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Case Report

Bilateral Chorioretinitis and Neurosensory Retinal Detachment after Nivolumab Therapy in a Young Patient with Hodgkin Lymphoma

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Abstract: We present the case of an 18-year-old male with recurrent Hodgkin's lymphoma who developed bilateral intraocular inflammation 47 days after initiating nivolumab. Examination revealed bilateral chorioretinitis and papillitis with subretinal fluid in the left eye. Nivolumab was discontinued, and combined oral and topical corticosteroid therapy was initiated, leading to the resolution of symptoms 37 days after presentation. Nivolumab-induced ocular inflammation is a rare but significant immune-related adverse event in patients receiving immune checkpoint inhibitor therapy. To our knowledge, this is the youngest reported case of posterior uveitis associated with nivolumab and the youngest patient with Hodgkin's lymphoma to develop this complication following immunotherapy. Although previously documented in older populations, its occurrence in an adolescent highlights the need for routine ophthalmic evaluations in all individuals undergoing this treatment. These findings suggest that early recognition, prompt cessation of the offending agent, and timely management with both oral and topical corticosteroids can prevent long-term visual impairment. With the expanding use of immune checkpoint inhibitors across all populations, proactive ophthalmic monitoring will be essential for preserving long-term ocular health.

Keywords: immune checkpoint inhibitor; intraocular inflammation; subretinal fluid; Hodgkin's lymphoma; corticosteroids

1. Introduction

Immune checkpoint inhibitors (ICIs) are highly effective against multiple types of cancer including Hodgkin lymphoma (HL) [1]. Nivolumab, an immunoglobulin G4 (IgG4) monoclonal antibody that targets programmed cell death protein 1 (PD-1) receptor, is an ICI that induces anti-tumor effects through disruption of inhibitory interactions between antigen-presenting cells and T lymphocytes [2]. ICIs as a class are typically well-tolerated, but patients can experience immune-related adverse events (irAEs) like skin lesions, autoimmune colitis, autoimmune hepatitis, and endocrinopathies [2]. Ocular complications, including uveitis, occur in less than 1% of patients [2]. This report summarizes the case of an 18-year-old male who developed bilateral intraocular inflammation following treatment with nivolumab.

2. Case Report

An 18-year-old Caucasian male presented to the ophthalmology clinic at a tertiary academic medical center with acute-onset, bilateral scotomas and floaters. Six days prior, he received cycle three of four of brentuximab/nivolumab for relapsed Hodgkin lymphoma (HL) and was subsequently admitted for desensitization under close observation. Brentuximab and nivolumab were administered concurrently as a combination regimen. The patient's only other medication at the time of symptom onset was ondansetron as needed for nausea, which is not associated with intraocular inflammation. While admitted, the patient reported no ocular symptoms, and an initial eye exam revealed no abnormalities. Ocular history was significant for mild hyperopia and astigmatism treated with contact lenses. The remainder of the patient's medical, family and social history was unremarkable.

On examination after visual symptoms had developed, the patient's visual acuity was 20/20-1 and 20/25-1 in the right and left eye, respectively, with intraocular pressure (IOP) of 10 mm Hg, full confrontational visual fields (CVF) (counting fingers), full extraocular movements (EOM) and no afferent pupillary defect. Slit lamp

examination revealed 4+ anterior chamber (AC) cell and 2–3+ vitreous chamber (VC) cell in the right eye and 3+ AC cell and 2–3+ VC cell in the left eye (graded per Standardization of Uveitis Nomenclature [SUN] criteria). Fundus examination revealed poorly demarcated yellow lesions throughout the posterior pole and mid-periphery of the left eye (Figure 1A), which were not present in the right eye (Figure 1B). Optical coherence tomography (OCT) revealed subretinal fluid in the temporal macula in the left eye (Figure 1C) and normal retinal architecture in the right eye (Figure 1D). There was mild optic disc edema present in both eyes, with an average retinal nerve fiber layer (RNFL) thickness of 135 μm and 132 μm in the right and left eye, respectively. There was no evidence of intraretinal fluid in either eye.

