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Research Article

CARBOPLATIN INDUCED ISCHEMIC OPTIC NEUROPATHY

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Abstract:

Background: Cisplatin and carboplatin are the two platinum based alkylating agents used widely as anti-cancer therapy. Carboplatin is more chemically stable and better tolerated with comparatively favourable profile. Reports of Carboplatin precipitating optic neuropathy is rarely reported.

Case presentation: A 54-year-old female underwent combination chemotherapy (Carboplatin and Pemetrexed) for non-small cell lung cancer.1 month after completion 6 cycles of chemotherapy, she reported diminision of vision in both eyes with BCVA of 6/24 and 6/12(P). Indirect Ophthalmoscopy showed blurring of disc margin with peripapillary hemorrhages &parafoveal exudates arranged in radial manner.HFA shows Arcuate field defect. Patient was started on Oral Steroids. Patient's visual acuity returns to normal while field defects continue to worsen.

Conclusions: Early Diagnosis and promt treatment of Carboplatin induced optic neuropathy can prevent irreversible visual loss. Carboplatin therapy requires continuous monitoring and detailed ophthalmologic review for any potential ocular side effects.

Keywords: Carboplatin, Optic Neuropathy, Field defects, Anticancer agents.

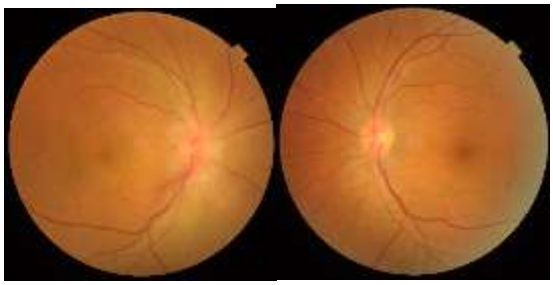
INTRODUCTION

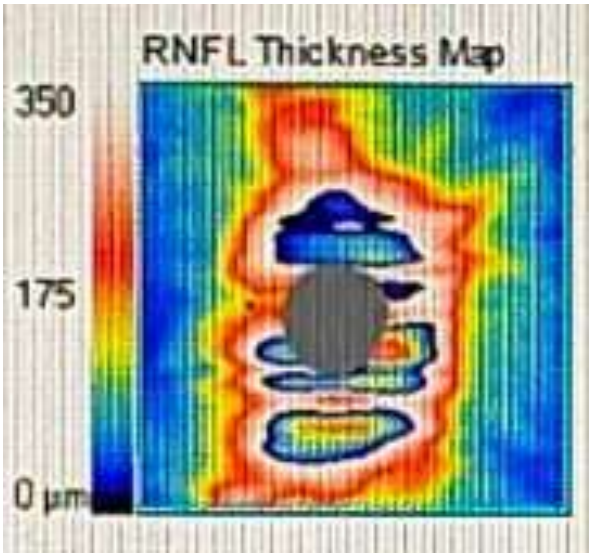
Carboplatin, a platinum containing alkylating agentis used as chemotherapeutic for vastforms of malignancies includingovarian cancer and metastatic non-small cell lung cancer. It binds to and cross-links DNA, interfering with the replication and suppressing growth of the cancer cell. It is considered to be better tolerated than cisplatin, the other similar category chemotherapeutic.Cisplatin have various documented reports for ocular side effects for which ischemic optic neuropathy is thought to be the underlying cause[1,2].While not much data is present for carboplatin.The pathogenesis remains undefined, necessitating further investigations into and reporting of this rare adverse effect. Here we present a caseofCarboplatin induced Bilateral optic neuropathy in a patient with Non squamous cell Lung cancer.

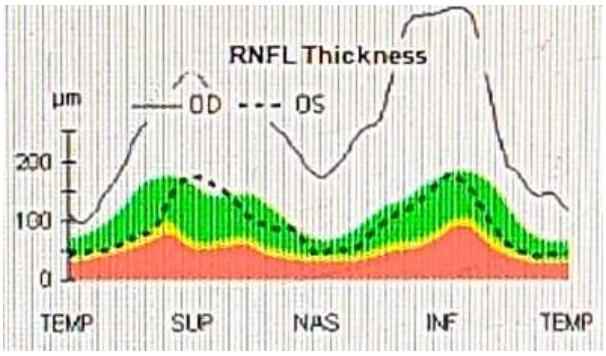
CASE PRESENTATION

A 54 yr old female with Non small cell lung cancer completed treatment with 6 cycles of combination therapy including Carboplatin and Pemetrexed came to eye OPD with complaint of blurring of vision in BE after 1 month of completing last cycle of chemotherapy.

