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(CASE REPORT)

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Retinal capillary hemangioblastoma in a patient with von Hippel-Lindau syndrome: Case report and a brief review of the literature

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Abstract

Von Hippel–Lindau (VHL) syndrome is a hereditary disorder predisposing patients to multiple vascular tumors, including retinal capillary hemangioblastomas (RCH), which may cause progressive vision loss if undetected or untreated. We describe a 31-year-old man with known VHL syndrome who presented with a blind, painful left eye following recurrent retinal detachments and secondary glaucoma despite multiple surgeries. Ophthalmic examination demonstrated no light perception, rubeosis iridis, and corneal degeneration. B-scan ultrasonography revealed total retinal detachment with an intraocular mass showing calcification, and orbital MRI demonstrated an enhancing intraocular lesion without extraocular extension. Systemic imaging excluded other VHL manifestations. Due to intractable pain and irreversible ocular damage, enucleation was performed. Histopathology confirmed a capillary hemangioblastoma composed of thin-walled vascular channels positive for CD31 and CD34. Genetic testing verified a pathogenic VHL mutation. Postoperatively, the patient achieved pain relief, with no recurrence or systemic involvement at two-year follow-up. This case illustrates advanced ocular complications of VHL-associated retinal hemangioblastoma and highlights the importance of early diagnosis, coordinated multidisciplinary management, and lifelong surveillance to prevent devastating visual outcomes.

Keywords: Retinal capillary hemangioblastomas; Von Hippel–Lindau; Ocular; Enucleation; Retinal detachment; Immunohistochemistry

1. Introduction

Retinal capillary hemangioblastoma (RCH) is a benign vascular neoplasm that threatens vision. Von Hippel-Lindau (VHL) syndrome is a rare autosomal dominant neoplastic disorder, and retinal capillary hemangioblastoma is often the presenting symptom in up to 73% of cases. [1] An autosomal dominant condition, VHL syndrome is a neoplastic disorder caused by a germline mutation in the VHL tumor suppressor gene on chromosome 3p25-26. This mutation dysregulates hypoxia-sensitive factors (HIFs), HIF-1 and HIF-2, leading to excess production of vascular endothelial growth factor (VEGF) and other angiogenic factors. [2] Single RCH can sometimes happen on its own, but the occurrence of several lesions, or bilateral lesions, is typical for VHL and raises the need for lifelong systemic monitoring for visceral malignancies like renal cell carcinoma and pheochromocytoma. [3]

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Just as the treatments involved in retinal vascular tumor management have evolved, modern clinical practices are opening new possibilities in the management of VHL-related tumors through minimally invasive therapeutic options, such as the HIF-2α inhibitor belzutifan, which recently received FDA approval. [4] Most eye tumors, however, remain highly problematic at all stages of the disease. Vitreous hemorrhage, exudative, and tractional retinal detachments can cause secondary neovascular glaucoma, chronic ocular decompensation, and may lead to pain and blindness. [5]

Currently, there is a scarcity of resources documenting histopathological changes in RCH in advanced ocular VHL disease, especially regarding atypical sequelae such as osseous metaplasia. While the formation of intraocular bone is usually associated with phthisis bulbi and chronic inflammatory conditions, in a retinal capillary hemangioblastoma, it is extremely unusual. [6] Knowing these final structural modifications is important for better prognostic stratification and for approaching patients symptomatically who do not respond to conservative or systemic treatment. We present an unusual case of a 31-year-old male with VHL disease who presented with a blind, painful eye, illustrating the progression to end-stage disease and the unusual histopathologic feature of osseous metaplasia in the retinal capillary hemangioblastoma.

2. Case Presentation

2.1. Clinical Presentation

A 31-year-old man with a known diagnosis of von Hippel-Lindau (VHL) syndrome presented with a blind, painful left eye. His clinical course had been progressive and complicated, marked by recurrent retinal detachments necessitating multiple surgical interventions and the development of secondary glaucoma. The pain was attributed to elevated intraocular pressure and chronic ocular decompensation. Visual loss was complete in the affected eye, the consequence of longstanding retinal detachment, chronic ischemia, and tumor-related architectural disruption. The overall presentation was consistent with end-stage ocular disease driven by an underlying retinal capillary hemangioblastoma in the setting of a well-established hereditary tumor predisposition syndrome.

