronal section shows loss of cerebrospinal fluid intensity around the left optic nerve

On follow up after 3 weeks his BCVA in the left eye had improved to 6/9, N8. Dental examination showed complete resolution of the gingivitis. However, he had developed macular fibrosis [Fig.6].



**Fig.6**–Fundus photo showing fibrous scar in the macula on follow-up

A review after 6 months showed a final visual acuity at 6/9, N8. OCT Macula during the follow-up shows decreased thickness in the superior parafoveal, perifoveal and temporal perifoveal and parafoveal areas [Fig. 7].



**Fig.7**–OCT macula of the patient on follow-up

**DISCUSSION:**

The most common cause of neuro-retinitis is cat scratch disease. We considered tubercular retinitis with contiguous inflammation. An aetiology of TB is highly likely in an endemic area such as India. Our patient however did not have clinical or serological results to support these infections. In the early stages, based on clinical examination, neuro-retinitis can sometimes be confused with other types of disc oedema such as papilledema or anterior ischemic optic neuropathy. Presence of papilledema with macular involvement can resemble hypertensive retinopathy. A typical leakage of fluorescein and staining of optic disc with fundus fluorescein angiography and thickening of neurosensory retina in OCT will confirm hypertensive retina. Focal chorioretinitis, vasculitis, posterior scleritis can sometimes mimic neuroretinitis but characteristic ocular findings in each of the conditions and a relevant history would help to differentiate between the conditions. OCT would help to ascertain causes of visual loss, in follow up and to assess the response to treatment.

The typical arrangement of hard exudates in a macular star pattern is due to the anatomical, radial orientation of Henle's fibres in the outer plexiform layer<sup>1</sup>. Sudden and painless blurring of vision in neuroretinitis has been reported as the first sign of Lyme disease. Neuroretinitis can be the presenting and sometimes the only sign of Lyme disease<sup>2,3</sup>. Chikungunya fever was followed by neuroretinitis like features in a patient who presented with sudden painless diminution of vision in both eyes during his recovery phase<sup>4</sup>. Pan uveitis with neuroretinitis, frosted branch angiitis and paracentral acute middle maculopathy has been associated with syphilis<sup>5, 6</sup>. Neuroretinitis due to infectious causes remains a rare entity with its own unique diagnostic challenges. Hereby we report a rare case of neuro-retinitis,

likely following dental sepsis due to *Enterococcus faecium* which was treated successfully with a combination of antibiotics and corticosteroids. We strongly believe and suspect that neuro-retinitis secondary to dental sepsis, due to the simultaneous resolution of both infections and prompt response to treatment. We also would like to highlight the importance of oral examination in patients presenting with neuro-retinitis and inflammation along with an ophthalmic examination.

### CONCLUSIONS

Neuro-retinitis can occur following dental sepsis. Oral examination forms a crucial part of initial patient evaluation in patients who present with neuro-retinitis and ocular inflammation. Severe visual complications can be avoided with oral corticosteroids under cover of dual antibiotics.

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