Variability between Expected and Actual Outcome Following a Severe Traumatic Brain Injury

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Abstract

Traumatic brain injury remains a challenging and complicated disease process to care for, despite the advance of technology used to monitor and guide treatment. Currently the mainstay of treatment is aimed at limiting secondary brain injury, with the help of multiple specialties in a critical care setting. Prognosis after TBI is often even more challenging than the treatment itself, although there are various exam and imaging findings that are associated with poor outcome. These findings are important because they can be used to guide families and loved ones when making decisions about goals of care. However in this case report, we demonstrate the unanticipated recovery of a patient with a severe traumatic brain injury who presented with several exam and imaging findings that are statistically associated with increased mortality and morbidity.

ABBREVIATIONS

TBI: Traumatic Brain Injury; ADLs: Activities of Daily Living; GCS: Glasgow Coma Scale; GOS: Glasgow Outcome Scale; CT: Computed Tomography; ICP: Intracranial Pressure; CSP: Cerebral Spinal Fluid; EVD: External Ventricular Drain; CPP: Cerebral Perfusion Pressure; HD: Hospital Day; FIO2: Fraction of Inspired Oxygen; PEEP: Positive End Expiratory Pressure; ARPV: Airway Pressure Release Ventilation; CMV: Continuous Mandatory Ventilation; CPAP: Continuous Positive Airway Pressure; IV: Intravenous

INTRODUCTION

In the United State alone, there are approximately 1.5 million traumatic brain injuries (TBI) per year, and TBI is the leading cause of death among individuals under the age of 45 [1,2]. Annually, these injuries result in 50,000 deaths and 80,000-90,000 cases of debilitating head injuries [2]. In the US, the estimated annual economic cost of TBIs is $76.5 billion, and we must not forget the emotional and physical toll that disability inflicts on patients and their families [3]. In many cases, patients are left without the ability to work, or perform activities of daily living (ADLs) [4]. Initial management of TBI is the most critical time period, because it will have the greatest effect on mortality and degree of debility that surviving patients will experience.

In TBI, the most important tool used to assess degree of brain injury and prognosis is exam findings. According to the guideline Early Indicators of Prognosis in Severe Traumatic Brain Injury, on average, 88% of patients who presented with bilaterally unreactive pupils became vegetative or died, and 4% had good recovery or moderate disability [5]. Glasgow Coma Scale (GCS) uses exam findings to quantify level of consciousness following TBI, with 3 being the worst defined as deep coma or death, and 15 being the best, a fully awake person. In a study by Fearnside et al, out of 315 patients with severe TBI, 65% with initial GCS of 3 died [6]. In a larger study by Marshall et al. out of 746 patients, 78.4% with initial GCS of 3 died and 7.2% had mild to moderate disability [7].

Radiographic findings can also be used to predict morbidity and mortality, and can be used to guide surgical intervention. A review of 753 Computed tomography (CT) studies that revealed abnormal mesencephalic cisterns, midline shift, and subarachnoid hemorrhage were associated with an increased risk of elevated intracranial pressure (ICP) and death [8]. Here we describe a patient who had all of the above CT findings, and who presented with a GCS of 3 and bilaterally dilated and fixed pupils.

CASE PRESENTATION

Bystanders found a 28-year-old, unhelmeted, Caucasian male prone and unconscious after he had lost control of his motorcycle and went off the road. He was brought to the Emergency Department via ambulance intubated as Level 1 Trauma activation where his physical examination revealed a GCS of
3T, 4 mm bilaterally fixed pupils, negative corneal response, right parietal cephalohematoma, and cerebral spinal fluid (CSF) otorrhea on the right. CT of the head showed subarachnoid hemorrhage with left frontal and temporal subdural hemorrhage (Figure 1), effacement of the supracellar cistern (Figure 2), and effacement of the 3rd ventricle, and 4th ventricle (Figure 3). In addition, CT studies showed a left frontal/temporal and parietal hematoma with mass effect and cerebral edema causing 5.38 mm left to right midline shift (Figure 4), a frontal skull base fracture, and a complex non-displaced comminuted fracture of the right temporal bone. He was bradycardic, with his lowest heart rate recorded at 28 bpm and hypertensive with an initial blood pressure of 172/118 mmHg and markedly elevated blood pressure of 221/118 mmHg 30 minutes after his arrival to our facility. He required Atropine push and Nicardipine infusion. An arterial line and central venous catheters were placed for fluid and medication administration. Emergent treatment for herniation syndrome included endotracheal intubation, 30 grams of IV Mannitol, hypertonic solution of 23% (weight/volume) sodium chloride (NaCl), and left sided decompressive craniectomy. Post operatively, an external ventricular drain (EVD) was placed; the initial intracranial pressure (ICP) was 14 mmHg. The patient was examined post operatively and also after EVD placement. His GCS was 5T with bilaterally reactive pupils, and positive corneal reflex in the left eye. CT of his head showed improvement of midline shift (Figure 5) and the ventriculostomy catheter tip to be in the proper location in frontal horn of the right lateral ventricle (Figure 6). The patient was then started on 3% NaCl continuous infusion. ICP and cerebral perfusion pressure (CPP) displayed normal values between 3-4 and 70-75 mm Hg, respectively, for the first 24 hours with the EVD open at 10 cm H2O.

On hospital day (HD) 2 the patient’s ICP was elevated in the upper 20s in the setting of shivering, hypertension, and fever, which were controlled with increased sedation, Nicardipine infusion, and targeted temperature management, respectively. Repeat CT revealed cerebral edema in evolution, with no worsening midline shift or hemorrhage. The sub-galeal drain was removed.

On HD 3 his GCS improved from 6T to 8T with intact brain stem reflexes. The next day, HD 4, the patient’s oxygen saturation dropped from 96% to 87% requiring an increase in fraction of inspired oxygen (FIO2) from 50% to 80% and positive end expiratory pressure (PEEP). Respiratory Cultures were obtained and a chest x-ray revealed worsening bibasilar opacities (Figure 7) therefore he was empirically started on IV Vancomycin.

On HD 5, the patient’s GCS deteriorated from 8T to 3T in the setting of elevated ICP. During bronchoscopy his ICP was noted to be markedly elevated at 46 therefore 23% NaCl was administered intravenously over ten minutes. Subsequently, a repeat CT of the head was obtained that showed cerebral edema in evolution, no change in midline shift or hemorrhage.
Respiratory cultures resulted on HD 6 and revealed methicillin-sensitive *staphylococcus aureus* (>10,000 cfu/ml) and *pseudomonas aeruginosa* (>10,000cfu/ml). Repeat chest x-ray showed worsening infiltrates and bilateral pleural effusions (Figure 8). The antibiotic was switched from Vancomycin to IV Levofloxacin IV Cefepime.

Examination on HD 8 revealed an improved GCS of 6T and his ICP was controlled during an EVD clamp trial, and the EVD was then removed. There were also copious amounts of thick, tan endotracheal secretions therefore another bronchoscopy was performed using saline lavage. The patient was started on IV Zosyn and Cefepime was discontinued. ICP was controlled by administering 23% NaCl. During a sedation vacation, his oxygen saturation decreased from 95% to 70% and the ventilator settings were therefore changed to airway pressure release ventilation (APRV) mode.

On HD 14, his oxygenation was improving and remained stable at 97% . The ventilator mode was weaned from APRV to continuous mandatory ventilation (CMV). Patient was taken off sedation. His GCS was 10T and he was started on Amantadine. He was started on oral metronidazole due to multiple loose stools, which was positive for *clostridium difficile* toxin B.

On HD 15, he began blinking to threat, with a GCS of 10T. CT of his head showed improvement in diffuse cerebral edema and effacement of basal cisterns (Figure 9). He underwent a percutaneous tracheostomy with video assisted bronchoscopy.
Figure 8 Chest x-ray showing bilateral interstitial infiltrate and left base atelectasis and pleural effusion.

Figure 9 Chest x-ray showing worsening interstitial and alveolar infiltrates and pleural effusions bilaterally.

Figure 10 New left occipital horn intraventricular hemorrhage, increased external herniation of left frontal contusion, resolving diffuse cerebral edema with decreased effacement of third ventricle.

and open gastrostomy tube placement by trauma surgery. The patient remained neurologically unchanged on HD 16, but repeat chest x-ray revealed an interval increase in right sided infiltrates with resolution of left lung opacity. On HD 17, he continued to maintain adequate oxygen saturation at 97% and was started on continuous positive airway pressure (CPAP).

On HD 18 the patient developed tachypnea and he was placed back on CMV. The antibiotics were changed from Zosyn to Levofoxacin and nebulized Tobramycin due to Piperacillin/Tazobactam resistant pseudomonas pneumonia. On HD 19 his neurologic status was unchanged and was started on Provigil. He was also given a protective helmet. Two days later, on HD 21, the patient’s neurologic status remained unchanged and he was discharged to a long term care facility.

Follow up visit three months later revealed the patient was living at home with his mother. In the interim his tracheostomy and gastrostomy tube had been removed. His major neurologic sequelae were transcortical motor aphasia and mood disorder. His GCS was 13 (E4, V3, M6). His GOS, on a 5 point scale, was 3 with severe injury and permanent need for help with daily living. His Modified Rankin Scale was 3 with moderate disability, requiring some help, but able to walk without assistance. His Lawton Instrumental Activities of Daily Living Scale was 4/8. Barthel Index was 95/100, and National Institutes of Health Stroke Scale was 5. Eleven months after the accident he had similar outcome scores and had developed a seizure disorder, however his speech was markedly improved with speech therapy. A repeat CT of the head showed improved external herniation (Figure 10).

DISCUSSION

When predicting mortality and unfavorable outcome following TBI, exam, laboratory, and imaging findings can be used together by utilizing the CRASH and IMPACT calculators [9,10]. Unfavorable outcome is described as death, vegetative state, or severe disability. Here we describe a patient who presented with a GCS of 3, bilaterally fixed pupils, and CT findings of subarachnoid bleeding, midline shift, subdural hematoma, effaced 3rd ventricle,
effaced 4th ventricle, and effaced basal cisterns. Therefore according to the CRASH calculator, which takes into account country, age, GCS, pupil reactivity, and CT findings, he had a 14-day mortality risk of 91.8% and a 95.7% chance of unfavorable outcome at six months [9]. His pertinent laboratory studies, which are utilized along with exam and imaging findings in the IMPACT calculator, revealed an initial glucose concentration of 260 mg/dL and a hemoglobin concentration of 15.4 g/dL. Using the IMPACT calculator, at six months, the patient’s predicted probability of mortality was 62% and the probably of unfavorable outcome was 77%. He left our facility bedbound, ventilator and tube feed dependent, and in a minimally conscious state with a GCS 10 T. Yet despite all this, he had a favorable recovery. Within one year of discharge he was able to live at home, interact and go shopping with his mother, walk, feed himself, and perform simple chores and ADLs. This is a poignant reminder that variability between individual patients makes prognosticating after traumatic brain injury difficult and uncertain.

Our case shows that severe caution should be taken when using prior studies to make medical decisions about individual patients. