Neuropsychiatric and Cognitive Sequalae of Traumatic Brain Injury Among Military Personnel - A Case Series

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ABSTRACT:

Post traumatic brain injury (TBI) neuropsychiatric sequelae can be as or even more devastating than the physical deficits. It does not only interfere with day-to-day function, but may hinder rehabilitation efforts and general outcome of the patient. Despite that, neuropsychiatric and cognitive sequalae often left unattended. This case series documents 4 military personnel, referred to psychiatric clinic HAT Tuanku Mizan in Kuala Lumpur for declination of performance following motorvehicle accident (MVA) with polytrauma. All sustained severe traumatic brain injury with physical deficit. They return to work within 6-12 months. Despite evidences of neuropsychiatric symptoms early in the presentation, the detail assessment were not carried out. They were only referred much later for neuropsychiatric evaluation (1.6 – 6 years). Only case 4 was refer earlier (1 year and 7 months) as he manifested persistent psychotic symptoms. Neuropsychiatric assessment demonstrated that all 4 cases were suffering from neuropsychiatric and cognitive sequalae of TBI. Three out 4 cases were discharged from the service. Clinicians should be aware of the complexities of TBI and possibilities of neuropsychiatric and cognitive sequalae following head injury in order to deliver effective and early intervention for better outcome.

KEYWORDS: Traumatic Brain Injury, Malaysia Armed Forces, Military, Neuropsychiatric

BACKGROUND

Traumatic brain injury (TBI) is defined as physiological disruption of the brain function induced by trauma, indicated by at least one of the following clinical signs - any period of loss of or a decreased level of consciousness (LOC); any loss of memory for events immediately before or after the injury (post-traumatic amnesia); any alteration of consciousness level/mental state (confusion, disorientation, slowed thinking, etc.); neurological deficits (weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia, etc.); or intracranial lesion [1-3].

Previously, TBI are commonly seen in war injury. However, in this peace era, motorvehicle accidents (MVA) accounts for the most cause of TBI among Malaysian Armed Forces (MAF) personnel. Many of these patients suffer from polytrauma especially muskuloskeletal injuries. More than half of TBI cases had other associated trauma [4]. Although most patients recovered from a mild TBI, 10-30% of people will experience prolonged chiatric and cognitive sequelae [5]. This include neuropsychiatric disorders, personality changes and neurocognitive disorders. Often left unattended, post TBI neuropsychiatric and cognitive sequelae can be as or even more devastating than the physical deficits. It does not only interfere with day-to-day function, but can severely hinder rehabilitation efforts and general outcome of the patient.

CASE PRESENTATION

This case series involved 4 male personnel with severe TBI. Clinical history of each patient were obtained from medical record (Bat F4), neuropsychiatric assessment report, medical report from previous hospitals and clinical notes from each visit. All the patient returned to work after recovery from physical deficit. Poor performance at workplace and behaviour changes subjected them for psychiatric referral. The referral was considerably late, ranged from 19 months to 5 years.

a. Patient characteristic

All 4 patient are male military personnel, age ranged from 24 to 50 years old. 3 out of 4 were young and healthy personnel whereas patient 2 has comorbid diabetes and hypertension.

Case 1 is a 34-year-old Malay military officer with high premorbid functioning. He had an alleged MVA on 16th April 2015 in which he sustained multiple skull fractures with right facial nerve injury with lower motor neuron lesion, left temporo-parietal-occipital subarachnoid haemorrhage and left parietal subdural haemorrhage. He was discharged after 14 days of admission.

Case 2 - 56 years old, Indian, air force officer, alleged MVA on 17th May 2010. He sustained bilateral cerebral frontal contusion, cerebral oedema, left frontal bone fracture, multiple base of skull and facial bone fracture with left facial nerve injury, lung contusion and open fracture of left Olecranon (grade I). He was mechanically ventilated and admitted for 1 days. Upon discharge he was noted to have headache, dizziness, easily irritable and memory impairment.

Case 3 - 28 years old navy personnel, alleged MVA on 5th August 2012. He sustained subarachnoid bleed at left high vertex and prepontine cistern,small contusion bleed at left thalamic region, left eye trauma with left hemiparesis. He was intubated and underwent tracheostomy due to prolonged mechanical ventilation. He was discharged from hospital after 28 days.

