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A glaucoma drainage implant functioning as a sanctuary site for vitreoretinal lymphoma

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ABSTRACT

Purpose: To report an unusual case of vitreoretinal lymphoma (VRL) in which a glaucoma drainage implant (GDI) likely functioned as a sanctuary site for relapsing disease.

Observations: A 54-year-old female with recently diagnosed CNS diffuse large B-cell lymphoma (DLBCL) was referred for evaluation of VRL. Ocular history at an outside center included a 4-year reported history of uveitis complicated by glaucoma and a GDI in the left eye (OS). Initial examination revealed keratic precipitates (KP), vitreous haze with clumps of white cells OS, and vitreous biopsy revealed DLBCL OS. Intravitreal methotrexate injections were initiated for primary VRL alongside systemic chemotherapy for CNS involvement with resolution of disease. One year later, the patient returned with 2+ anterior chamber (AC) and vitreous cells OS, and vitreous biopsy again revealed DLBCL OS. External radiation treatment was administered for recurrent VRL in the left eye, followed also by the right eye due to the high risk of fellow eye involvement. Autologous stem cell transplantation was then performed. Five months later, the patient returned with worsening KPs and new vitreous cells OS, and vitreous biopsy again revealed DLBCL OS. Enucleation was performed, and histopathology revealed DLBCL cells lining the GDI fibrous capsule, consistent with the GDI likely having served as a sanctuary site and source for continued local relapse.

Conclusions and Importance: We report a case in which a GDI functioned as a probable sanctuary site for VRL. Sanctuary sites of malignancy should be considered in patients with pre-existing ocular hardware, particularly when recurrent relapses occur despite complete treatment.

1. Introduction

Primary vitreoretinal lymphoma (PVRL) or primary central nervous system lymphoma-ophthalmic variant (PCNSL-O) is a rare, high-grade form and ocular subset of primary central nervous system lymphoma (PCSNL).[1,2] Over 15 % of patients with PCNSL will develop PVRL, while up to 90 % of patients with PVRL will develop CNS disease.[3] Timely diagnosis of PVRL is essential to reduce the risk of CNS progression, which accounts for the high mortality of 9 to 81 % seen in the disease.[3] Due to its rarity however, PVRL is often misdiagnosed, frequently as chronic uveitis, resulting in treatment with corticosteroids with transient improvement and further delay in diagnosis.[1]

The management of PVRL remains challenging due to lack of standardized guidelines for diagnosis and treatment.[4] PVRL is definitively diagnosed when the presence of malignant cells in the eye is confirmed via diagnostic vitrectomy or, less commonly, aqueous or retinal or choroidal biopsy.[3] Management generally includes local therapies (e.g., radiotherapy, intravitreal chemotherapy) and systemic therapies, with CNS involvement strongly influencing therapeutic decisions.[5] High-dose chemotherapy followed by autologous stem cell transplantation (ASCT) has proven benefit in survival and remission rates among patients with PCNSL. Despite this, relatively frequent local and CNS relapses present therapeutic challenges.

Here, we report the clinical course of a patient with PVRL who developed multiple intraocular relapses despite complete treatment. Given the severity of disease, enucleation was eventually performed and revealed a glaucoma drainage device (GDI) had likely been serving as a sanctuary site for relapsing disease.

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2. Case report

A 54-year-old woman was referred to the ocular oncology service for evaluation of VRL. Two months prior, she had been admitted for altered mental status and computed tomography (CT) had revealed a 4.2-cm parietal lesion, which was resected and revealed diffuse large B-cell lymphoma (DLBCL). Lumbar puncture (LP) was negative. She had also reported mild blurry vision in the left eye and was evaluated by an outside retina specialist who noted vitreous haze and large clumps of white cells in the macula of the left eye (Fig. 1). She received a vitreous biopsy which revealed DLBCL (Fig. 3A), and PCR-based next generation sequencing targeting 54 genes associated with myeloid disorders revealed *MYD88* and *ZRSR2* mutations. For her new diagnosis of CNS-DLBCL, high-dose intravenous methotrexate (MTX) and rituximab had been initiated during her hospitalization. Her past medical history was otherwise non-contributory. Ocular history was significant for chronic uveitis diagnosed in Mexico four years prior. She had received two pars plana vitrectomies (PPV) and treatment with topical and oral steroids, MTX, and azathioprine. She had later developed glaucoma in both eyes (OU) and received an Ahmed GDI OS.

