



Isolated Trochlear Nerve Palsy in Post Traumatic Brain Injury: Incidental Detection Using Vestibular Screening Tests

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Abstract

Isolated trochlear Nerve Injury (TNI) following Traumatic Brain Injury (TBI) is uncommon. There are diagnostic challenges in the early detection of isolated TNI and there is currently no agreed screening tool for its diagnosis. We report a 30 year-old gentleman with severe TBI, who was detected to have diplopia during routine screening vestibular tests. He was subsequently diagnosed to have isolated TNI following a positive Bielschowsky head tilt test. This was managed conservatively and his symptom of diplopia improved 5 months following head injury.

Keywords: Traumatic brain injury; Trochlear nerve injury; Vestibular screening tests

Introduction

Cranial Nerve (CN) injury is a potential complication of TBI [1]. However, unilateral traumatic lesion of trochlear nerve is relatively uncommon [2] and its incidence has been reported between 0.23% to 13% [1,3-5]. Previous studies have suggested that CNs I, VII, VIII and in particular the olfactory nerve; are most frequently injured after TBI [6].

The diagnosis of CN injury after TBI can be challenging. Several TBI patients suffer from altered consciousness, agitation, cognitive problems like attention and visuospatial deficits. They also sustain neurological deficits like aphasia and neglect, making it difficult for history taking and physical examination. The clinician has to differentiate CN neuropathies from vestibular ocular dysfunction, as both can present with diplopia and dizziness.

Currently, there is no consensus on the diagnosis and treatment of CN injury post TBI [1]. We report a case of post TBI related TNI in which the diagnosis was provoked on routine vestibular screening.

Case Report

A 30-year-old Chinese male with no past medical history sustained severe TBI after falling approximately 3.5 meters from a rooftop glass panel while at work. Glasgow Coma Scale (GCS) score was 15 on arrival to the emergency department. Initial Computed Tomography (CT) brain revealed acute subarachnoid haemorrhage along sulci of the left frontal lobe with contusion over the left temporal lobe. There was no evidence of skull vault fracture or raised intracranial pressure (Figures 1A and 1B). His TBI was managed conservatively. His GCS score remained 15 throughout his acute neurosurgical stay. He also sustained a right clavicular fracture which was managed conservatively. He was transferred to an inpatient rehabilitation facility after 2 weeks post TBI.

Initial physical examination did not reveal any focal neurological signs and motor power was full throughout. Using the West mead Post Traumatic Amnesia (PTA) scale [7], daily recordings showed that his PTA duration was 21 days. Initial PTA score was 3 out of 7.

A few days following admission, he complained of giddiness with diplopia. The diplopia was described as binocular with a vertical component and was persistent throughout the day but worsened with downward gaze. It resolved on unilateral occlusion. On further examination, he had diplopia on downward gaze but there was no obvious loss of in torsion, depression and adduction of his eyes. Examination of his other CNs, motor power, limb ataxia, gait and balance were normal.

To rule out vestibular-ocular causes for giddiness, vestibular screening was done during a physiotherapy session. Both Dix-Hallpike and Supine Log Roll tests were negative. However, his

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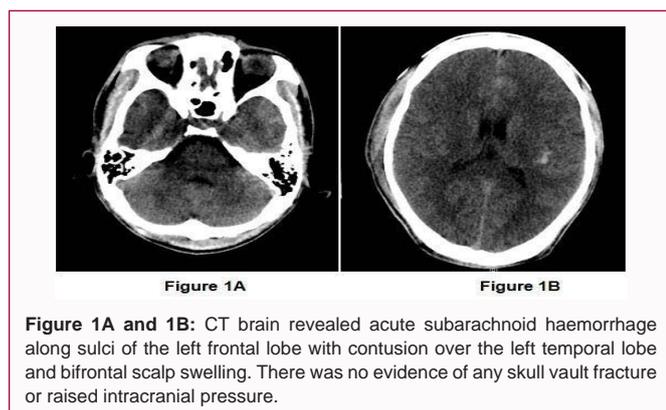
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diplopia was significantly worse on left head tilt during both tests.

He was subsequently referred to the ophthalmologist and found to have a positive Bielschowsky head tilt test on left head tilt. He was diagnosed to have left isolated superior oblique nerve palsy from TNI. This was managed conservatively.

He was discharged from the rehabilitation centre at 5 weeks post-TBI requiring supervision in ambulation and basic activities of daily living. He had some residual deficits in memory and abstract thinking at time of discharge. There was no noticeable recovery of his left superior oblique nerve palsy during inpatient stay. During outpatient follow-up at 5 months post injury, he had persistent left TNI with a positive Bielschowsky head tilt test but his symptoms of diplopia improved significantly.

Discussion

Epidemiology and aetiology

The most common acquired cause of TNI is trauma [8]. TBI accounts for approximately one third of cases with trochlear nerve palsy [9]. The incidence of TNI in TBI patients ranges from 0.23% to 13% [1,3-5]. In one study involving retrospective analysis of 31 TBI patients, the incidence of TNI was 3.2% [3]. In another study evaluating 3417 patients after head injury, 0.23% of patients had isolated TNI [1].

