Case Report

Recovery of Post Traumatic Brown's Syndrome

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Brown's syndrome[1, 2, 3] is a motility defect which is characterized by an inability to raise the adducted eye above the horizontal midline, less or no elevation deficit in abducted position. There is slight down shoot of the adducting involved eye with widening of the palpebral fissure on adduction. Exodeviation usually increases as the eyes are moved upward in the midline (V-pattern).

CASE REPORT

A seven years old male child presented with an abnormal head posture after trauma by donkey's hoof two months back. There was no history of pain, tenderness and double vision. The patient gave no history of any previous ocular, periocular or orbital surgery. Systemic evaluation revealed no evidence of sinusitis and juvenile chronic arthritis. Family history was unremarkable. On general physical examination child was very much co-operative and well appearing with no signs of acute distress.

There was a scar mark of trauma at the junction of medial and middle third of the left eyebrow. There was left-sided head turn with a slight chin up position (Fig. 1). Left eye was slightly hypotropic. Extra ocular movements showed restricted elevation in adduction of left eye (Fig. 2). There was no tenderness and palpable mass in the trochlear region of left eye.

Visual acuity was 6/6 in both eyes. Pupils were round and normally reacting to light. Eyelids, adnexa and anterior segment examination showed no abnormality. Fundi were normal.

Forced duction test was positive. On the basis of the above clinical findings the patient was diagnosed as a case of Brown's syndrome. The parents were reassured and the patient was put on oral syrup of Ibuprofen (1 TSF * TDS). The child was called for follow up after one month who showed improvement in subsequent visits and after three months there was complete recovery (Fig. 3, 4).

DISCUSSION

Brown's syndrome also known as superior oblique tendon sheath syndrome was first described by Harold W Brown in 1950. He hypothesized that

Brown's syndrome occurred as a result of innervational deficit to inferior oblique muscle with secondary contracture of the anterior sheath of superior oblique tendon. Electromyography did not support the idea[4].

Later, Brown HW[5] redefined the disease and categorized it into congenital (short anterior sheath of superior oblique tendon) and simulated sheath syndrome (all cases caused by anomaly other than short anterior sheath of superior oblique tendon). In mid 1970s, Park[6] and Crawford disagreed the idea of short anterior tendon sheath. They proposed that Brown's syndrome was caused by tight or short superior oblique tendon. Electromyographic studies confirmed this idea.

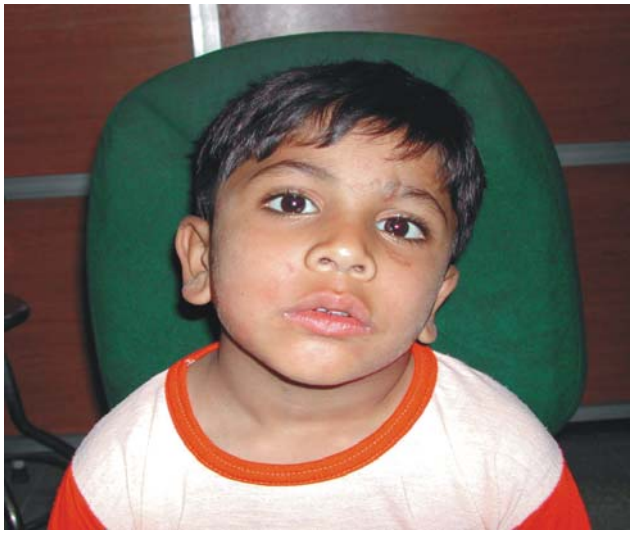

Fig. 1: Left sided head tilt with slight chin up position

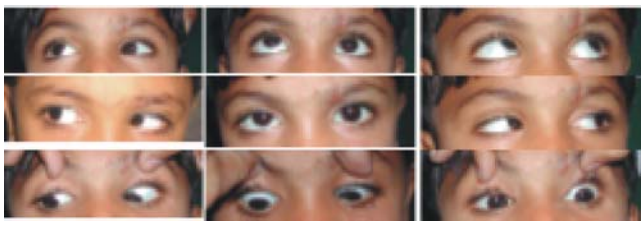

Fig. 2: Left hypotropia in primary position and limitation of elevation in adduction of left eye (left brown's syndrome).

It is interesting that some Greek doctors have attributed the arrogant posture of Alexander the Great[7, 8] to this syndrome. In the Lancet (April 1996) John Lascaratos from Athens University report that Alexander the Great might have suffered from Brown's syndrome of left eye as he had to hold his head with raised chin, face turned to right and neck tilting to the left.