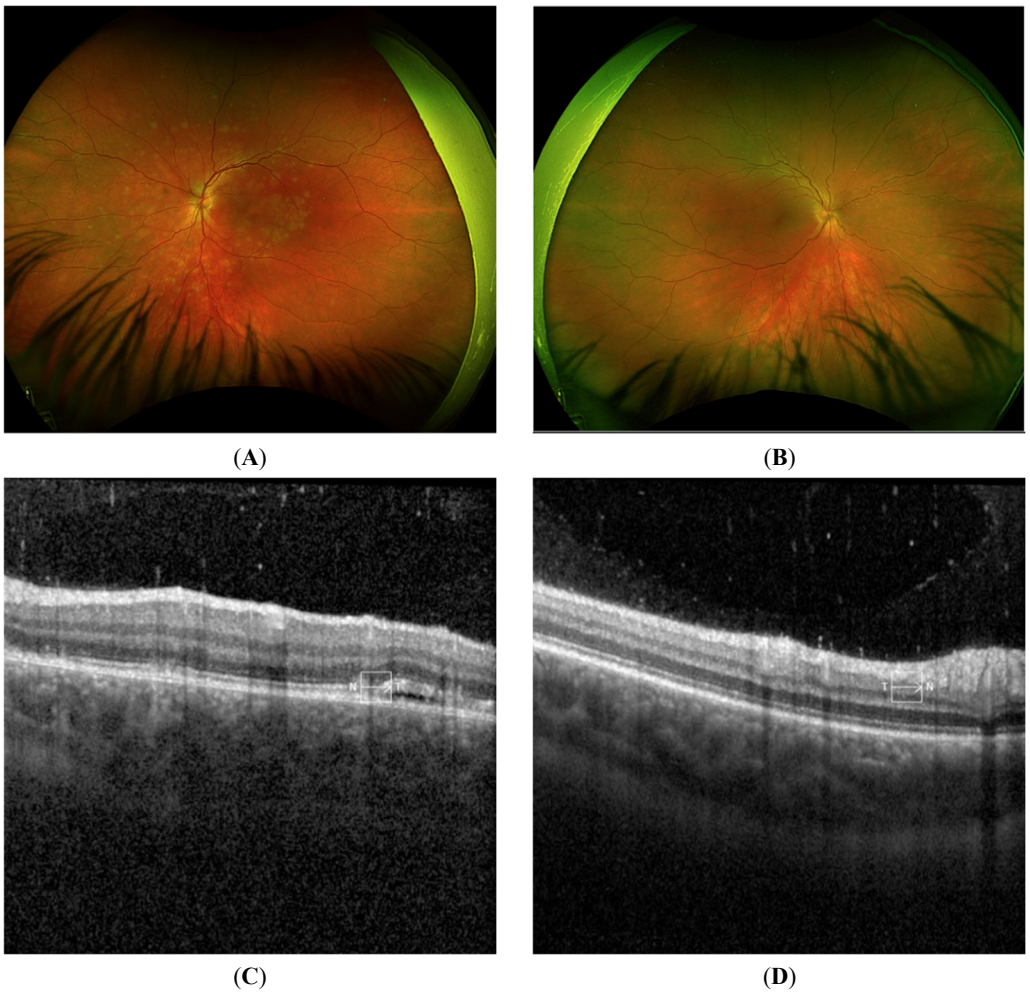

Figure 1. Widefield fundus photo of the left eye (**A**) at presentation, most notable for poorly demarcated hypopigmented retinal lesions throughout the posterior pole and mid-periphery; (**B**) right eye showing no significant fundus abnormalities, though anterior chamber and vitreous inflammation were present bilaterally on slit lamp examination; macular optical coherence tomography of the left eye (**C**), showing vitreous hyperreflective material, subfoveal and parafoveal retinal pigment epithelium changes and ellipsoid zone disruption, and extrafoveal subretinal fluid in the temporal macula; (**D**) right eye showing similar but less severe findings.

Serum polymerase chain reaction (PCR) testing for cytomegalovirus, toxoplasmosis, and herpes simplex virus were negative. Beta-d-glucan, rapid plasma reagin with reflex titer, QuantiFERON-Tb gold and human immunodeficiency virus (HIV) serology tests were also negative.

The patient was diagnosed with bilateral chorioretinitis and papillitis, along with a neurosensory retinal detachment in the left eye. Nivolumab infusions were suspended, and the patient was placed on 60 mg oral prednisone once daily and topical prednisolone acetate 1% four times daily in both eyes.

Three weeks later, the patient was examined again. Visual acuity, IOP, pupil exam, CVF and EOM were stable. Slit lamp examination revealed quiet anterior chambers, rare cells in the anterior vitreous of the right eye, and trace cells in the anterior vitreous of the left eye. Fundus examination revealed improved appearance of the peripheral chorioretinal lesions in the left eye (Figure 2A), and no new lesions in the right eye (Figure 2B). OCT revealed decreased RNFL thickening in both eyes and resolved subretinal fluid with trace irregularities in the outer

retina of the left eye (Figure 2C), with continued improvement in the right eye (Figure 2D). The patient continued tapering doses of oral and topical corticosteroids. At follow up 37 days after initial presentation, the patient's intraocular inflammation, papillitis, and chorioretinitis had resolved. Following permanent discontinuation of ICI therapy, the patient transitioned to consolidative external beam radiation therapy, with successful disease control and stable lymphoma maintained at over one year of follow-up.