Case 4 is 27 year-old army personnel, a clerk. He was alleged involved in MVA in Jan 2016 in which he sustained left temporal contusional bleed and base of skull fracture, closed fracture of midshaft right femur, closed fracture of right radius and ulnar. He arrived to hospital with GCS of 8/15 and subsequently intubated. He regained consciousness after 4 days in ICU. He was discharged home after 12 days.

Table 1 : Patient Characteristic and Injuries.

	Age (year of trauma)	Sex	Military rank	Date of trauma	Details of TBI	Details of other trauma
Patient 1	32	Male	Officer	16/4/15	Multiple intracranial bleed - Left temporo-parietal-occipital subarachnoid hemorraghe, left parietal subdural hemorrhage Intrparenchymal bleed in th left parieto-occipital region	Multiple facial bone fracture Right facial nerve injury with lower motor neuron lesion
Patient 2	50	Male	Officer	17/5/2010	Bilateral cerebral frontal contusion, cerebral oedema,	Multiple base of skull, left frontal bone fracture and facial bone fracture Left facial nerve neuropathy. Lung contusion Left olecranon fracture – complicated with left elbow deformity
Patient 3	24	Male	NCO	5/8/12	Subarachnoid bleed at left high vertex and prepontine cistern Small contusion bleed at left thalamic region	Left eye trauma
Patient 4	25	Male	NCO	26/4/2016	Bifrontal and Left temporal contusional bleed and base of skull fracture	Closed fracture of midshaft right femur Closed fracture of right radius/ulnar

b. Severity of TBI

All 4 patients sustained severe TBI; all have duration of LOC more than 24 hours with GCS less than 10, PTA more than 1 week and abnormal structural imaging. The details are summarized in Table 2. TBI was classified into mild, moderate and severe based on Glasgow Coma Scale (GCS) in first 24 hours of presentation, period of loss of consciousness (LOC), presence of post-traumatic amnesia (PTA) as well as abnormality of structural imaging. This classification is based on Guideline Management of TBI as shown in Table 3 [3].

Table 2 : Severity of TBI

	GCS	LOC	PTA	Structural imaging
Patient 1	7/15	7 days	2 years	Abnormal
Patient 2	9/15	3 days	1 year	Abnormal
Patient 3	8/15	10 days	2 years	Abnormal
Patient 4	8/15	4 days	19 months	Abnormal

Table 3: Classification of TBI severity - based on Clinical Practice Guideline For Management Of Concussion/Mild Traumatic Brain Injury (2009)

Criteria	Mild	Moderate	Severe
Structural imaging	Normal	Normal or abnormal	Normal or abnormal
Loss of Consciousness (LOC)	0–30 min	> 30 min and < 24 hrs	> 24 hrs
Alteration of consciousness/mental state (AOC)	a moment up to 24 hrs	> 24 hours. Severity based on other criteria	
Post-traumatic amnesia (PTA)	0-1 day	> 1 and < 7 days	> 7 days
Glasgow Coma Scale (best available score in first 24 hours)	13-15	9-12	< 9

c. Physical deficit recovery, neuropsychiatric and cognitive sequalae and employability

All 4 patient showed tremendous physical recovery and returned to work within 12 months. However all could not perform as they used. Subsequently they were referred for neuropsychiatric assessment. All 4 patients were diagnosed as personality changes and neurocognitive disorders due to severe TBI. Only patient 2 has comorbid MDD. 3 of them totally recovered from physical deficit of TBI and other physical injuries. The other 1 has fixed deformities of left elbow due to olecranon fracture. 3 out 4 patient were discharge from military service. The other 1 were downgraded to BE (Employable but unfit for deployment to the field). The details are shown in Table 4.

Table 4 : Physical Deficit, Neuropsychiatric and Cognitive Sequalae of TBI and Employability

	Neuropsychiatric and cognitive deficit		RTW (months)	Physical deficit	Employability
	Neurocognitive disorder	Personality changes			
Patient 1	Memory impairment	Yes	6	Mild facial nerve paresis	Discharge from military service
Patient 2	Memory impairment	Yes	8	Fixed deformity of left elbow Headache, lethargy, poor sleep	Initially downgraded to BE due to physical deficit, discharge from military service after 5 years
Patient 3	Memory impairment	Yes	12	Fully recovered	Downgraded to BE (Employable but unfit for deployment to the field)
Patient 4	Memory impairment	Yes	9	Fully recovered	Discharge from military service