On initial evaluation, visual acuity (VA) was 20/25 OD and 20/30 OS. Intraocular pressure (IOP) was 10 mm-Hg OD and 9 mm-Hg OS on latanoprost OS and dorzolamide, brimonidine, and timolol OU. Slit-lamp examination was unremarkable OD but revealed fine white keratic precipitates (KP) and 1+ vitreous haze OS. Posterior chamber intraocular lenses were present and clear in both eyes. A superotemporal GDI OS was noted to be in good position and otherwise unremarkable. Fundus examination showed fibrotic white sub-RPE deposits in the macula OD and large clumps of white cells in the macula and periphery OS. Due to active disease OS, intravitreal MTX injections were initiated in the left eye. A modified protocol was used and included injections once weekly for 4 weeks, once biweekly for 4 weeks, and once monthly for 3 months for a total of 15 injections.[6] After completing 2 cycles of high-dose intravenous MTX and rituximab, followed by oral temozolomide, maintenance oral ibrutinib was also started but discontinued prematurely due to urosepsis. On six-month follow up, no new cells were observed in either eye.

Sixteen months later, the patient returned with new 2+ anterior chamber (AC) cells and clumps of white cells in the periphery of the left eye (Fig. 2). Of note, IOP remained stable OU. B-scan revealed interval increase in vitreous cells. Repeat vitreous biopsy OS returned positive for DLBCL, consistent with recurrence of disease. MRI was stable and LP was negative. Radiotherapy (XRT) (36 Gy in 20 fractions) was initiated for the left eye, followed by prophylactic XRT (36 Gy in 20 fractions) also for the right eye due to her severity of CNS and left eye disease. Four months later, examination appeared clear of disease and patient was started on definitive treatment with ASCT.

Four months later, the patient returned with worsening KP and yellow-white vitreous cells OS. Repeat vitreous biopsy returned positive for DLBCL, consistent with a second recurrence of disease in the left eye. Given the history of multiple intraocular recurrences, belief that the rest of the CNS was disease-free following ASCT, patient preference, and concern for the GDI serving as a sanctuary site, enucleation of the left eye with removal of the implant was performed to eliminate all current disease.

Histopathology revealed DLBCL cells lining the inner layers of the glaucoma reservoir fibrous capsule (Fig. 3B–G). Cells were also present on the posterior surface of the ciliary body and IOL as well as throughout the vitreous, with rare cells also noted on the optic nerve head and at the level of the lamina cribrosa. Notably, lymphoma cells were present in the vitreous, but not in the subretinal space. Overall findings were deemed most consistent with the reservoir having been the source of lymphoma cells continuously trickling into the AC and vitreous.

Following enucleation, the patient was followed very closely. Four months later, however, she developed new activity in the right eye. B-scan confirmed an interval increase of vitreous cells of the right eye (Fig. 4). MRI was stable and she was initiated on intravitreal MTX injections of the right eye as well as systemic immunotherapy with ibrutinib. Since the completion of intravitreal MTX OD, the patient has remained disease-free (Fig. 5) for 13 months without therapy of any kind. She has also remained without CNS relapse and on her last follow-up visit 5 years since her initial evaluation, VA OD was 20/60.

3. Discussion

This report describes a case of a GDI functioning as a probable sanctuary site for relapsing lymphoma. The disease was treated aggressively with both systemic and intraocular therapies yet continued to recur. In VRL, the eye serves as a sanctuary site where lymphoma cells can grow undisturbed, and the efficacy of systemic chemotherapy is limited by intraocular pharmacokinetics. Thus, primary CNS lymphoma with ocular involvement is often supplemented with local therapies. In this case, both intravitreal MTX and XRT and SCT were attempted yet ineffective, we suspect because the GDI functioned as an additional sanctuary site within the eye and harbored lymphoma cells capable of evading local therapies.

The management of VRL in eyes with intraocular hardware is unknown, as the prevalence of VRL is low and insertion of hardware is generally avoided in these cases. As such, other complications from GDIs may better shed light on management and prognosis. For example, GDI-related endophthalmitis is a well-described complication with varying management strategies.[7–11] Treatment typically involves intravitreal

