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Mortality rates of severe traumatic brain injury patients: impact of direct *versus* nondirect transfers

Kartik Prabhakaran, MD, MHS, FACS,[a,*] *Patrizio Petrone, MD, MPH, FACS,*[b] *Gary Lombardo, MD, FACS,*[a] *Christy Stoller, MD,*[a] *Anthony Policastro, MD, FACS,*[a] *and Corrado P. Marini, MD, FACS*[b]

[a] *Division of Trauma Surgery, Surgical Critical Care and Emergency General Surgery, Department of Surgery New York Medical College, Westchester Medical Center University Hospital, Valhalla, New York*

[b] *Division of Trauma Surgery, Surgical Critical Care and Emergency General Surgery, Winthrop University Hospital, Mineola, New York*

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ABSTRACT

Background: Direct transport of patients with severe traumatic brain injury (sTBI) to trauma centers (TCs) that can provide definitive care results in lower mortality rates. This study investigated the impact of direct *versus* nondirect transfers on the mortality rates of patients with sTBI.

Methods: Data on patients with TBI admitted between January 1, 2012, and December 31, 2013, to our Level I TC were obtained from the trauma registry. Data included patient age, sex, mechanism, and type of injury, comorbidities, Glasgow Coma Scale, Injury Severity scores, prehospital time, time to request and to transfer, time to initiation of multimodality monitoring and goal-directed therapy protocol, dwell time in the emergency department (EDT), and mortality. Data, reported in means ± standard deviation, were analyzed with the Student *t*-test and chi-square. Statistical significance was accepted at a P value < 0.05.

Results: sTBI direct transfer to TC *versus* transfer from non-TCs (NTC): Of the 1187 patients with TBI admitted to our TC, 768 (64.7%) were admitted directly from the scene, whereas 419 (35.3%) were admitted after secondary transfer. One hundred seventy-one (22.2%) of the direct transfers had Glasgow Coma Scale < 8 (sTBI) and 92 (21.9%) of the secondary transfers had sTBI. The transfer time: Time from scene to arrival to the EDT was significantly shorter for TC *versus* NTCs 43 ± 14 *versus* 77 ± 26 min, respectively ($P < 0.05$). EDT dwell time before transfer and time from injury to arrival to TC were 4.2 ± 2.1 and 6.2 ± 8.3 h, respectively. Mortality: There was a statistically significant lower mortality for patients with sTBI transferred directly from the scene to TCs as opposed to patients secondarily transferred, 33/171 (19.3%) *versus* 33/92 (35.8%), respectively ($P < 0.05$).

Conclusions: To decrease TBI-related mortality, patients with suspected sTBI should be taken directly to a Level I or II TC unless they require life-saving stabilization at NTCs.

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* *Corresponding author*. Division of Trauma Surgery, Surgical Critical Care and Emergency General Surgery, Department of Surgery New York Medical College, Westchester Medical Center University Hospital, E150 Taylor Pavilion, 100 Woods Road, Valhalla, NY 10595. Tel.: +1 914 493-6826; fax: +1 914 493-5271.

E-mail address: Kartik.Prabhakaran@wmchealth.org (K. Prabhakaran).

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Introduction

Traumatic brain injury (TBI) represents a disease associated with a high degree of morbidity and mortality and constitutes a significant public health burden when taking into account resultant disability.[1,2] Particularly for patients with severe TBI (sTBI), evidence-based and timely delivery of care in accordance with established recommendations, such as the Brain Trauma Foundation guidelines, can reduce both mortality and the economic burden associated with major disability.[3] In addition to timely neurosurgical intervention when indicated, patients with sTBI are conventionally treated using the Brain Trauma Foundation guidelines for monitoring and maintaining cerebral perfusion pressure (CPP), intracranial pressure (ICP) and more recently, brain oxygen tension ($PbtO_2$).[4] In view of more recent evidence that ICP–CPP targeted therapy may be unable to prevent the development of secondary brain injury because of the inability to assure the brain oxygen tension necessary to prevent the molecular processes responsible for the extension of the injury beyond the impacted area of the brain, a few centers, including our own have replaced the ICC–CPP targeted therapy with one that includes monitoring of $PbtO_2$, bi-frontal brain oxygen saturation by near-infrared spectroscopy (NIRS) and a goal-directed therapy targeted to maintenance of brain oxygen tension and a normal lactate/pyruvate ratio (LPR) measured from the effluent of the cerebral microdialysis (CMD).[5,6]