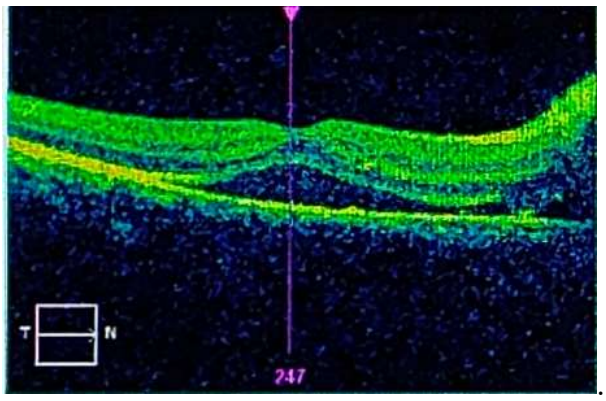
On examination, patient BCVA was 6/24 in RE & 6/12(P) in LE. Color vision was reduced to reading 3 plates only in RE, while LE have normal color vision. Anterior Segment examination was within normal limits with no restriction in Extra-ocular movements. Indirect Ophthalmoscopy of RE shows hyperemicoptic disc with blurred margins(Gd III disc edema) and peripapillary haemorrhages with parafoveal exudates arranged in radial manner. Indirect Ophthalmoscopy of LE was normal. OCT shows increased RNFL thickness with Sub retinal Fluid at fovea extending upto disc margin in RE while LE scan was normal. Visual Field analysis shows Inferior altitudinal field defect with enlargement of blind spot in RE. Neurological evaluation was unremarkable with CE-MRI showing no compressive pathology.



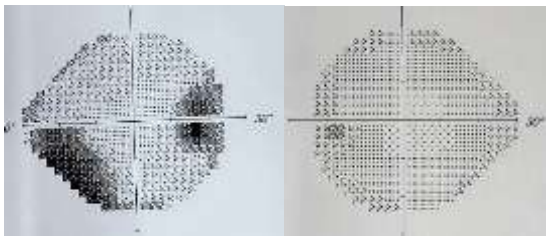


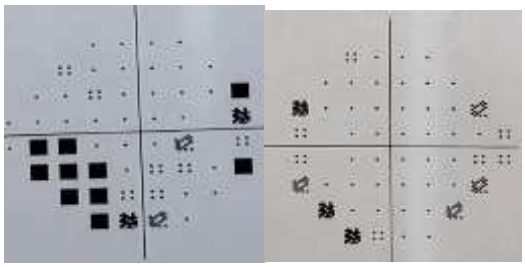


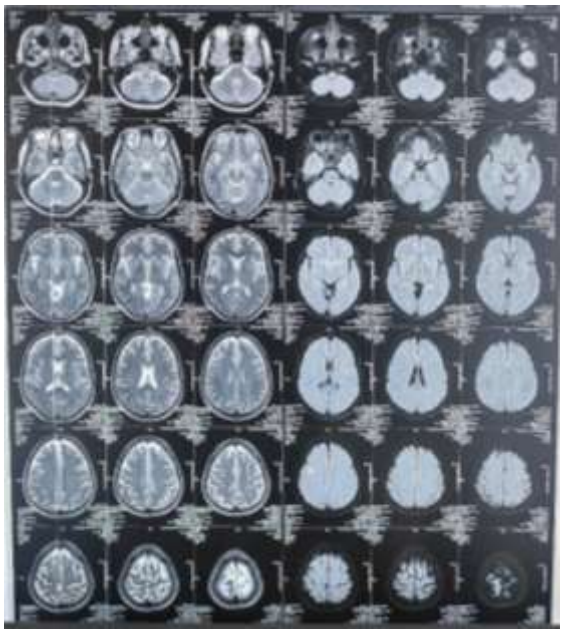
	OD	OS
Average RNFL Thickness	270 µm	96 µm
RNFL Symmetry	69%	
Rim Area	2.03 mm²	1.56 mm²
Disc Area	1.88 mm²	1.67 mm²
Average C/D Ratio	0.06	0.25
Vertical C/D Ratio	0.06	0.17
Cup Volume	0.000 mm³	0.000 mm³



	Central Subfield Thickness (µm)	Cube Volume (mm³)	Cube Average Thickness (µm)
ILM - RPE	442	12.5	346