2.2. Physical Examination, History, Risk Factors, and Initial Testing

On examination, the left eye demonstrated no light perception, elevated intraocular pressure, and a shallow anterior chamber. Slit-lamp evaluation revealed corneal degenerative changes and rubeosis iridis. Funduscopic examination was limited due to media opacity and total retinal detachment. The patient reported a longstanding history of retinal surgeries, scleral buckle placement, and laser photocoagulation. Family history was significant for VHL syndrome. Risk factors included his hereditary syndrome, young age, and prior retinal vascular lesions. Initial testing included intraocular pressure measurement, B-scan ultrasonography, fluorescein angiography when feasible, and complete blood count with systemic baseline labs.

2.3. Radiographic Studies and Diagnostic Considerations

Comprehensive imaging was undertaken to characterize the ocular lesion and screen for systemic manifestations of VHL syndrome. B-scan ocular ultrasonography demonstrated total retinal detachment, an intraocular mass with mixed echogenicity, and evidence of calcification consistent with osseous metaplasia. Magnetic resonance imaging of the orbits with gadolinium contrast revealed an enhancing left intraocular mass without extraocular extension, scleral breach, or optic nerve involvement. MRI of the brain and spine was performed to exclude central nervous system hemangioblastomas, a hallmark of VHL disease; no intracranial or spinal lesions were identified. Computed tomography of the abdomen and pelvis was assessed for renal cell carcinoma, pancreatic cysts, and pheochromocytoma, all of which were absent. Retinal fluorescein angiography had previously characterized the vascular tumor. Diagnostic considerations included RCH, cavernous hemangioma, retinal vasoproliferative tumor, and choroidal melanoma, though the clinical context of VHL syndrome strongly favored hemangioblastoma as the primary diagnosis.

2.4. Tumor Board Multidisciplinary Discussion

The multidisciplinary tumor board convened a discussion centered on the management of an end-stage blind and painful eye in a young patient with VHL syndrome and no evidence of systemic tumor burden. Participants included ophthalmology, medical oncology, neuroradiology, pathology, and genetic counseling. The discussion acknowledged that all vision-preserving options had been exhausted through prior surgical and laser interventions. Given the irreversible visual loss, intractable pain, and progressive ocular disorganization, the board unanimously recommended left eye enucleation for palliation and definitive histopathologic diagnosis. Genetic counseling and ongoing systemic surveillance for other VHL-associated neoplasms were also strongly recommended for the patient and his at-risk family members.

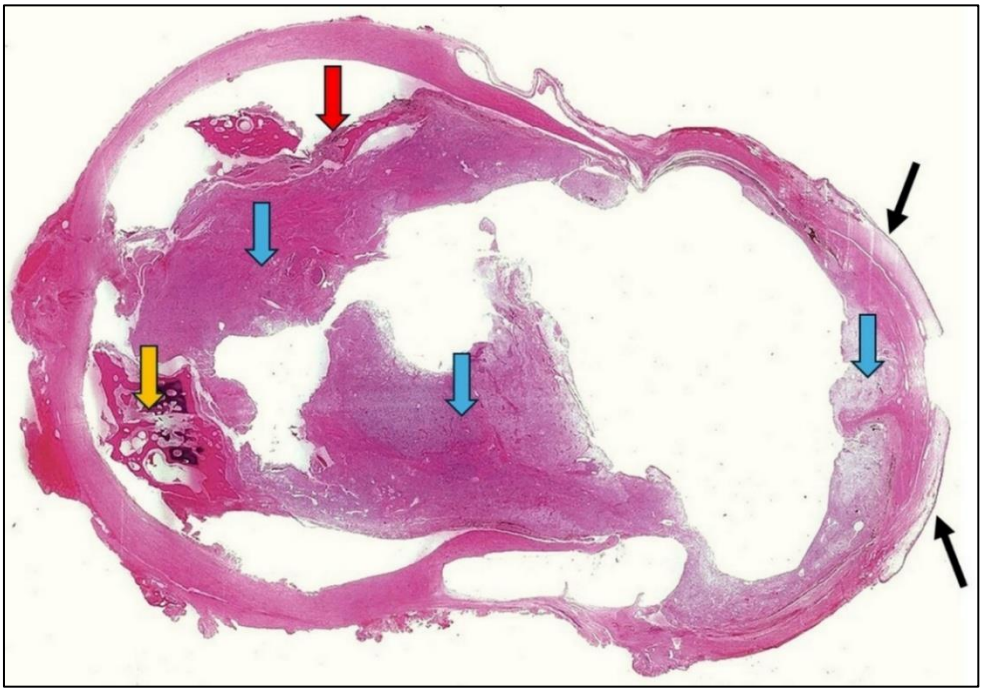
2.5. Surgery, Pathology, Immunohistochemistry, and Molecular Studies

