A Case of Holmes Adies pupil following herpes zoster ophthalmicus

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Abstract:
Holmes Adies pupil is a type of pupil with parasympathetic denervation in which the affected pupil is large, poorly reacting to light, but reacting better to accommodation. The pupil is supersensitive to cholinergics but dilates to mydriatics normally. Holmes Adie pupil by itself is rare. Herpes zoster ophthalmicus as a cause of Holmes Adie pupil is still a more rare presentation. This presentation is one such rare combination of Holmes Adie pupil following herpes zoster ophthalmicus.

Keyword: Holmes Adie pupil, Herpes zoster, Ciliary ganglionitis, Denervation supersensitivity.

INTRODUCTION HISTORICAL NOTE
Holmes Adie pupil was first described by Adie in his first publication under the title “Pseudo-Argyll Robertson pupils with absent tendon reflexes: a benign disorder simulating tabes dorsalis,” in 1931. In the consecutive year itself he published another report of 8 patients under the title of “Tonic pupils and absent tendon reflexes; a benign disorder; its complete and incomplete forms.” He considered this type of pupillary defect to be an incomplete form of Holmes adies syndrome and clearly postulated that tonic pupil is not secondary to syphilis. The names of Gordon Holmes' and W James Adie are traditionally attached to the syndrome of myotonic pupils and tendon areflexia. Some 50 years earlier in 1881, mydriasis with pupillary paralys is described by Hughlings Jackson. In 1914 Oloff found negative Wasserman reactions in blood and CSF in an 18 year old boy with tonic pupils, thereby showing that syphilis which was previously implicated was not the cause. The characteristics of Holmes adies pupil include:

1. It is larger than its fellow pupil
2. It poorly reacts to light
3. It reacts to accommodation very slowly and remains tonically small for a longer time
It exhibits supersensitivity to cholinergic drops like 0.125% pilocarpine.

CASE REPORT:
Our patient is a 35 year old gentleman who was admitted with history of pain in the left side of the forehead and left eye of 3 months duration following an episode of Herpes Zoster involving the area of ophthalmic division of the left trigeminal nerve (left V1). Patient was first seen only three months after the onset of Herpes zoster. Physical examination showed scar over the left V1 area associated with conjunctival congestion. The uncorrected visual acuity of the right eye was 6/6 and that of the left was 6/18. The right pupil was 3mm in diameter which was reacting to light (both direct and consensual) and accommodation normally. Left pupil was 7mm in diameter, which did not react to light both direct and consensual. On near vision, the left pupil very slowly reacted, which remained as small pupil even after the near stimulus was taken off. On Instillation of 0.125% of pilocarpine the normal pupil did not show any reaction, but the pupil of the affected pupil on the left side became constricted after a period of 25 minutes after instillation of pilocarpine. Extraocular movements of both sides were full. The ankle jerks were normal on both sides. The blood RPR reaction and HIV by ELISA were unreactive. Hence a diagnosis of Holmes Adie pupil was made secondary to ciliary ganglionitis caused by Herpes Zoster infection.

DISCUSSION:
Holmes Adie pupil or tonic pupil occurs due to diseases of the ciliary ganglion. Infections like bacterial or viral may be causative. Diseases of the autonomic nervous system that affect the ciliary ganglion can also cause Holmes Adie pupil. Following damage to the parasympathetic ciliary ganglion, reinnervation and up regulation of the post-synaptic receptors occurs, leading to denervation supersensitivity. In ciliary ganglionitis, the light fibres, the near fibres, and the dilator fibres can be affected differentially. This results in an efferent pupillary defect manifested as poor light reflex in the affected eye, with a preserved consensual light reflex in the opposite eye. The accommodation reflex is delayed due to the differential involvement of the fibres that carry convergence across the ciliary ganglion. The denervation supersensitivity manifests as a sustained contraction of the iris muscles even to 1/8 the dose of pilocarpine. Holmes Adie syndrome includes similar pupillary changes with loss of ankle reflexes and this occurs in diseases of autonomic system, sometimes it may also be idiopathic. The common complications of herpes zoster ophthalmicus include eyelid rash and vesicles, loss of lashes, conjunctivitis, corneal ulcers, corneal inflammation and scarring, uveitis, glaucoma, Retinitis, cystoid macular edema, and optic neuritis. Holmes Adie pupil in a setting of herpes zoster ophthalmicus is thus a rare presentation.

TREATMENT Patient was treated with pilocarpine drops and tricyclic antidepressants for the pain.

PROGNOSIS Recovery may be delayed. The affected pupil is reported to become smaller than the normal pupil. The pupillary response may not completely recover.

REFERENCES:


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**Figure 1**: THIS SHOWS THE EVIDENCE OF THE HERPETIC SCAR OVER LEFT OPHTHALMIC AREA WITH DILATED PUPIL.

**Figure 2**: PICTURE SHOWS MIOSIS TO 0.125%PILOCARPINE IN THE LEFT EYE WHEREAS THE RIGHT EYE DOES NOT. THE LEFT PUPIL IS SMALLER THAN THE RIGHT.

**Figure 3**: SHOWS A SMALLER PUPIL FOLLOWING ACCOMADATION.