RTW : Return to work; BE : Base Everywhere; employable in the base area only in any part of Malaysia

Patient 1 returned to work at 6 months post trauma. Except of mild residual facial nerve palsy, he was fully recovered from all physical deficit. Poor performance at work made him subjected for further psychiatric evaluation. Neuropsychological assessment revealed memory impairment in all memory indexes except the working memory. He displayed impulsivity (snap decision and poor judgement), irritability and awareness deficit. Brain MRI (2 years post injury) showed mild but significant global cerebral atrophy with focal parenchymal changes at left medial temporal and right medial temporal with bilateral hippocampi atrophy. He sustained severe to very severe traumatic brain injury with personality changes and cognitive impairment especially memory domain and subsequently discharged from military service.

Patient 2 returned to work 8 months post trauma. Apart from fixed deformity of left elbow he was physically recovered. He was noted to have changed from his premorbid personality. He became easily irritable, verbally abusive with hypersexuality and emotional lability. On top of that, he also has persistent depressive symptoms suggestive of major depressive disorder which is most likely secondary to disability caused by the accident (physical and cognitive). He was initially downgraded to BE due to his physical disabilities and subsequently discharged from the service.

Patient 3 - He completed physical rehabilitation program and returned to work after 12 months. Despite prominent behavior changes, he was medically graded as FE (Forward Everywhere) and was sent for deployment. His was totally changed in which he became more talkative, joyful, and more confidence with inflated self-esteem, impulsive behaviour with poor judgement. At times, he portrayed himself as someone who is very capable and talented. However, inability to follow deployment and operational rules and poor work performance brought him to psychiatric evaluation only after 3 years and 7 months post trauma. He was subsequently downgraded to BE.

Patient 4 - He returned to work after 9 months of medical leave after recovered from his physical deficits. He could not perform as he used to. He was noted to be forgetful, easily distracted and difficulty to recall certain information. He displayed impulsivity, irritability and disinhibition. He was referred for psychiatric evaluation as he manifested inability to follow military command, and had few episodes of outburst anger with aggressive tendencies. His poor recovery subjected him for discharge from military service.

DISCUSSION

TBI can cause not only focal physical deficits but also a variety of potentially disabling neuropsychiatric and cognitive disorders. These include personality changes, secondary psychiatric disorders and cognitive impairment. The severity neurocognitive sequalae of TBI depends on the degree of diffuse axonal injury as well as the volume and location of focal injuries [6]. While majority of patient have recovered, up to 15% of patients with mild TBI, the neuropsychiatric and cognitive symptom persist beyond 3 months and contribute to long-term social and occupational difficulties [7,8].

Personality changes

Post TBI changes in personality may manifested in two ways either exaggeration of pre-injury traits, or ultimate changes. This include impulsivity, irritability, lability (instability) and apathy Impulsivity may be manifest in the form of verbal utterances, physical actions, snap decisions, and poor judgment; irritability means the response is characteristically out of proportion to the triggering stimulus, range from verbal outbursts to dangerous aggressive and assaultive behavior; instability is exaggeration displays of emotional expression (sadness, angry, happiness) or also known as affective lability, pseudobulbar affect or affective incontinence; apathy is referring to lack of interest or concern, can occur in association with injury to brain area involved in reward circuit [9,10]. Although not as overtly disturbing as some of the other changes, apathy is frequently associated with poor progress in rehabilitation programs. It is also often misinterpreted as laziness.[11]

Cognitive deficit

Initial and persistent cognitive deficits are the most common complaints after TBI. [12,13] These include frontal executive functions (problem solving, impulse control) attention, short-term memory, learning, speech and language functions. Commonly, several cognitive domains are affected, typically at varying level of impairment.

TBI and psychiatric disorders

In addition to the cognitive deficit and personality changes, TBI has been associated with increased risk of developing psychiatric disorders [14-17]. Even though, not all of these symptoms will fulfil the criteria of a disorder but it may sufficient to interfere with social or occupational function.

CONCLUSIONS

TBI commonly associated with polytrauma. TBI may not only lead to focal physical deficits but also a variety of potentially disabling neuropsychiatric or cognitive disorders which commonly left unattended. Consideration to the diagnosis and management of the neuropsychiatric and cognitive sequelae of TBI is critical in advancing the rehabilitative process. Furthermore, neuropsychiatric and cognitive symptoms appear to have an important role in determining long-term outcomes, particularly related to employability [18,19].