The patient underwent left eye enucleation under general anesthesia. The globe was submitted in its entirety for ophthalmic pathology evaluation. Gross examination revealed a disorganized eye with total retinal detachment and an intraocular mass. (Figure 1) Microscopic examination demonstrated a capillary hemangioblastoma characterized by a dense network of thin-walled capillaries lined by endothelial cells immunoreactive for CD31 and CD34, confirming its vascular nature. Interspersed stromal cells exhibited predominantly bland, hyperchromatic nuclei with occasional bizarre forms and lipid-laden vacuolated cytoplasm; these cells were immunoreactive for vimentin and neuron-specific enolase (NSE), consistent with the neoplastic stromal cell phenotype of hemangioblastoma. Stromal cells were negative for Cam5.2, glial fibrillary acidic protein (GFAP), and CD68, excluding carcinoma, glial, and histiocytic differentiation, respectively. Rare mast cells were highlighted by Toluidine Blue staining. The tumor exhibited large cystic formation without necrosis or notable mitotic activity and was entirely confined to the globe without scleral excavation, vortex vein involvement, or perineural involvement. Focal peritumoral reactive gliosis with rare Rosenthal fibers was identified. Additional findings included chorioretinal scars, osseous metaplasia, retrocorneal fibrosis, and corneal degenerative changes. (Figure 2 A, B, C)

Molecular testing for the VHL gene mutation was performed on germline DNA, confirming a pathogenic variant consistent with the clinical diagnosis of retinal capillary hemangioblastoma (RCH). After enucleation, a temporary conformer was inserted, followed by fitting of a custom-made artificial eye (prosthesis) by an ocularist at 6 weeks post-surgery.

2.6. Outcome and Follow-Up

Following enucleation, the patient experienced complete relief of ocular pain. Pathologic staging confirmed tumor confinement to the globe with negative surgical margins, which was a favorable prognostic indicator. The patient was enrolled in a structured VHL syndrome surveillance program in accordance with established guidelines, including annual brain and spine MRI, annual abdominal imaging, and regular ophthalmologic evaluation of the contralateral eye for early detection of new hemangioblastomas. Genetic counseling was provided to first-degree relatives. Should new retinal lesions develop in the right eye, early intervention with laser photocoagulation or anti-VEGF therapy was planned. During a two-year follow-up, there was no evidence of recurrence or tumor in the contralateral eye or other organs.