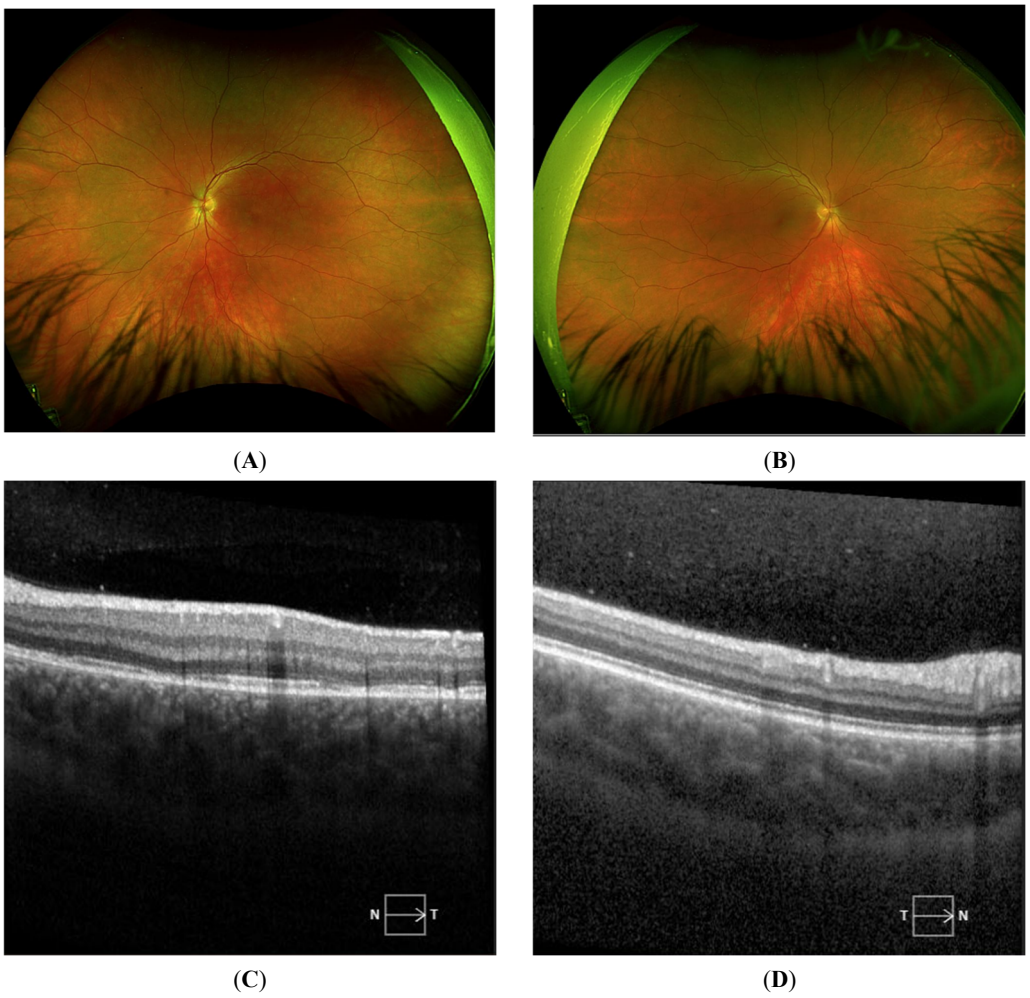

Figure 2. Widefield fundus photo of the left eye (**A**) and right eye (**B**), and macular optical coherence tomography of the left eye (**C**) and right eye (**D**), obtained three weeks after initiating treatment with systemic and topical corticosteroids, showing marked clinical improvement in findings bilaterally compared to Figure 1.

3. Discussion

ICIs suppress T-cell inhibition, impairing immune homeostasis and enhancing inflammatory responses [3]. Adverse events, such as ICI-related uveitis, typically arise within weeks to months of starting therapy and can affect various ocular structures, including the cornea, uvea and retina [2]. Management of ICI-induced uveitis depends on symptom severity: mild cases are often treated with topical corticosteroids, while severe cases may require systemic corticosteroids and cessation of the offending agent [4].

