Optometry: Open Access
ISSN: 2476-2075

Editorial Open Access

Uveal Melanoma: Current Perspectives in Diagnosis and Management

Ethan Martin*

Department of Neuroscience, University of Adelaide, Australia

Introduction

Uveal melanoma is the most common primary intraocular malignancy in adults, arising from melanocytes within the uveal tract, which includes the iris, ciliary body, and choroid. Though rare compared to cutaneous melanoma, uveal melanoma carries significant morbidity and mortality due to its high potential for metastasis, particularly to the liver. The disease often presents asymptomatically or with subtle visual changes, making early detection challenging. Advances in imaging, histopathology, and molecular genetics have improved diagnostic precision, risk stratification, and treatment planning. Understanding the clinical features, diagnostic modalities, and therapeutic options is essential for optimizing patient outcomes [1,2].

Discussion

Clinically, uveal melanoma may manifest as a pigmented or non-pigmented intraocular mass, often detected during routine ophthalmic examinations. Symptoms, when present, include blurred vision, visual field defects, flashes, or floaters. Iris melanomas typically present with visible lesions and are less aggressive, whereas ciliary body and choroidal melanomas often remain asymptomatic until significant growth occurs. The prognosis is closely linked to tumor size, location, and histopathologic characteristics [3-6].

Diagnostic modalities have evolved significantly. Fundus examination remains the initial step, supplemented by ultrasonography, which provides information on tumor thickness, internal reflectivity, and extrascleral extension. Optical coherence tomography (OCT) and fluorescein angiography aid in assessing retinal involvement and subretinal fluid, particularly for smaller or juxtapapillary lesions. Magnetic resonance imaging (MRI) and computed tomography (CT) are valuable for evaluating orbital or systemic involvement. Fine-needle aspiration biopsy can provide cytologic confirmation and allow molecular profiling, including gene expression analysis, which is crucial for metastatic risk assessment. Chromosomal abnormalities, such as monosomy 3 and gains in chromosome 8q, are associated with higher metastatic potential [7,8].

Treatment strategies depend on tumor size, location, and patient-specific factors. Eye-conserving therapies, such as plaque brachytherapy or proton beam therapy, aim to eradicate the tumor while preserving vision. Enucleation is reserved for large tumors, those with significant ocular complications, or when conservative treatment is infeasible. Emerging treatments, including targeted molecular therapies and immunotherapies, are under investigation, particularly for metastatic disease. Regular systemic surveillance, especially for hepatic metastasis, is vital due to the high incidence of liver involvement [9,10].

Conclusion

Uveal melanoma represents a rare but clinically significant intraocular malignancy with a high risk of metastasis. Early detection through comprehensive ocular examination and advanced imaging is critical, as is accurate risk stratification via molecular profiling. Treatment strategies range from eye-preserving radiotherapy to enucleation, tailored to tumor size, location, and patient factors. While localized disease can often be managed effectively, metastatic uveal melanoma continues to pose a therapeutic challenge, underscoring the need for continued research in targeted therapies and immunomodulation. Integration of multidisciplinary care, vigilant surveillance, and patient-centered decision-making remains the cornerstone of managing this complex ocular malignancy. Advances in diagnostics, treatment modalities, and molecular understanding promise improved survival and quality of life for affected patients.

References

1. Nichols KK, Foulks GN, Bron AJ, Glasgow BJ, Dogru M et al. (2011) The International Workshop on Meibomian Gland Dysfunction: Executive Summary. Invest Ophthalmol Vis Sci 52: 1922-1929.
2. Olson MC, Korb DR, Greiner JV (2003) Increase in tear film lipid layer thickness following treatment with warm compresses in patients with meibomian gland dysfunction. Eye Contact Lens 29: 96-99.
3. Goto E, Monden Y, Takano Y, Mori A, Shimmura S et al. (2002) Treatment of non-inflamed obstructive meibomian gland dysfunction by an infrared warm compression device. Br J Ophthalmol 86: 1403-1407.
4. Greiner JV (2013) Long-term (12-month) improvement in meibomian gland functions and reduced dry eye symptoms with a single thermal pulsation treatment. Clin Exp Ophthalmol 41: 524–530.
5. Thode AR, Latkany RA (2015) Current and Emerging Therapeutic Strategies for the Treatment of Meibomian Gland Dysfunction (MGD). Drugs 75: 1177–1185.
6. Schiffman RM, Christianson MD, Jacobsen G, Hirsch JD, Reis BL (2000). Reliability and Validity of the Ocular Surface Disease Index. Arch Ophthalmol 118: 615-621.
7. Efron,N (2018) Contact Lens Complications. 4th edn Elsevier, USA.
8. Sim HS, Petznick A, Barbier S, Tan JH, Acharya UR et al. (2014) Randomized, Controlled Treatment Trial of Eyelid-Warming Therapies in Meibomian Gland Dysfunction. Ophthalmol Ther 3: 37–48.
9. Murakami DK, Blackie CA, Korb DR (2015) All Warm Compresses Are Not Equally Efficacious. Optom Vis Sci 92: 327- 333.
10. Blackie CA, Solomon JD, Greiner JV, Holmes M, Korb DR (2008) Inner eyelid surface temperature as a function of warm compress methodology. Optom Vis Sci 85: 675–683.

***Corresponding author:** Ethan Martin, Department of Neuroscience, University of Adelaide, Australia, Email: ethan937@gmail.com

Received: 03-May-2025, Manuscript No: omoa-25-171446, **Editor Assigned:** 05-May-2025, pre QC No: omoa-25-171446 (PQ), **Reviewed:** 17- May -2025, QC No: omoa-25-171446, **Revised:** 23-May-2025, Manuscript No: omoa-25-171446 (R), **Published:** 29-May-2025, DOI: 10.4172/2476-2075.1000319

Citation: Ethan M (2025) Uveal Melanoma: Current Perspectives in Diagnosis and Management. Optom Open Access 10: 319.