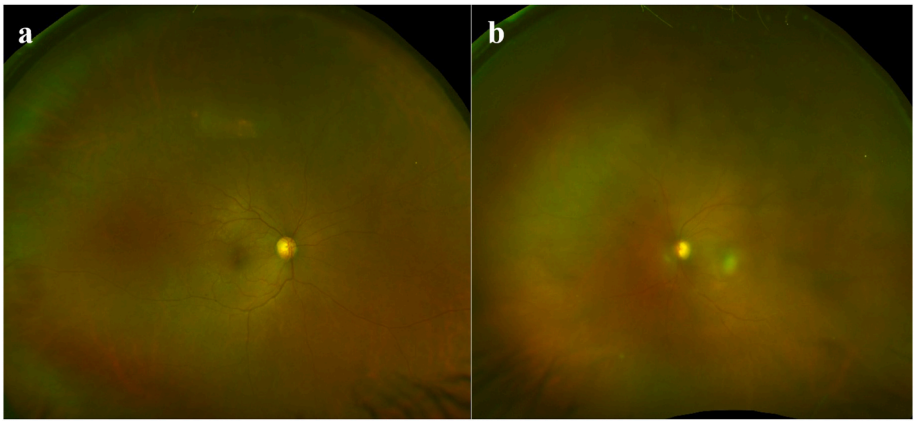

Fig. 1. Color fundus photographs at initial presentation (a) of the right eye showing no active evidence of disease and (b) of the left eye showing vitreous haze and large clumps of white cells in the macula and opacities in the periphery. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

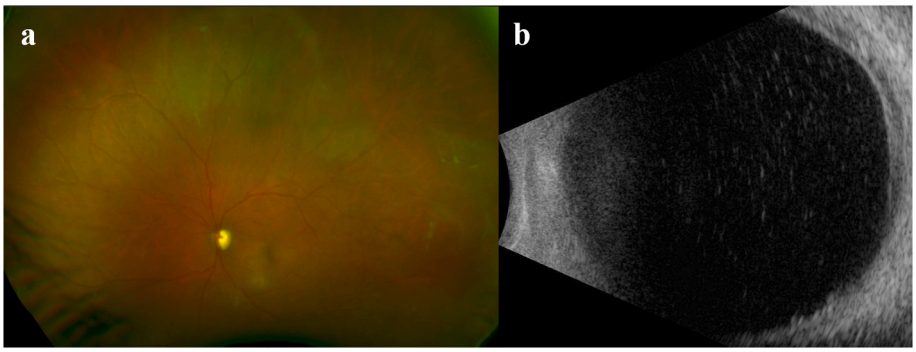

Fig. 2. First recurrence in the left eye as evidenced on (a) color fundus photograph revealing white cells greatest in the temporal periphery (b) B-scan showing an interval increase in vitreous cells. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

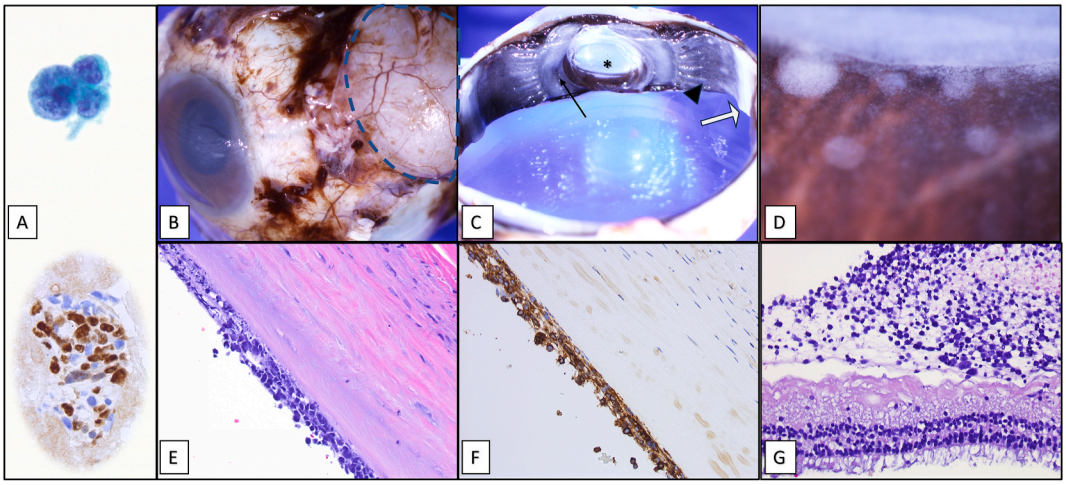

Fig. 3. Pathology of the enucleated eye revealing (A) cytology of large, atypical lymphoma cells in the vitreous (top), and immunohistochemistry (IHC) staining for PAX5 using DAB chromogen confirming the presence of large B cell lymphoma (bottom), (B) macroscopic view of the glaucoma reservoir, surrounded by a fibrosed vascularized capsule (interrupted circle), (C) central section through the globe with view of the posterior iris surface, revealing white infiltrates lining the peripheral retina (white arrow), pars plana and plicata (black arrowhead), and zonules (black arrow); the intraocular lens (asterisk) was clear, (D) magnified view of the peripheral retina at the ora serrata lined by numerous cells and opacities (E) hematoxylin and eosin (H&E) stain demonstrating the fibrosed implant capsule wall lined by multiple layers of large atypical lymphoma cells, (F) IHC staining positive for CD79a and confirming B-lymphocytes, (G) H&E stain of the peripheral retina revealing inner retinal atrophy and a dense epiretinal infiltrate of atypical lymphoma cells.

