Recovery of Post Traumatic Brown’s Syndrome

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Case Report

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Brown’s syndrome 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.

Later, Brown HW 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 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.

It is interesting that some Greek doctors have attributed the arrogant posture of Alexander the Great 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.

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 with reversed asymmetry (mirror image). Delayed development of trochlea is also reported to be the cause of Brown's syndrome. Acquired cases of Brown's syndrome are related to peritrochlear scarring and adhesion caused by chronic sinusitis, trauma, Blepharoplasty, trochleitis with superior oblique myositis, adult rheumatoid arthritis, juvenile chronic arthritis, systemic lupus erythematosus, 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, 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 erythematosus, 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 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 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 series 10% cases of the presumed congenital Brown’s syndrome experienced a complete spontaneous resolution. Luigo Capasso 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 described ten cases of Brown’s syndrome out of which three developed normal motility after sometime. Waddell 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, 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 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