Editorial Open Access

Argyll Robertson Pupil: Clinical Significance and Underlying Mechanisms

Ashish Tiwari*

Department of Optometry, Birla Institute of Technology and Science, India

Introduction

Argyll Robertson pupil (ARP) is a distinctive ocular sign characterized by bilateral small pupils that constrict during accommodation but fail to constrict in response to direct light stimulation. First described by the Scottish ophthalmologist Douglas Argyll Robertson in the 19th century, this clinical feature is most commonly associated with neurosyphilis, though it may rarely appear in other neurological disorders. The presence of ARP indicates damage to the midbrain pretectal area or the pupillary light reflex pathway, highlighting its value as a neuro-ophthalmic marker. Understanding its pathophysiology, clinical presentation, and differential diagnosis is essential for accurate recognition and timely intervention [1,2].

Discussion

Clinically, patients with Argyll Robertson pupil present with bilateral, small, irregular pupils that show minimal or absent response to bright light but retain normal constriction when focusing on a near object (accommodation reflex). This dissociation between the light reflex and near response is sometimes summarized as "light-near dissociation." Pupils are typically asymmetric in size but maintain their characteristic response pattern. Patients may be asymptomatic regarding vision, although visual disturbances can occur if the underlying condition progresses [3,4].

The pathophysiology of ARP is rooted in selective involvement of the pretectal area of the dorsal midbrain, which contains neurons responsible for the pupillary light reflex. In neurosyphilis, tabes dorsalis leads to degeneration of the dorsal columns and associated midbrain structures, sparing the edinger–Westphal nucleus pathways responsible for accommodation. This selective impairment explains why the pupillary light reflex is lost while the near response is preserved. Other causes of light-near dissociation include diabetic neuropathy, multiple sclerosis, pineal tumors, and midbrain lesions, but ARP remains most classically linked to syphilitic infection [5-8].

Diagnosis relies primarily on clinical examination, including careful assessment of the pupillary responses to light and near stimuli. Slit-lamp examination may assist in ruling out anterior segment abnormalities, while neuroimaging is reserved for atypical presentations to exclude structural lesions. Serologic testing for syphilis, including VDRL and FTA-ABS, is essential when ARP is suspected, as it often signals neurosyphilitic involvement requiring urgent treatment. Differential diagnosis must consider other causes of light-near dissociation, such as Adie's tonic pupil, which typically presents unilaterally and shows delayed constriction to near stimuli [9,10].

Conclusion

Argyll Robertson pupil remains a hallmark sign of neurosyphilis and a classic example of light-near dissociation. Its recognition is clinically significant, offering insights into midbrain integrity and serving as a window to underlying systemic or neurologic disease. Careful pupillary assessment, combined with serologic testing and appropriate management of the primary cause, is essential for optimizing patient outcomes. While ARP itself may persist despite

therapy, timely diagnosis and treatment of the underlying condition can prevent further neurologic damage and reduce morbidity. As a historical yet still relevant clinical sign, Argyll Robertson pupil underscores the enduring importance of meticulous ophthalmic examination in modern medicine.

References

- Stochholm K, Juul S, Juel K, Naeraa RW, Højbjerg Gravholt C (2006) Prevalence, incidence, diagnostic delay, and mortality in Turner syndrome. J Clin Endocrinol Metab 91: 3897–3902.
- Gravholt CH, Viuff MH, Brun S, Stochholm K, Andersen NH (2019) Turner syndrome: Mechanisms and management. Nat Rev Endocrinol 15: 601–614.
- Khater F (2006) Autoimmune diseases in Turner syndrome: An overview. Acta Biomed 90. 341–344.
- Klein KO, Rosenfield RL, Santen RJ, Gawlik AM, Backeljauw PF, et al. (2018) Estrogen Replacement in Turner Syndrome: Literature Review and Practical Considerations. J Clin Endocrinol Metab 103, 1790–1803.
- Wikiera B, Mulak M, Koltowska-Haggstrom M, Noczynska A (2015) The presence of eye defects in patients with Turner syndrome is irrespective of their karyotype. Clin Endocrinol 83: 842–848.
- Chrousos GA, Ross JL, Chrousos G, Chu FC, Kenigsberg D, et al. (2014)
 Ocular Findings in Turner Syndrome: A Prospective Study. Ophthalmology 9: 926–928.
- Denniston AKO, Butler L (2004) Ophthalmic features of Turner's syndrome. Eye 18: 680–684.
- Adhikary HP (2005) Ocular manifestations of Turner's syndrome. Trans Ophthalmol Soc United Kingd 101: 395–402.
- Kapoor S, Dasgupta J (1979) Chromosomal anomaly in a female patient with anterior lenticonus. J Int Ophthalmol 179: 271–275.
- Accorinti M, La Cava M, Speranza S, Pivetti-Pezzi P (2020) Uveitis in Turner's syndrome. Graefe's Arch Clin Exp Ophthalmol 240: 529–532.

*Corresponding author: Ashish Tiwari, Department of Optometry, Birla Institute of Technology and Science, India, Email: tiwari284@gmail.com

Received: 03-May-2025, Manuscript No: omoa-25-171447, Editor Assigned: 05-May-2025, pre QC No: omoa-25-171447 (PQ), Reviewed: 17- May -2025, QC No: omoa-25-171447, Revised: 23-May-2025, Manuscript No: omoa-25-171447 (R), Published: 29-May-2025, DOI: 10.4172/2476-2075.1000320

Citation: Ashish T (2025) Argyll Robertson Pupil: Clinical Significance and Underlying Mechanisms. Optom Open Access 10: 320.

Copyright: © 2025 Ashish T. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.