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Left Eye Brown Syndrome: A Case Report

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Abstract:

Background: Aim to report an unusual case of left eye Brown syndrome with no amblyopia.

Case report: Here we report a 10 years old male child presented with abnormal head posture(AHP), normal vision in both eyes with no history of diplopia. A detail history taken from child and his mother. At the time of presentation his vision was normal in both eyes. He was misdiagnosed as a case of ocular palsy elsewhere and was treated outside one year back. He had visual acuities of 6/6 in both eyes on Snellen visual acuity chart. There was a left hypotropia in primary position of gaze incorporated with limitation of elevation in adduction and positive forced duction test. On external inspection of a face shows an AHP and face turn to right. These positive findings demonstrate case of left eye Brown syndrome with no amblyopia and diplopia.Slit lamp biomicroscopic examination of both eyes was within normal limit. Strabismus and orthoptic examination weredone to confirm the diagnosis. **Conclusion**: The most cases of congenital Brown syndrome may be associated with amblyopia but here no amblyopia was detected. Eye health workers, ophthalmic assistant and optometrist are motivating to quickly refer all patients with ocular motility disorders to ophthalmologist.

Keywords: abnormal head posture (AHP), Brown syndrome,amblyopia,forced duction test

Introduction: Brown syndrome is ocular motility defect identify by limitation of elevation of aneye inadduction.[1] It is one of the causes of an isolated inferior oblique muscle palsy.[2]Brown syndromes is also called superior oblique tendon sheath syndrome which is mostly mechanical restriction defect. It leads to difficulty in moving the affected eye upward in adduction with mostly positive forced duction test. Normally the oblique muscles have antagonist action in moving the eyes in vertical plane. The primary action of superior oblique muscle is intorsion, secondary is depression and tertiary are abduction. The primary action of inferior oblique is extorsion, secondary is elevation and tertiary are abduction. In other words, the action of inferior oblique muscle is to move eyeball upward and inward in adduction that is with eye looking in the direction of forehead glabella similarly action of superior oblique muscle is to move eyeball downward and inward that is with the eye looking towards same side of nose. Superior and inferior oblique both having antagonist action. Brown syndrome is also called superior oblique tendon sheath syndrome (due to short tendon than normal) which was first described by Harold Brown in 1950.[3] Most cases of Brown syndrome are congenital The acquired cases are due to infection, inflammation, mass or neoplasm, sports injury or trauma and idiopathic.[4] Brown syndromes does not have gender differentiation. It is mostly unilateral sporadic and in 10% cases it is seen bilateral. [1]

Case history and Examination:A 10-year-old male child presented to pediatric eye out patient department at Sagarmatha Choudhary Eye Hospital, Lahan,Nepal with a history of abnormal head posture and strabismus. His mother had noticed an ocular misalignment and head abnormality 5 years before. He was misdiagnosed as a case of ocular palsy elsewhere and was treated outside one year back. He had visual acuity of 6/6 in both eyes on Snellen visual acuity chart. There was no history of trauma or fall, no history of double vision (uniocular or binocular diplopia). There was no history of fever, vomiting, loss of consciousness, epilepsy, skin rashes or lesion. Birth history was normal with full term normal hospital delivery. Developmental milestones were normal. Immunization history is complete till date. Family history of strabismus was not significant. Old photograph of a child showed no significant abnormal head posture.

Work up of a patient: Strabismus and orthoptic evaluation shows visual acuity unaided in both eyes 6/6. AHP (abnormal head posture) shows face turn to right tilted and left chin up position. Cover -uncover test unaided shows right hypertropia (R/L). On head tilt test towards right side R/L increases while on tilting head towards left show orthotropia. Right eye extraocular movements show full and free in all diagnostic cardinal gaze

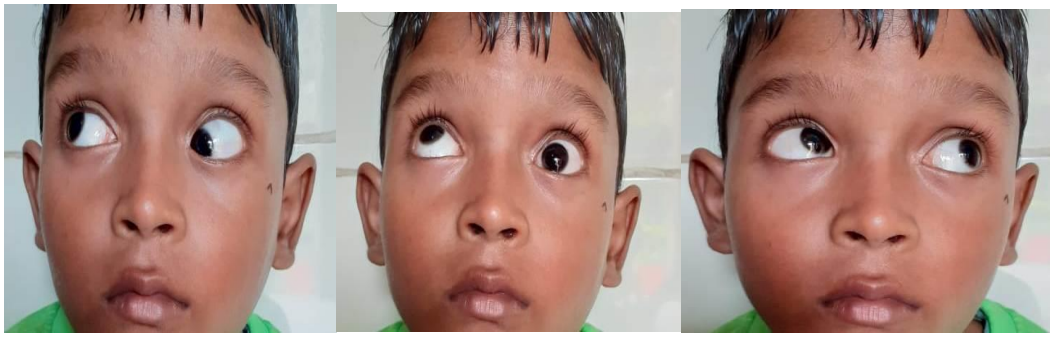
position, while left eye show (-4) in dextroelevation that is limitation of elevation in adduction. PBCT/PBRT (prism barcover test/ prism bar reflex test) shows following findings in chart:

