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Case Report

A Rare Case of Neuroretinitis with Neurosensory Detachment

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Case Report

A 25 year old man, smoker, non -diabetic, non-hypertensive, a driver by occupation presented with two days history of sudden diminution of vision in the left eye associated with mild pain on and off on the same side.

On examination the visual acuity was 6/6 in the right eye and finger counting at half meter in left eye at presentation. He had RAPD and defective color vision in left eye. Fundus examination showed disc edema on left side without hemorrhages and a patch of pale retina along the distribution of cilioretinal artery.

The physical examination was normal with pulse 72 bpm, was afebrile and BP recorded was 110/70 mmHg.

All the baseline investigations were done on day 1 of admission and the results were:

Table – 1	
Hb	14.6g/dl
TLC	8700/µL
Blood Sugar(F)	93mg/ dl
DLC	$L_{40}N_{45}M_{14}$
Blood Urea	27 mg/Dl
Creatinine	0.96mg/Dl
Na^+	129
K+	3.90
PLT Count	$233 \times 10^{3/}$ µL
ESR	25mm
PBF	Normocytic Normochromic picture with mild increase in eosinophils.
Urine Routine Examination	Normal
TORCH	Negative
Triple Serology	Negative
ANA/ANCA	Negative
Carotid Doppler	Normal Study

On Day 2 of admission CEMRI was done which showed mildly bulky left optic nerve (5-6mm in dia) and showed hyperintense signal on T2/FLAIR images suggestive of L) Optic neuritis.

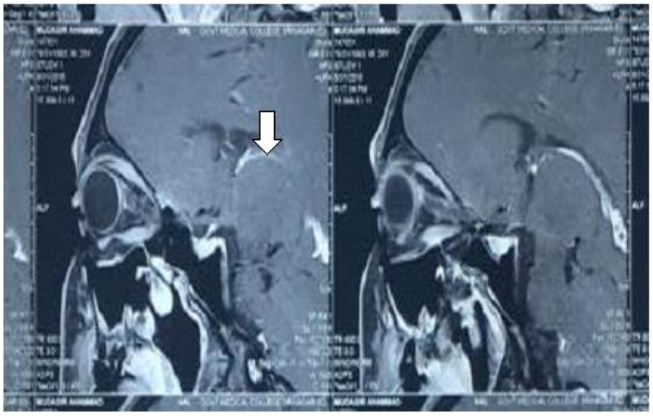


Figure 1

On Day 3 of admission OCT was done which on the right side was normal and on left side showed neurosensory detachment with height of 488µm.

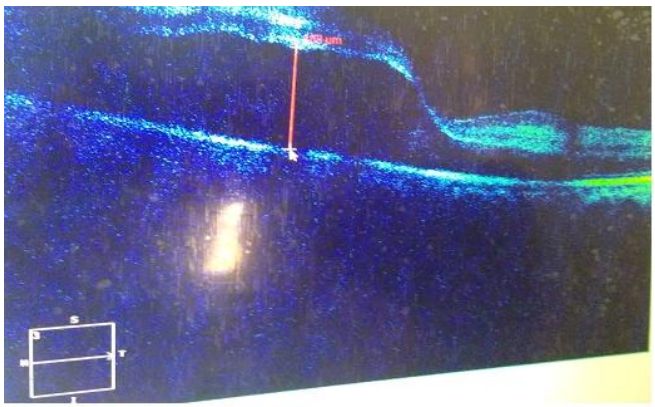


Figure 2

Visual Field Testing demonstrated the altitudinal field defect in the left eye.

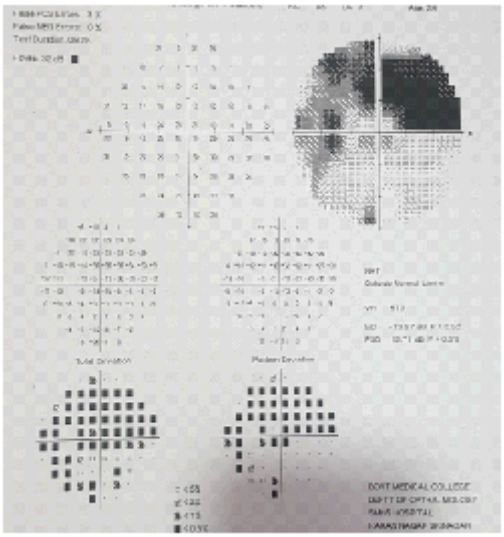


Figure 3

Fundus Fluorescein Angiography (FFA) done on the same day showed leakage from the disc which gradually increased in size and intensity towards late phase .Arterial attenuation giving beaded appearance was also noted along the area of neurosensory detachment with no filling of the peripheral vessel.