Blue arrow: Tumor, hemangioblastoma.; Yello arrow: Hemangioblastoma with osseous metaplasia.; Red arrow: Hemangioblastoma arising from detached retina.; Black arrows: Cornea

Figure 1 Microscopic examination of the enucleated eye with hemangioblastoma

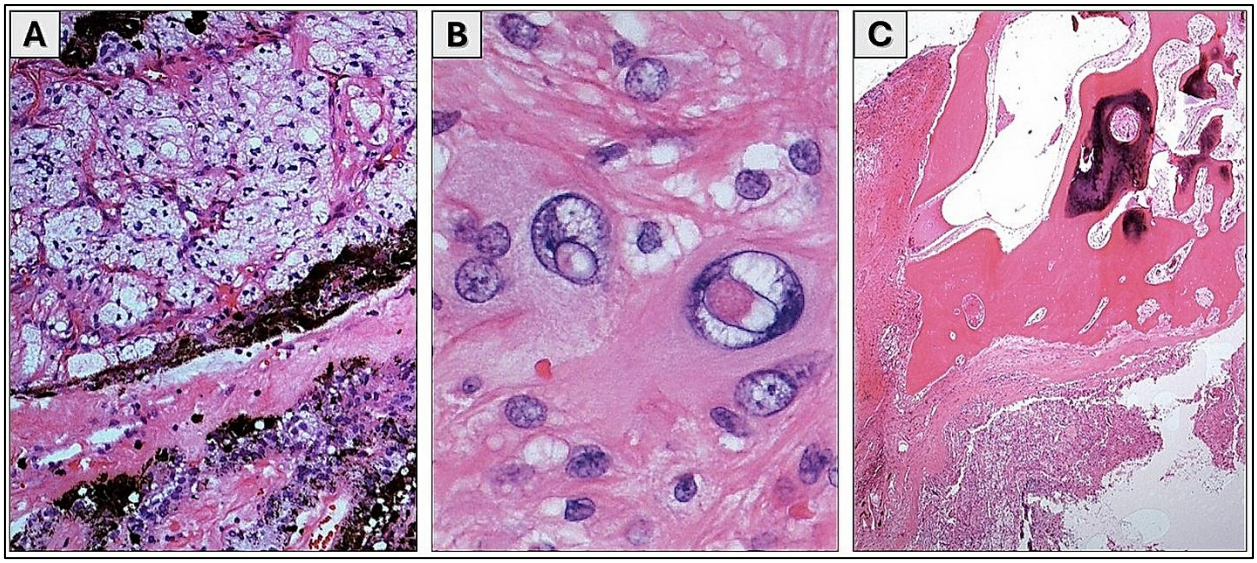


Figure 2 Microscopic examination of the enucleated eye with hemangioblastoma

2A: Intermediate power view showing hemangioblastoma with vacuolated cells arising from detached retina (H&E Stain X40).; 2B: High power view showing hemangioblastoma with bizarre nuclei, but no mitosis or necrosis (H&E Stain X60).; 2C: Low power view showing hemangioblastoma with osseous metaplasia (H&E Stain X60).

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

Von Hippel-Lindau syndrome (VHL) is a rare autosomal-dominant inherited disorder characterized by the development of various neoplasms, including retinal hemangioblastomas, central nervous system hemangioblastomas, renal cell carcinomas, pheochromocytomas, and pancreatic cysts. [7]

Retinal hemangioblastoma was first reported in 1911 by the German ophthalmologist Eugen von Hippel, who initially called them retinal vascular tumors. [8] Retinal capillary hemangioblastomas have also been shown to be the most common and earliest manifestation in 49%-85% of patients with VHL disease. [8] Early detection and management are essential to prevent blindness. [9] Retinal capillary hemangioblastomas in VHL can be unilateral (42%) or bilateral (58%). [10] Risk factors such as younger age at onset, involvement of the contralateral eye, and type of genetic variant have been associated with a higher likelihood of multiple retinal tumors. [11]

The incidence of VHL is approximately 1 in 36,000. [7] Retinal hemangioblastomas have nearly complete penetrance, ranging from 90% to 100% in VHL patients aged 60 years or older. [7] The prevalence of retinal hemangioblastoma is 1 in 73080. [8]

Although retinal hemangioblastomas are histologically benign, they can result in significant ocular complications. These complications include progressive exudation, macular edema, retinal detachment, and eventual vision loss if left untreated. [12] Therefore, early identification is imperative, as ophthalmologic findings may provide the first indication of systemic VHL and require further genetic testing and surveillance for associated neoplasms.