The vast majority of patients with TBI seen in emergency departments (EDTs) are discharged without intervention or further treatment.[7] However, for patients with moderate (Glasgow Coma Scale [GCS] 9-12) and severe (GCS $\leq$ 8) TBI, avoidance of secondary brain injury and protocol-driven timely delivery of care is essential to decrease morbidity and mortality and poor functional outcome.[8] Such care is usually predicated on the availability of an interdisciplinary approach and institutional resources typically present in Level I and Level II trauma centers (TCs). For these reasons, current guidelines suggest direct transport of patients with TBI to facilities that offer neurosurgical expertise in the form of timely computed tomography scans, ICP monitoring, availability of a neurosurgeon, and intensive care unit admission.[9]

Earlier studies have suggested that the direct transport of sTBI patients to Level I or Level II TCs can significantly reduce mortality in patients with sTBI.[10] However, subsequent studies have suggested that patients with sTBI transferred to a Level I or II TC were less likely to die when compared with sTBI patients directly admitted to a Level I or II TC, and that consequently sTBI patients should be stabilized at a non-TC (NTC) before transfer when applicable.[11]

Given the regionalization of trauma resources as well as the importance of timely neurosurgical care, we sought to examine the impact of direct transfer (i.e., scene to Level I TC) *versus* indirect transfer (scene to NTC to Level I TC) on the mortality of patients with both nonsevere TBI and sTBI. We further sought to examine the relationship between direct *versus* indirect transfer on time to arrival at a Level I TC and time to initiation of multimodality neurophysiological monitoring and a goal-directed therapy protocol (GDTP).

Methods

Data on all patients with TBI admitted to Westchester Medical Center, a Level I TC from January 1, 2012, to December 31, 2013, were obtained from the trauma registry. Patients were evaluated as part of an observational prospective database with Institutional Review Board approval. Data collected included patient age, sex, mechanism and type of injury, comorbidities, GCS, Injury Severity Scores (ISS), time to request for transfer, time to arrival after transfer (from scene or referring facility), dwell time in the EDT, time to initiation of multimodality monitoring and goal-directed therapy protocol (MM&GDTP), craniotomy *versus* no craniotomy, and mortality. Patients were stratified according to GCS >8 (non-STBI) and GCS $\leq$ 8 (sTBI).

All patients with TBI received a neurosurgical consultation and were admitted to either the Trauma or Neurosurgical service. All patients with sTBI were admitted to the Trauma Intensive Care Unit and underwent between 3 and 5 d of MM&GDTP (Fig. 1) with the exception of patients with brain injuries deemed to be nonsurvivable where surrogates elected to pursue withdrawal of care. Multimodality monitoring (MM) included ICP, CPP, $PbtO_2$ (Licox; Integra Life Sciences), bi-frontal brain oxygen saturation by NIRS (Covidien), and CMD (M Dialysis, Stockholm, Sweden). CMD was performed via a dual lumen catheter inserted in juxtaposition to the brain oxygen catheter. The CMD catheter was perfused at a rate of 0.3 μL/min and the effluent was analyzed every hour. Samples were analyzed for glucose (normal values 14.4-46.8 mmol/L), lactate (normal values 2.0-3.8 mmol/L), pyruvic acid (normal values 119-213 μmol/L), glutamate (normal values 0.0-32.0 μmol/L), glycerol (normal values 38.0-126 μmol/L), and LPR. All intracranial monitors were inserted within 2-4 h of diagnosis of sTBI except for patients who went directly to the operating room for evacuation of mass lesions who had monitors placed after the craniotomy.

The GDTP included maintenance of ICP $\leq$20 mm Hg, CPP $\geq$60 mm Hg, $PbtO_2$ $\geq$20 mm Hg, and NIRS $\geq$55%, CMD LPR $\leq$25 with a normal glucose and pyruvate level. Normothermia (37°C) was maintained with dry water immersion technique using Arctic Sun (Company name and address). Post-pyloric peptide-based enteral nutrition was started on completion of the resuscitation phase. All patients were sedated to synchrony with the ventilator, avoidance of cough, and a modified Ramsey score of two with a combination of midazolam infusion and/or propofol. Osmotherapy for elevated ICP included 3% saline and the addition of mannitol when appropriate. Burst suppression was initiated when ICP, low $PbtO_2$, or low NIRS were not responsive to therapy. Burst suppression was achieved with an infusion of midazolam at a dose up to 15 mg/h and/or propofol at a dose up to 100 μg/kg/min; infusions were titrated to 2-4 burst per screen while using continuous electroencephalogram. Decompressive hemicraniectomy was performed in patients with surgical lesions causing mass effect and increased ICP. Predicted mortality (PM) was calculated based on the CRASH model (Corticosteroid Randomization after Significant Head Injury).[12]

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