Studies have shown that most patients with TNI have moderate to severe TBI [9] and this is consistent with TBI in this patient since his PTA was 21 days, in spite of a normal GCS of 15, indicative of severe TBI. PTA duration is used as an index of TBI severity. PTA is defined as inability to lay down memories from one day to the next, and is assessed subjectively [7]. The duration of PTA is defined as the period from time of injury until return of continuous memory. However, there is evidence that traumatic TNI can also occur with mild TBI [2].

Other causes of acquired trochlear nerve palsy include vascular, neoplastic or inflammatory diseases, which are less common than traumatic causes [10].

TNI and TBI

The trochlear nucleus is located at the inferior colliculus, ventrolateral to the cerebral aqueduct [9]. The fibres pass to decussate in the superior medullary velum [9]. It courses around the midbrain's lateral aspect, passes between the posterior cerebral and superior cerebellar arteries and courses along the tentorium cerebelli [9]. It subsequently pierces the dura to near the cavernous sinus and enters the superior orbital fissure [9].

The trochlear nerve supplies only the superior oblique muscle. The muscle's functions include intorsion, depression and abduction.

The trochlear nerve is susceptible to injury after TBI due to its long intracranial course (75mm) and thin structure [2,9]. The rigid tentorium lies next to the trochlear nerve where it can be easily compressed [2].

Most traumatic TNI are associated with midbrain parenchymal contusions and hematomas following impact of midbrain against the tentorium, as well as injury in the cisterns along the nerve's extra-axial course [4].

Other mechanisms include orbital fractures or frontal or orbital impact causing a contusion against the trochlear nerve's attachment at the medullary velum [2,9]. This patient had extensive bilateral frontal scalp swelling which could suggest a significant impact on his forehead possibly leading to TNI.

This patient also had left frontal lobe injury and diffuse traumatic subarachnoid haemorrhage which were likely causative mechanisms for TNI. No midbrain lesions were seen on CT brain.

Clinical presentation and investigations

In subjects with TNI who complain of diplopia, this is often vertical and torsional; and worse on downward gaze and gaze away from side of affected muscle. Reading or walking down stairs often worsens diplopia due to the downward head position [2]. Patients can have torticollis to the side of the unaffected eye with depressed chin. This is a compensatory head posture to place the unaffected eye in the position of least deviation [2].

There is a lack of literature on the diagnosis of TNI after TBI [9]. TNI can be difficult to assess and are frequently missed because the patient's eye position may appear normal with compensatory measures from other recti muscles [2]. TBI patients suffer from reduced sensorium, altered consciousness, agitation, cognitive deficits hence leading to under-reporting of symptoms of diplopia and delays in the diagnosis and treatment of TNI. This patient had reduced attention at the beginning of admission.

Hypertropia can occur greatest in adducted depression position of involved eye, more pronounced in unilateral TNI [9]. Other examination includes the Bielschowsky head tilt test, where there is hyperdeviation of the affected eye when the patient's head is tilted to the affected eye. This is positive in 90% of TNI patients [9]. Other clinical tests include the Parks' three step diagnostic technique [11] and tests for cyclotropia like the Moaddox double prism and Lancaster-Hess-Lees tests [9]. Another test is the vertical saccadic velocity determinations which will reveal slowing down saccades in the adduction position in TNI patients [9].

In this patient, diplopia was worsened during vestibular screening tests with Dix-Hallpike and Supine Log Roll Screening Tests. It is possible that during these tests, the patient's head position is manipulated in a manner that mimicked the Bielschowsky head tilt test, resulting in torsional movements of the eyes, thereby accentuating his TNI symptoms.

A confirmatory radiological diagnosis for TNI is thus far challenging as there are only a few cases where a brain stem lesion was detected [12]. Nevertheless, magnetic resonance imaging brain is the modality of choice for further investigation when initial CT does not indicate neurological damage for symptomatic patients with TNI.

Management and prognosis

There is a lack of consensus on management of ocular motor nerve palsies after TBI [3]. Management of traumatic isolated trochlear nerve palsy can include measures to overcome diplopia. These include the use of prisms and eye patch for symptomatic relief [2]. There is usually a 6 month period post injury where the patient's condition is observed for any spontaneous recovery, before surgical intervention is considered [2]. Steroids use in terms of dosage, duration and efficacy for traumatic TNI has not been well studied [3]. One study has evaluated the use of galantamine in the treatment of traumatic oculomotor palsies or TNI in 5 patients [13].

The trochlear nerve has the highest rate of recovery amongst the ocular motor nerves and at least 50% of patients recover [9]. Recovery time is an average of 10 weeks and ranges from 1 week to 6 months [9].

Conclusion

Isolated TNI be easily missed in TBI patients due to its subtle clinical findings. Early detection of CN palsies may positively impact patient's rehabilitation progress, functional independence and quality of life.

This case illustrates the incidental use of vestibular screening manoeuvres to provoke diplopia associated with isolated TNI, which subsequently facilitated an early referral to ophthalmology for definitive diagnosis. Vestibular screening manoeuvres are easy to perform, safe and require minimal training. There is a need for further research with regard to the utility of vestibular test manoeuvres to screen for isolated TNI post TBI, evaluation of prognostic factors and specific interventions to improve spontaneous recovery from post-traumatic TNI.

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