3.2. Pathogenesis, Pathophysiology

The von Hippel-Lindau (VHL) tumor suppressor gene is critical for the development of. In the context of VHL syndrome, which leads to the development of hemangioblastoma, there is a suggestion of a germline mutation and a somatic “second hit”. [3] The VHL protein (pVHL) is responsible for the degradation of hypoxia-inducible factors (HIF) such that, in the absence of pVHL, HIF is constitutively stabilized (i.e., HIF is not degraded). Such a scenario leads to "overdrive" of the angiogenic process, with an overwhelming increase in vascular endothelial growth factor (VEGF) expression. Such a mechanism accounts for the excessive proliferation of thin-walled, leaking capillaries in the patient's retinal lesions. [13] This cycle of chronic pseudohypoxia leads to an endless cycle of angiogenesis and further impairment of the integrity of the blood-retina barrier.

The stromal cell is the neoplastic component of RCH and originates from primitive, developmentally arrested hemangioblasts. In the microscopic evaluation of the patient's enucleated eye, these stromal cells are seen to have lipid-laden, vacuolated cytoplasm. These cells also express neuron-specific enolase. [14] The invasion of these cells into retinal structures disrupts the normal function of neural cells, and the invasion also forms a large, highly vascularized mass. In this case, the tumor's clinical progression, characterized by repeated retinal detachments, rubeosis iridis, and secondary glaucoma, is due to excessive angiogenesis and the subsequent fibrovascular overgrowth. Furthermore, the pathology report describes the tumor mass as having osseous metaplasia, a sign of severe architectural disruption. This unusual feature indicates a prolonged state of chronic inflammation and ischemia of the eye, in which multipotent cells transform into mesenchymal bone-forming cells, suggesting a total breakdown of the eye. [15]

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical-radiology-pathology, lab, diagnosis, management, and outcome)

3.3.1. Clinical Presentation

The typical presentation of retinal hemangioblastoma (RH) in VHL disease is highly variable. A case study from a 2024 publication on Juxtapapillary RH reported a mean age of 26 years (range 6-51 years), and our patient falls within the anticipated clinical spectrum. [16,17] Peripheral retinal lesions are common, but RH near the optic disc or macula poses treatment challenges due to risks to central vision. [15] This patient had end-stage disease with a blind, painful eye. This result is consistent with other research showing that patients without follow-up developed complications requiring enucleation. [18] This underscores a critical lesson that Juxtapapillary RHs, though benign and slow growing, pose a serious threat to vision if left untreated and could lead to exudative retinal detachment and neovascular glaucoma, among other complications. [18,19] Early diagnosis and treatment of RH with laser photocoagulation can preserve vision, in stark contrast to the end-stage disease seen in our patient. [12,19] Our case is representative of advanced treatment rather than untreated pathology. Despite previous retinal surgeries and laser photocoagulation, secondary glaucoma, progressive retinal detachment, and ocular disorganization led to vision loss.

3.3.2. Diagnostic Workup

A 2022 study on retinal hemangioblastomas emphasized the use of imaging to improve diagnostic accuracy. [20] B-scan ultrasonography assesses tumor size and retinal detachments and complements fluorescein angiography to characterize the tumor's vascular composition. [20] Consequently, feeding vessels and late vascular leakage can be identified. Our case highlights the use of MRI to rule out CNS hemangioblastomas. The IHC (CD31, CD34 positive in endothelial cells; vimentin, NSE positive in stromal cells) is the gold standard for confirmation and eliminating differential diagnoses for tumors. [20, 21] Identifying germline VHL mutation solidifies diagnosis and is crucial for counseling [20, 21]. The VHL gene encodes a tumor suppressor protein linked to the degradation of HIF. VHL function loss leads to stabilization of HIF-α and upregulation of angiogenic pathways, which influences the growth of highly vascular tumors. [3,14] In progressive cases with opaque media or total retinal detachment, ophthalmoscopic evaluation may be inadequate, and thus, B-scan ultrasonography and orbital MRI become essential. Imaging can help distinguish hemangioblastoma from other vascular tumors. [8,11] Histopathologically, RHs are characterized by dense capillary networks and lipid-rich stromal cells, features representative of the tumor's neoplasticity. Immunohistochemistry shows CD31 and CD34 in vascular channels, and stromal cells show markers including vimentin and neuron-specific enolase. [1] The findings help differentiate hemangioblastoma from other intraocular tumors.