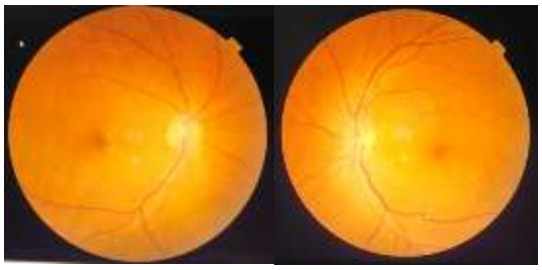




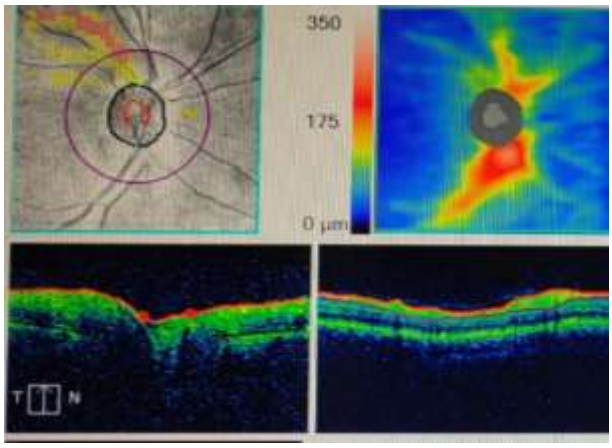


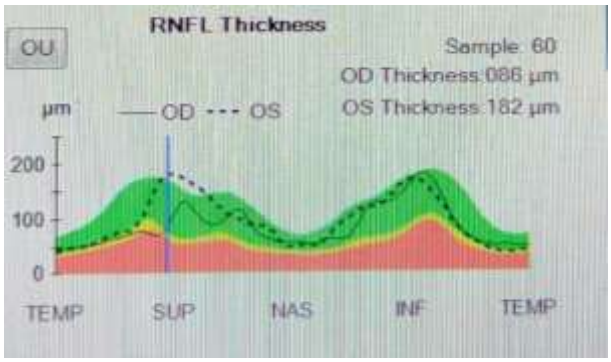
Patient was diagnosed to be a case of Carboplatin induced Ischemic Optic Neuropathy. Chemotherapy was stopped immediately. Patient was started on Tab Prednisolone 1mg/kg tapered over 1 week.

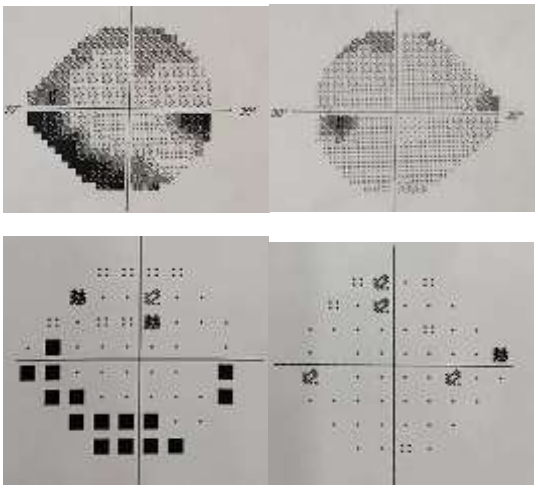
Review after 1 month shows improvement with visual acuity back to normal (6/9)and resolution of disc edema. However there was worsening of visual field changes on subsequent follow-ups in BE.



⚠	OD	OS
Average RNFL Thickness	85 µm	95 µm
RNFL Symmetry	81%	
Rim Area	1.40 mm²	1.61 mm²
Disc Area	1.70 mm²	1.71 mm²
Average C/D Ratio	0.42	0.25
Vertical C/D Ratio	0.43	0.15
Cup Volume	0.003 mm³	0.000 mm³







DISCUSSION

Carboplatin induced ischemic optic neuropathy is not well known in the past. The studies includes a few cases of optic neuropathy secondary to carboplatin.In 2009, Fischer et al managed a case with carboplatin-induced optic neuropathy with bilateral disc edema causing incomplete recovery ofvision[3]. In 2014, Lewis et aldescribed blurring of vision in the LE of a 48-year-old female five days after completion of the fifth cycle of adjuvant carboplatin chemotherapy[4]. Elhusseiny and Smiddy reported a patient with diminision of vision after combination therapy with Carboplatinand/or Paclitexel[5]. Gonzalez et al.reported a patient with metastatic lung cancer who developed sudden visual loss in their left eye after a combi-

nation treatment of carboplatin and cisplatin[6].Kanat et al. demonstrates that carboplatin has a much higher threshold for causing neurotoxicity than other platinum[7].

The cause of retinal ischemia induced by cisplatin/carboplatin is uncertain, but studies revealed various mechanismassociated with it to be the primary cause. Oxidative stress induced retinal toxicity is one of them[8]. Some studies have identified microvascular thrombosis as the cause of visual loss in patients receiving chemotherapy with cisplatin and carmustine[9]. Another hypothesis suggests that decreased renal excretion may lead to drug accumulation in the central nervous system[10].

Our case presenting with mild diminision of vision with significant fundal changes and visual field defects leads to early detection. Prompt intervention with discontinuation of carboplatin and starting systemic steroids lead to complete restoration of vision in both eyes.

CONCLUSION

Carboplatin is a generally well-tolerated platinum analogue where complications like precipitating optic neuropathy or other adverse ocular manifestations are rarely reported. Patient have varied presentation.Need for the close monitoring of patients with prompt treatment as in our case during their course of treatment with intravenous carboplatin therapy to discover pathology during the early stages of treatment and determine changes in visual acuity, visual field, and fundoscopy. Any subtle changes or vision complaint may rationalize the discontinuation of carboplatin in addition to starting systemic corticosteroids. These recommendations, however, need to be discussed with patients and the oncology team to ensure compatibility with the requirements of the case.

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Conflict of Interest: None

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