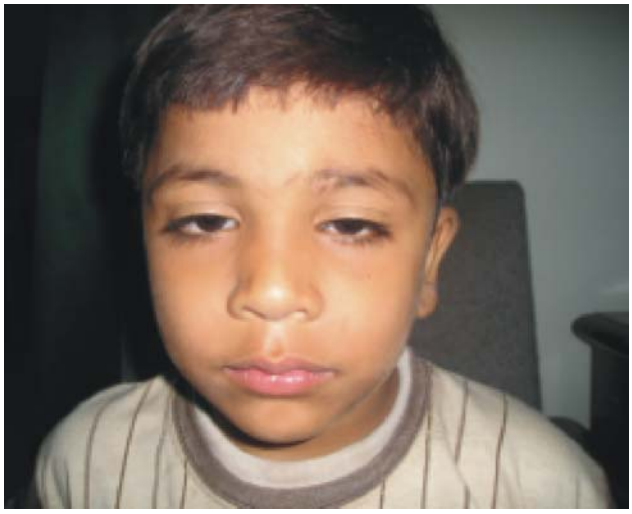

Fig. 3: Normal head position after recovery

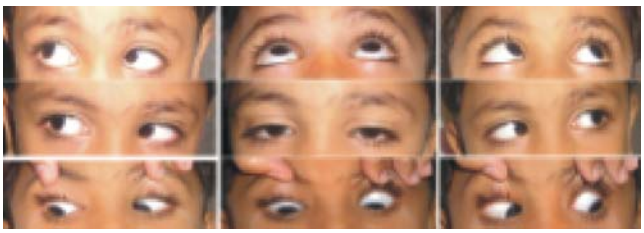

Fig. 4: Normal extra ocular movements after recovery

So far various etiologies of Brown's syndrome have been described. Iannaccone A has reported a family with three siblings having unilateral late onset Brown's syndrome. Congenital Brown's syndrome has been reported in monozygotic twin girls[9] with reversed asymmetry (mirror image). Delayed development of trochlea is also reported to be the cause of Brown's syndrome[10]. Acquired cases of Brown's syndrome are related to peritrochlear scarring and adhesion caused by chronic sinusitis[11], trauma, Blepharoplasty[12], trochleitis with superior oblique myositis, adult rheumatoid arthritis, juvenile chronic arthritis, systemic lupus erythematosis[13], superior nasal orbital mass, glaucoma implant and scleral buckling procedures.

The conditions included in the differential diagnosis of Brown's syndrome are primary superior oblique over action, inferior oblique paresis and monocular elevation deficit. Forced duction test is

negative in superior oblique over action and Inferior oblique paresis. There is V-pattern exotropia in Brown's syndrome while patients with primary superior oblique over action and inferior oblique paresis have A-pattern exotropia in down gaze and A-pattern esotropia in up gaze respectively. In monocular elevation deficit[14], elevation is worse in all positions.

No laboratory tests are required in the work up of congenital Brown's syndrome while in acquired cases systemic lupus erythematosis, juvenile rheumatoid arthritis and rheumatoid arthritis should be excluded.

Management of Brown's syndrome includes pharmacotherapy with oral NSAIDS and local corticosteroids injections in the trochlear region. It is usually indicated in acquired cases of active inflammation which can be post traumatic, related to periocular surgeries or rheumatoid arthritis. The exact mechanism of action is not known but may inhibit Cyclooxygenase[15] activity and Prostaglandin synthesis, inhibition of leukotrienes synthesis, lysozomal enzyme release, lipoxygenase activity, neutrophil aggregation and various cell membrane functions.

Surgical treatment is indicated when there is chin elevation and severe limitation of elevation in adduction, which interfere with quality of life. Surgical procedures[16, 17] include superior oblique tendon lengthening, tendon expander technique, tenotomy, and superior oblique recession while sheathectomy and superior oblique trochlear luxations have been abandoned.

Few cases of spontaneous resolution of congenital as well as acquired Brown's syndrome have been reported in the literature. In T.J. Kaban's[18] series 10% cases of the presumed congenital Brown's syndrome experienced a complete spontaneous resolution. Luigo Capasso[19] and coworkers reported a case of bilateral Brown's syndrome in a four years old girl. After seven months there was spontaneous resolution in right eye while her left eye did not show any significant change over thirty-six months of follow up. Gregersen and Rindziunski[20] described ten cases of Brown's syndrome out of which three developed normal motility after sometime. Waddell[21] reported resolution of Brown's syndrome in 24 out of 36 (67%) patients who showed improvement from 1 to 14 years after the initial diagnosis. According to WN Clarke[22], Brown's syndrome associated with over action of contra lateral inferior oblique muscle probably begins as bilateral Brown's syndrome, followed by spontaneous improvement of Brown's syndrome on one eye and subsequent secondary inferior oblique over action.