Dextroelevation 80 PD R/L	Elevation 40PD R/L	Levoelevation ORTHO
Dextroversion 60 PD R/L	Primary position 10 PD R/L	Levoversion ORTHO
Dextrodepression R/L increased	Depression ORTHO	Levodepression ORTHO

PD: prism diopter

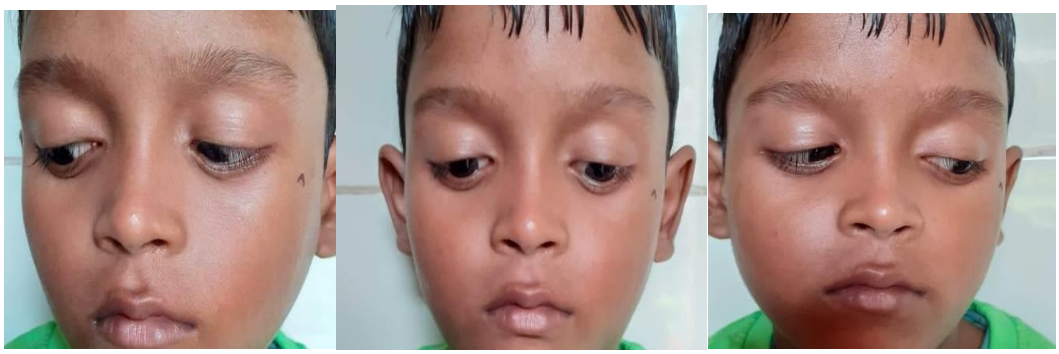
Stereopsis checked using Titmus fly test (polaroid vectograph) which is absent in primary position, but it is normal (40 second of arc with AHP). Forced duction test is positive. The Worth four dot tests using red – green goggles that's is red color in front of right eye and green color in front of left eye (WTDT) shows binocular single vision with AHP in both distance and near check. WFDT shows alternate suppression in primary position. Wet retinoscopy with cyclopentolate (1%) eye drops both eyes shows normal finding. Right eye shows +2.25D both horizontal and vertical axis. Left eye shows +2.25D both horizontal and vertical axis. No amblyopia was detected. Slit lamp biomicroscopic examination shows normal anterior segment and fundus of both eyes within normal limit. Intraocular pressure with Goldman applanation tonometry is 18mm Hg in both eyes. His systemic examination was completely normal.Following are the picture of a patient with nine cardinal gazes from (A to I). Picture J showing abnormal head posture. We couldn't perform Hess chart here because of unavailability.

A B C



D E F

G H I



J K L

Picture K and A showing left eye marked limitation of elevation in adduction whereas picture C showing normal elevation of right eye in laevoelevation. A diagnosis of acquired left brown syndrome was made with no history of amblyopia and diplopia. His parents were prevailed and inform about the condition as well as

management plan. We advised surgical plan to parents to correct squint. We planned left eye superior oblique tendon lengthening along with right eye superior rectus recession (2-3mm).However, parents refused for surgery and back out after first visit.