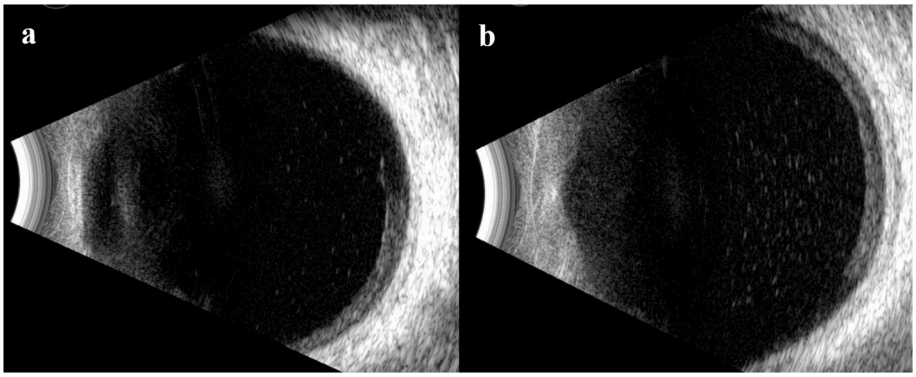

Fig. 4. B-scan of the right eye obtained (a) following autologous stem cell transplantation (ASCT) and revealing very mild vitreous opacities as well as (b) six months later and demonstrating an interval increase in vitreous opacities.

antibiotics, with a recent review revealing 37.8 % of patients additionally undergoing PPV.[7,8] Explantation of the GDI was performed in 70.5 % of patients and has been suggested for the purpose of removing the contaminated foreign body and keeping intravitreal antibiotics in the eye longer.[7,10,11] While VA outcomes were similar, eyes that received GDI explantation had lower rates of evisceration and enucleation.[9]

Interestingly, three cases placed the tube into the subconjunctival space; however, one had the tube reinserted into the AC after the eye was quiet for several months and infection recurred requiring eventual implant removal.[12–14] The latter case may have resulted due to the

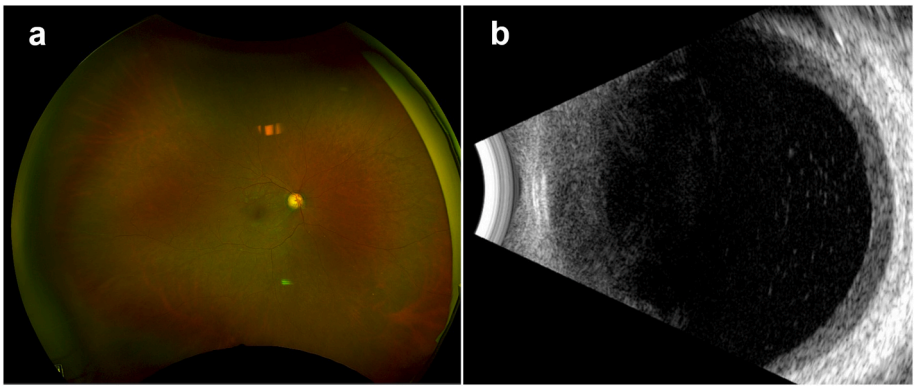

Fig. 5. At the most recent follow-up visit, (a) color fundus photograph and (b) B-scan of the right eye showing no evidence of active disease. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

infection having already spread to the subconjunctival space surrounding the GDI prior to tube withdrawal. Of note, while there are cases of GDI-related endophthalmitis successfully managed without removing the tube, we suggest a much lower threshold for tube removal or enucleation in the context of PVRL where life may be at stake. Above all however, the insertion of new hardware into an eye with suspected or known PVRL should be primarily avoided.