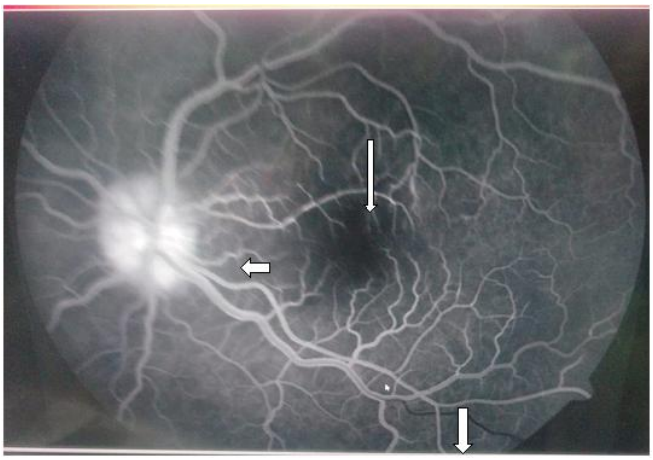


Figure 4

VEP done on 5th day of admission revealed mild delayed P100 on left side (~borderline delay)

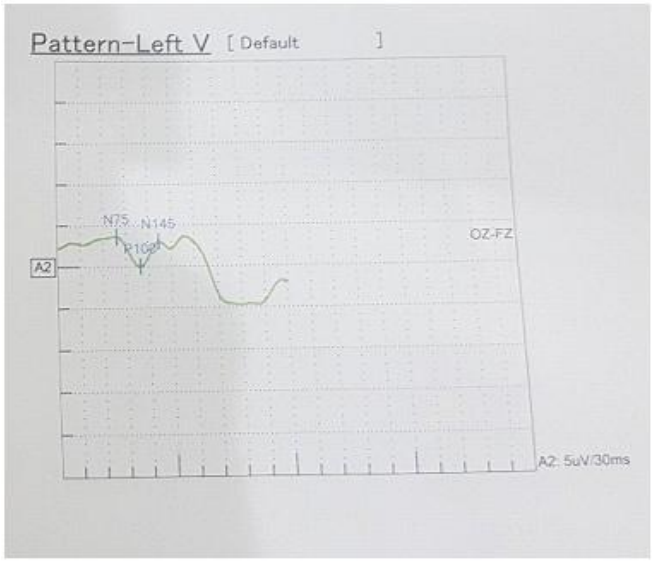


Figure 5

Injection Methyl Prednisolone 1gm i.v OD was started on day 3 of admission and the patient received 3 doses of the same for 3 days.

On day 4 of the dose patient showed significant improvement in VA upto 6/12 which initially was only counting finger.

Repeat OCT was done after 1 week of receiving the steroids and it showed significant improvement in the neurosensory detachment.

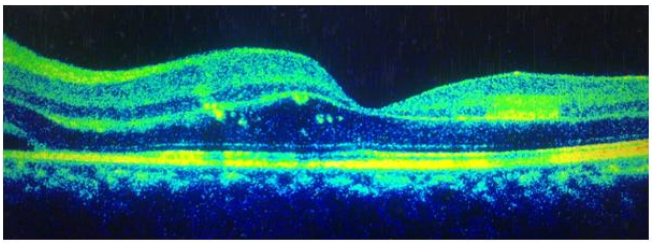
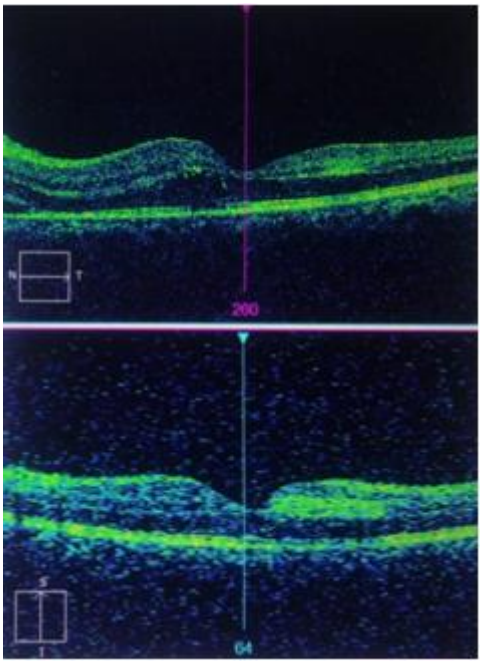


Figure 6



The patients vision had improved to 6/9 and the RAPD grading had improved. The disc edema started settling and the patient developed macular star after 2 weeks of admission.

Neuroretinitis represents an inflammatory optic neuropathy characterized by increased permeability of deep optic nerve blood vessels, leading to macular edema and lipid exudates in a star pattern. The disease usually runs a 6- to 12-week course, typically with good visual recovery.[1] Peripapillary neurosensory retinal detachments are believed to result from fluid that escapes from deep optic disc vessels and seeps directly into the subretinal space[2]. The OCT findings in our case are consonant with subfoveal detachments. The OCT electronic calipers measured a 488µ diameter neurosensory detachment. The OCT showed extensive fluid within the outer plexiform layer but no evidence of communication between the neurosensory detachment and the optic disc margin. These findings resemble those of Puliafito[3], who noted substantial cystic outer plexiform layer edema, and a neurosensory detachment that did not communicate with the disc in a patient with an optic pit. Similar exudative neurosensory retinal detachments secondary to macular edema can occur in retinal vascular diseases, suggesting that limited subfoveal fluid accumulation may occur more commonly than previously believed[4].

Because the fluorescein angiogram showed no evidence of retinal vascular or retinal pigment epithelial leakage, the exact mechanism of the neurosensory detachments remains uncertain in our case. Subfoveal neurosensory detachment secondary to macular edema can occur because of protein and fluid movement across the outer limiting membrane[5]. We postulate that retinal edema in neuroretinitis occurs primarily by fluid movement from the optic disc into the outer plexiform layer, with secondary transit through the outer limiting membrane, resulting in neurosensory subfoveal detachments.

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