3.3.3. Management

RH management has evolved, with a focus on vision-preserving treatments for early-stage disease. A 2024 prospective trial showed that several sessions of indocyanine green-enhanced transpupillary thermotherapy (ICG-TTT) can be a valid new alternative for Juxtapapillary RH, with local tumor control in 75% of cases and visual acuity remaining stable or even improving. [22] A 2024 case report demonstrated, in the context of VHL, significant tumor regression with belzutifan, a hypoxia-inducible factor 2α (HIF-2α) inhibitor, in an optic nerve hemangioblastoma. [23] These findings support minimally invasive therapies and systemic medical therapies. However, in our case, management is enucleation, which represents the final option for end-stage disease. The case underscores that the goal of modern VHL care is to prevent this endpoint through rigorous surveillance and early intervention. [16, 22,23] In addition, enucleation is accepted, particularly when vision cannot be restored. Surgery, in such instances, serves both palliative and diagnostic goals, hence providing pain relief.

3.3.4. Outcome and Follow-up

Post-enucleation, our patient experienced comprehensive pain relief and no recurrence at two years. This aligns with expectations for a tumor confined to the globe with negative surgical margins. [18,23] The critical aspect is the patient's

enrollment in a structured, lifelong VHL surveillance program since patients with VHL are at risk for new tumors. [15,17] The literature also emphasizes a multidisciplinary approach and individualized treatment plans. [12,19] The two-year follow-up with no new manifestations is a positive early outcome, but recent literature highlights risks. For instance, the probability of developing renal cell carcinoma, a leading cause of death in VHL, increases with age. [15,17,21,23] Therefore, follow-up is not dictated by duration, but rather by adherence to ongoing and guideline-based surveillance.

4. What Have We Learned from This Case?

Recent literature confirms that, while advances in molecular understanding and treatment options for von Hippel–Lindau (VHL)-associated retinal capillary hemangioblastoma (RCH) continue to evolve, the fundamental principles of early diagnosis and timely intervention remain the most critical determinants of visual outcomes. This case aligns with the broader literature by illustrating that RCH can lead to severe secondary complications, such as retinal detachment and neovascular glaucoma. It underscores the importance of confirmatory histopathology and immunohistochemistry for a definitive diagnosis. Current research emphasizes early therapeutic strategies, including novel approaches such as indocyanine green-guided transpupillary thermotherapy (ICG-TTT) and systemic agents like belzutifan. [23] In the present case, clinical decision-making was guided not only by tumor control but also by palliation and diagnostic confirmation. As such, this case highlights the need for early intervention, lifelong surveillance, and a multidisciplinary approach to detect and manage RCHs at an early, treatable stage.

Abbreviations

- Retinal capillary hemangioblastomas (RCH; Von Hippel–Lindau (VHL);
- Immunohistochemistry (IHC)

Conclusion

This case underscores the potential for profound ocular morbidity in von Hippel–Lindau (VHL) syndrome when retinal hemangioblastoma progresses unchecked despite prior interventions. It highlights the importance of early recognition and lifelong multidisciplinary follow-up to prevent irreversible vision loss and to guide timely, definitive management. The detailed clinical, imaging, and histopathologic correlation in this report adds to the limited documentation of advanced VHL-related ocular disease culminating in enucleation. This case highlights the importance of proactive screening for at-risk individuals and coordinated care among ophthalmologists, geneticists, and oncologists. Such collaboration is critical not only for ocular preservation but also for detecting systemic manifestations that may influence patient outcomes in VHL syndrome.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

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- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

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