There are several reports of uveitis and serous retinal detachment following nivolumab therapy. Sun et al. described bilateral panuveitis in a 47-year-old male with malignant melanoma (MM), presenting 63 days after initiating therapy, and bilateral anterior uveitis in a 37-year-old female with HL, presenting 380 days after initiating therapy. In both instances, nivolumab was discontinued, and patients received oral and topical corticosteroids, with return to visual baseline in two months [5]. Obata et al. reported bilateral panuveitis in a 63-year-old female with metastatic MM, presenting 10 days after receiving the second administration of nivolumab. Nivolumab was discontinued, and the patient was treated with topical steroids and cycloplegics, with return to visual baseline in four months [6]. Yilmaz et al. described recurrent serous retinal detachment and anterior uveitis in a 60-year-old male with metastatic MM, presenting several days after receiving the third infusion of medication. Treatment involved oral and topical corticosteroids, with resolution of subretinal fluid and return to visual baseline after two

weeks. Despite a recurrence of anterior uveitis two months later, which also responded to treatment, nivolumab was continued throughout [7]. Miyamoto et al. reported bilateral serous retinal detachments in a 73-year-old male with metastatic MM, presenting two months after initiating nivolumab therapy. No ocular interventions were performed, and nivolumab was continued; the patient's vision remained stable until he succumbed to his progressive systemic disease [8].

Our patient, an 18-year-old male, developed bilateral chorioretinitis and papillitis with neurosensory retinal detachment in the left eye 47 days after initiating nivolumab for recurrent HL. This case is notably unique as it represents, to our knowledge following a review of the available literature, the youngest reported case of nivolumab-induced posterior uveitis, occurring in a patient with HL rather than the more commonly reported MM, with bilateral inflammation and rapid resolution. While alternative diagnoses including multiple evanescent white dot syndrome (MEWDS) and primary inflammatory choriocapillaropathy (PICCP) were considered given the patient's young age and lesion appearance, the absence of a viral prodrome, bilateral presentation, and clear temporal association with nivolumab support an ICI-induced etiology. Although brentuximab was administered concurrently, the development of intraocular inflammation is more strongly attributed to nivolumab. Uveitis is a well-established immune-related adverse event associated with PD-1 inhibitors. Conversely, safety analyses of brentuximab did not report uveitis, and its FDA labeling does not specifically address ocular toxicities [9]. While rare instances of brentuximab-associated ocular inflammation have been documented in the literature [10–12], the posterior segment inflammation observed here is mechanistically consistent with the T-cell disinhibition induced by PD-1 blockade. This contrasts sharply with the cytotoxic, microtubule-disrupting mechanism of brentuximab. Nevertheless, a synergistic contribution from brentuximab cannot be entirely excluded, and clinicians should remain vigilant when utilizing this combination regimen. Management of our patient mirrored that of other uveitis cases, utilizing oral and topical corticosteroids, but ICI therapy was discontinued in this case; however, such decisions should be made on a case-by-case basis in close collaboration with the treating oncologist and patient, weighing the ophthalmic risks against the oncologic benefits of continued therapy. The rapid resolution of inflammation and subretinal fluid within 37 days, the fastest among referenced cases, suggests that stopping the ICI may have contributed to the relatively rapid recovery observed. This approach remains the mainstay for managing nivolumab-related ocular toxicities and aligns with the proposed mechanism of ICI-induced uveitis, where corticosteroid therapy effectively manages inflammation by counteracting the T-cell-mediated immune response. We acknowledge that fluorescein angiography, indocyanine green angiography, and fundus autofluorescence were not obtained at initial presentation, which represents a limitation of this report and would have better characterized the acute inflammatory lesions.

This report highlights ocular complications associated with ICIs, reinforces the effectiveness of combined oral and topical corticosteroid therapy, and suggests that early discontinuation of the offending drug may contribute to recovery, though continuation of ICI therapy should be considered whenever feasible. The emergence of these complications in a younger patient marks a significant shift from the typical demographic and further emphasizes the necessity for routine ophthalmic evaluations. With expanding use of ICIs in all populations, proactive monitoring and timely intervention will be essential to ensure ocular health.

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Informed Consent Statement: Written informed consent has been obtained from the patient to publish this case report and any accompanying images.

Data Availability Statement: Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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Use of AI and AI-Assisted Technologies: No AI tools were utilized for this paper.

Abbreviations

AC, anterior chamber; CVF, confrontational visual field; EOM, extraocular movements; HIV, human immunodeficiency virus; HL, Hodgkin lymphoma; ICIs, immune checkpoint inhibitors; IgG4, Immunoglobulin G4; IOP, intraocular pressures; irAEs, immune-related adverse events; MM, malignant melanoma; OCT, optical coherence tomography; PCR, polymerase chain reaction; PD-1, programmed cell death protein 1; RNFL, retinal nerve fiber layer; SUN, Standardization of Uveitis Nomenclature; VC, vitreous chamber.

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