LEARNING POINTS

1. The initial steps of TBI treatment include a comprehensive neuropsychiatric and cognitive evaluation and testing in order to ascertain the diagnosis, document the deficits and baseline functioning and planning for cognitive rehabilitation.
2. Neuropsychiatric and cognitive sequalae of TBI have an important role in determining long-term outcomes, particularly related to employability.
3. Lack of awareness among clinicians in regards to cognitive and emotional concerns of the patient with TBI leads to late assessment and intervention hence poor outcome.

REFERENCES

1. Booth-Kewley S, Highfill-McRoy RM, Larson GE, Garland CF, Gaskin TA. Anxiety And Depression In Marines Sent To War In Iraq And Afghanistan. J Nerv Ment .2012 : 200(9):749–757.
2. Brenner LA, Terrio H, Homaifar BY, et al. Neuropsychological test performance in soldiers with blast-related mild TBI. Neuropsychology. 2010: 24(2):160–167.
3. Appendix C- Definition Of MTBI From The Va/Dod Clinical Practice Guideline For Management Of Concussion/ Mild Traumatic Brain Injury (2009)
4. Groswasser Z1, Cohen M, Blankstein E. Polytrauma associated with traumatic brain injury: incidence, nature and impact on rehabilitation outcome. Brain Inj. 1990;4(2):161- doi: 10.3109/02699059009026161. PMID: 2331545.
5. Silvana R. Traumatic Brain Injury and Its Neurobehavioral Sequelae. PlumX Metrics. Volume 29, Issue 1, Pages 35–47. DOI: https://doi.org/10.1016/j.ncl.2010.10.008
6. Liu B, Tian Y, Zhang Y, Zhang W: Therapeutic effect analysis of acute traumatic brain injuries. Neurol Res. 2008;30:594–5972008
7. Scheid R, Walther K, Guthke T, Preul C, von Cramon D Y. Cognitive sequelae of diffuse axonal injury. Arch Neurol. 2006: 63(3) 418-424
8. Lundin, A., de Boussard, C., Edman, G. et al. Symptoms and disability until 3 months after mild TBI. Brain Inj. 2006: 20: 799–806
9. McAllister TW. Neurobehavioral sequelae of traumatic brain injury: evaluation and management. World Psychiatry. 2008: 7(1):3-10. doi:10.1002/j.2051-5545.2008.tb00139.x
10. Arciniegas DB, Lauterbach EC, Anderson K. The differential diagnosis of pseudobulbar affect (PBA): distinguishing PBA from disorders of mood and affect. CNS Spectr. 2005: 10:1–14.
11. McAllister TW. Apathy. Semin Clin Neuropsychiatry. 2000: 5:275–282.
12. Lovell M, Franzen M, Silver JM, Yudofsky S, Hales RE. Neuropsychiatry of traumatic brain injury. Washington: American Psychiatric Press. 1994:133–160.
13. Whyte J, Polansky M, Cavallucci C. Inattentive behavior after traumatic brain injury. J Int Neuropsychol Soc. 1996: 2:274–281.
14. Deb S, Lyons I, Koutzoukis C. Neuropsychiatric sequelae one year after a minor head injury. J Neurol Neurosurg Psychiatry. 1998:65:899–902.
15. Hibbard MR, Uysal S, Kepler K. Axis I psychopathology in individuals with traumatic brain injury. J Head Trauma Rehabil. 1998:13:24–39.
16. van Reekum R, Cohen T, Wong J. Can traumatic brain injury cause psychiatric disorders? J Neuropsychiatry Clin Neurosci. 2000:12:316–327.
17. Koponen S, Taiminen T, Portin R. Axis I and II psychiatric disorders after traumatic brain injury: a 30-year follow-up study. Am J Psychiatry. 2002:159:1315–1321.
18. Lippert-Gruener M, Wedekind Ch, Klug N. Functional and psy-chosocial outcome one year after brain injury and early onset rehabilitation therapy. J Rehabil Med. 2002:34:211–214.3.
19. Warriner EM, Velikonja D. Psychiatric disturbances after trau-matic brain injury. Neurobehav Pers Changes Curr Psychiatry Rep.2006: 8:73–80

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