Author reply to comments

The term papilledema should be exclusively used when disc edema is secondary to raised intracranial tension. In the present case, raised intracranial pressure was ruled out by normal cerebrospinal fluid opening pressure on lumbar puncture. We feel bilateral disc edema will be the correct term to use here instead of papilledema. Toxic optic neuropathy secondary to oxaliplatin seems to the most plausible cause of bilateral optic nerve damage in this case. However, other causes, including ischemic optic neuropathy, need to be ruled out.

Reply by authors: The authors agree the term "Bilateral disc edema" can be used as papilledema by the definition should have associated intracranial space-occupying lesion. The term papilledema was used as the disease entity was initially suspected to be due to metastatic lesion, resulting in high intracranial pressure. Toxic and ischemic optic neuropathy can be the plausible etiology, but conclusive evidence is not available. Moreover, toxic optic neuropathy typically will have temporal pallor of disc and ischemic optic neuropathy presenting bilaterally is not very common.

The authors have mentioned that their patient is a 72-year-old male with stage III rectal carcinoma. In abstract, age is mentioned as 70 years. The authors have not mentioned if the patient suffered from any other comorbid conditions. Other than the investigations done, information about few other tests such as complete blood counts, lipid profile, C-reactive protein, blood sugar, and serum homocysteine should also have been provided.

Reply by authors: The age of the patient is 72 years and was in follow-up for 3 years. The patient has no known systemic condition associated and the investigations mentioned were normal. Serum homocysteine was not performed for the patient.[1]

The authors have mentioned that 2 months following initiation of chemotherapy the patient complained of giddiness and reduced visual acuity. His renal function was also deranged. It will be informative to know if the blood pressure of the patient was normal at this stage. A sudden rise in blood pressure can lead to optic neuropathy and can present with bilateral disc edema. The fields performed in this case showed centrocecal scotoma. Centrocecal scotoma has been reported in patients with anterior ischemic optic neuropathy.[2] Turner and Harrison[3] have reported bilateral optic disc edema and optic neuropathy in a 57-year-old patient of colorectal cancer being treated with Oxaliplatin, 5-Flurouracil, and leucovorin. The authors postulated that 5-flurouracil-induced arterial vasospasm might have resulted in bilateral disc edema and field defects in their case. In the present case too, the patient was treated with similar medications. The authors should try to look into any causative role of 5-flurouracil in their case.

Reply by authors: Blood pressure of the patient was normal throughout the follow-up visits for the patient. The authors do agree Grade IV hypertensive retinopathy can present with bilateral disc edema but less likely in the present case. Ischemic optic neuropathy characteristically cause altitudinal fields defect; however, optic disc pathologies can cause centrocecal scotoma in various conditions.

Platinum derivatives have reported to cause major neurological effects than 5-flurouracil like nerve palsy and disc edema. The study mentioned also only postulated the possible mechanism as arterial vasospasm. Arterial vasospasm more likely to cause a pallid disc edema, but in our case, it was hyperemic disc edema.

Finally, the authors have mentioned that oxaliplatin was stopped and the patient was treated on oral prednisolone in a tapering dose over 6 months. It was only 2 months when visual symptoms started and oxaliplatin was stopped. It will be informative to know how the systemic disease fared and if there was any recurrence or metastasis of the carcinoma following cessation of oxaliplatin.

Reply by authors: The patient in the follow-up with regional cancer center and disease process being closely monitored and no worsening or recurrence noted so far.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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