Discussion: Harold Brown in 1950 reported a patient with mild downward deviation the involved eye on adduction, v pattern exotropia, absence of overaction of ipsilateral superior oblique with restriction of elevation in adduction and positive forced duction test. [6] However, in our case a positive forced duction test with limitation of elevation in adduction and no overaction of ipsilateral superior oblique strongly suggestive of Brown syndrome. [7] There is differential diagnosis of Brown syndrome consist ipsilateral inferior oblique paresis [8], monocular elevation deficiency syndrome, orbital fibrosis syndrome / fibrosis of inferior rectus [7.8], simulated Brown syndrome (like in cases of orbital floor fracture, traumatic tenosynovitis, idiopathic orbital inflammation, metastasis to superior oblique muscle, orbital metastasis, frontal sinus osteoma or blunt orbital trauma) and superior oblique overaction. Isolated ipsilateral inferior oblique palsy is having negative forced duction test with ipsilateral superior oblique overaction. This patient had positive forced duction test with no superior oblique overaction which suggests Brown syndrome.Forced duction test (FDT) is basically used to differentiate between mechanical restriction or neurological (paralytic) cause. In FDT the paralyzed muscle is moved into passive direction of action of that muscle to rule out cause. As there is no history of trauma, loss of consciousness, no history of periorbital ecchymosis, fever, diplopia or proptosis which signifies the least chances of orbital floor fracture, orbital tumor or metastasis. Ipsilateral inferior rectus palsy or congenital fibrosis of inferior rectus had positive forced duction test with restriction of elevation of eye both in adduction and abduction which is absent in Brown syndrome.[7]There are two etiological types of brown syndrome one is congenital form and other is acquired form. Patient old photographs and detail history from parents indicate that this is a case of acquired (idiopathic) type with no diplopia and amblyopia. Acquired form of Brown syndrome mostly seen in conditions like Sjogren's syndrome [8], juvenile chronic arthritis [9], rheumatoid arthritis[10,11],SLE[12] and post-surgery induced (surgery of superior oblique tendon). [13] In this case the absence of diplopia, no history of suppression amblyopia, (R/L) right hypertropia with left hypotropia and an abnormal head posture (AHP-face turn to right) support the possibility that idiopathic etiology. Most likelihood cause of AHP with head tilt to left and chin up is to avoid diplopia.

Conclusion:

In the above case report, we diagnosed idiopathic acquired Brown syndrome with AHP. The most cases of congenital Brown syndrome may be associated with amblyopia and probably diplopia but here no amblyopia was detected. There is a significant possibility that the AHP found in this patient would have been less serious if the condition had been diagnosed earlier. Eye health workers, ophthalmic assistant and optometrist are motivating to quickly refer all patients with ocular motility disorders to ophthalmologist.

References:

1. Wilson ME, Eustis HS et al. Brown's syndrome. Surv Ophthalmol. 1989 Nov-Dec;34(3):153–7. [PubMed]
2. Alonso-Valdivielso JL, Alvarez Lario B, Alegre Lopez J et al. Acquired Brown's syndrome in a patient with systemic lupus erythematosus. Ann Rheum Dis. 1993 Jan;52(1):63–4. [PMC free article] [PubMed]
3. Brown HW. Congenital structural muscle anomalies. In: Allen JH, editor. Strabismus Ophthalmic Symposium I. (Google Scholar)
4. Attarzadeh A, Hoseinirad A et al. Brown syndrome in one pair of dizygotic twins: a case report. Cases J. 2010;3:1. [PMC free article] [PubMed]
5. American Academy of Ophthalmology. San Fransisco: American Academy of Ophthalmology, Eye MD Association; 2008. Paediatric Ophthalmology and Strabismus BCSC Section 6: 2007-2008. [Google Scholar]
6. Olver J, Laidler P. Acquired Brown's syndrome in a patient with combined lichen sclerosus et atrophicus and morphoea. Br J Ophthalmol. 1988 Jul;72(7):552–7. [PMC free article] [PubMed]
7. Von Noorden GK, Campos EC. 6th ed. St. Louis: Mosby; 2002. Binocular Vision and Ocular Motility-Theory and Management of Strabismus. [Google Scholar]
8. Brahma AK, Hay E, Sturgess DA, Morgan LH. Acquired Brown's syndrome and primary Sjogren's syndrome. Br J Ophthalmol. 1995 Jan;79(1):89–90. [PMC free article] [PubMed]
9. Wang FM, Wertenbaker C, Behrens MM, Jacobs JC. Acquired Brown's syndrome in children with juvenile rheumatoid arthritis. Ophthalmology. 1984 Jan;91(1):23–6. [PubMed]
10. Killian PJ, McClain B, Lawless OJ. Brown's syndrome. An unusual manifestation of rheumatoid arthritis. Arthritis Rheum. 1977 Jun;20(5):1080–4. [PubMed]

11. Cooper C, Kirwan JR, McGill NW, Dieppe PA. Brown's syndrome: an unusual ocular complication of rheumatoid arthritis. Ann Rheum Dis. 1990 Mar;49(3):188–9. [PMC free article] [PubMed]
12. McGalliard J, Bell AL. Acquired Brown's syndrome in a patient with SLE. Ann Rheum Dis. 1993 May;52(5):385–6. [PMC free article] [PubMed]
13. Bradbury JA, Martin L et al. Acquired Brown's syndrome associated with Hurler-Scheie's syndrome. Br J Ophthalmol. 1989 Apr;73(4):305–8. [PMC free article] [PubMed]