Our case of Brown's syndrome developed post traumatic Brown's syndrome which resulted from inflammation of the superior oblique tendon. The condition resolved after 3 months when the inflammation had subsided. It can be correlated with the study of Helveston[23] and associates who described fluid accumulation and vascular distention in the sheath as the cause of limitation of superior oblique tendon motion through the trochlea. When inflammation is controlled, fluid is absorbed and vascular distention settles down leading to resolution of Brown's syndrome. Oral NSAIDS (as in our case) accelerate the control of inflammation.

CONCLUSION

Cases of post traumatic Brown's syndrome should be observed for spontaneous recovery.

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REFERENCES

1. **Brown HW:** Congenital structural muscle anomalies. In Allen JH (Ed): Strabismus Ophthalmic Symposium 1. St Louis: CV Mosby. 1950: 205
2. **Brown HW:** Isolated inferior oblique paralysis: An analysis of 97 cases. Trans Am Ophthalmol Soc. 55:1957; 415

3. **Brown HW:** In Haik GM (Ed): Strabismus Symposium of the New Orleans Academy of Ophthalmology. St Louis: CV Mosby, 1962
4. **Brown HW:** True and simulated superior oblique tendon sheath syndromes. Doc Ophthalmol 34:1973; 123
5. **Brown HW:** True and simulated superior oblique tendon sheath syndromes. Doc Ophthalmol 1973; 34 (1):123-36
6. **Park MM, M:** Superior oblique tendon sheath of Brown. Am J Ophthalmol 1975 Jan; 79(1): 82-6
7. HW Brown: Strabismus symposium 1. St. Louis, Mosby, 1950: 205-236.
8. **Brown HW:** Congenital structural motor anomalies in strabismus. In Allen JH (ed): Ophthalmic Symposium 1, St Louis: CV Mosby, 1950:205-229
9. **Katz NNK, Whitmore PV, Beauchamp GR:** Brown's syndrome in twins. J Pediatr Ophthalmol Strabismus: 1981; 18:32
10. **Lauer S.A., Sauer H, Pak Sm:** Brown's syndrome diagnosed following repairs of an orbital roof fracture: a case report. J. Craniomaxillofacial Trauma 1998; 4(4) : 20-2
11. **Hermann JS.** Acquired Brown's syndrome of inflammatory origin. Arch Ophthalmol: 1978;96:1228-32
12. **Levine MR, Boyton J, Tenzel RR, Miller Gr:** Complications of Blepharoplasty. Ophthalmic surg 1975;6: 47-53
13. **Whitefield L, Isenberg DA, Brazier DJ, Forbes J:** Acquired Brown's syndrome in Systemic lupus erythematosis. Br. J. Rheumatol: 1995;34:1092-4
14. **Kanski JJ:** Clinical Ophthalmology (5th ed): 2003;549
15. **Wright KW:** Brown's syndrome diagnosis and management. Trans Am Ophthalmol soc 1999; 97:1023-109
16. **Scott AB, Knapp P:** Surgical treatment of the superior oblique tendon sheath syndrome. Arch Ophthalmol: 1972 Sept; 88(3): 282-6
17. **Wright KW:** Color Atlas of Ophthalmic Surgery-Strabismus-Philadelphia, Pa: Lippincott; 1991:201-219
18. **TJ Kaban, K Smith, RB Orton et al:** Natural history of presumed congenital Brown's syndrome. Arch Ophthalmol 1993 July; vol 111 No. 7
19. **Capasso Luigi, Torre Angelo, Gagliardi Vincenzo, Magli Adriano**: Spontaneous resolution of congenital bilateral Brown's syndrome Ophthalmologica: 2001; 215: 372-375
20. **Gregersen E, Rindziunski:** Brown's syndrome, a longitudinal long-term study of spontaneous course. Acta Ophthalmol:1993; 71:371
21. **Waddell E:** Brown's syndrome revisited. Br Orthop J: 1982; 39:17
22. **Clarke WN, Noel LP:** Brown's syndrome with contralateral inferior oblique overaction: A possible mechanism. Can J Ophthalmol: 1993; 28:213
23. **Helveston EM, Merriam WW, Ellis FD et al:** The trochlea: A study of the anatomy and physiology. Ophthalmology: 1982; 89:124