This case additionally highlights the importance of early diagnosis and maintaining high suspicion for VRL, as the consequences can be devastating. The diagnosis of PVRL is often delayed, with median time from symptom onset to diagnosis ranging from 6 to 40 months.[5] PVRL is often misdiagnosed as uveitis, with initial management often including corticosteroids. However, steroids have cytotoxic effects on lymphoma cells, inducing a transient improvement in up to half of patients and limiting the sensitivity of diagnostic testing.[15] Diagnosis via vitreous biopsy can additionally be challenging for both technical and pathological reasons.[16] Cytological assessment alone requires special expertise, with negative predictive value estimated at 60 %, and sensitivity ranging from 45 to 81 %.[17–19] As such, detection of PVRL may now be aided by employing multiple diagnostic modalities including cytology, flow cytometry, mutational analysis (e.g., *MYD88*, *CD79B*), cytokine levels, and PCR for monoclonal gene rearrangements. In our case, initial PPVs at an outside institution returned negative and were likely performed in the setting of steroid use. The patient was kept on steroids for uveitis and diagnosis was delayed until repeat vitreous biopsy four years later returned positive. This time likely allowed for the PVRL to embed within the glaucoma reservoir fibrous capsule as well as progress to CNS disease. While distinguishing PVRL from uveitis remains a diagnostic challenge, clinicians must always consider the shortcomings of diagnostic testing as delays in diagnosis enable continued disease progression.

Interestingly, despite her multiple intraocular relapses, the patient had a relatively benign systemic disease course. The period from onset of reported uveitis to diagnosis of PVRL was 4 years, and following initiation of therapy, she has persevered an additional 5 years despite two intraocular relapses. Her survival is striking in the context of a frequently deadly disease and may provide insight into prognostic factors for PVRL. The mortality of PVRL ranges from 9 to 81 % in follow-up periods ranging from 12 to 35 months.[3] The leading cause of death is considered CNS involvement, and as such, prognosis may hinge on preventing CNS dissemination. Early diagnosis is essential as patients with PVRL diagnosed before CNS involvement carry a survival advantage of 60 months over 35 months.[20] Preventing relapse of PCNSL is also crucial as these patients carry a poor prognosis, with a median survival time of only 7.2 months from first disease progression to death.[21] In our case, while the patient had multiple intraocular relapses, her CNS disease was treated promptly and then aggressively with ASCT. She was maintained on prophylactic systemic therapy for an extended period without CNS relapses, which we suspect explains her favorable systemic course.

The prognostic significance of CNS involvement also raises the question of management practices and risk factors for CNS progression. For patients with isolated PVRL, the benefit of prophylactic systemic treatment for occult CNS disease is yet unclear. Several studies demonstrate decreased incidence of CNS spread and longer survival with use of prophylactic systemic therapy.[3] High doses of systemic chemotherapy have also been suggested alongside local therapies for PVRL even in the absence of CNS involvement.[3] Our patient was treated systemically until deemed free of CNS disease and then received ASCT. However, she was enucleated soon after due to intraocular relapse and developed activity in the contralateral eye four months later. The rapid progression to bilateral disease may suggest that following ASCT, either systemic therapy failed to eliminate occult CNS disease or the GDI eye quickly reseeded the CNS. The rates of CNS progression in PVRL vary from 33 to 60 % and the risk factors for progression are largely unknown.[19,22] Early studies however suggest genetics may play a prognostic role. Relative to systemic DLBCL, PCNSL is characterized by frequent MYD88 and CD79B mutations, with the latter also having been detected in the vitreous of patients with PVRL.[23] Certain of these CD79B and gene mutations in PVRL have been associated with increased likelihood for and more rapid CNS progression.[24,25] PCNSL with CD79B mutations additionally may have poorer overall survival, though the data on MYD88 mutations are more inconclusive.[26–29] Of note, most studies were limited by sample size, but altogether may indicate a role for genetic markers in diagnosis, targeted therapies, and prognosis of PVRL. Mutational analysis in our patient revealed mutations in MYD88 and ZRSR2 of which the significance remains unknown and further study is necessary. However given her history of multiple relapses and rapid progression to the contralateral eye potentially suggestive of subclinical or incompletely treated CNS disease, she continues to be monitored very closely with a low threshold to reinitiate systemic therapy upon any sign of relapse.

4. Conclusion

We describe a case of a GDI functioning as a probable sanctuary site for recurrent PVRL. Following a delay in diagnosis, the patient presented with CNS disease and experienced multiple intraocular recurrences despite both systemic and local therapies. Eventual enucleation was required and revealed the GDI had been the underlying source for continued local relapse. This case highlights the importance of early recognition and diagnosis of VRL, and that sanctuary sites of malignancy should be considered when recurrent relapses occur despite complete treatment. There should be a low threshold for removal of the GDI or enucleation, as well as for prophylactic systemic therapy given the risk of CNS progression and mortality with incomplete treatment.

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CRediT authorship contribution statement

Debora H. Lee: Writing – original draft, Visualization, Data curation. **Jennifer Li-Wang:** Writing – review & editing, Visualization, Data curation. **Patricia Chevez-Barrios:** Writing – review & editing, Supervision, Investigation, Data curation. **Amy C. Schefler:** Writing – review & editing, Supervision, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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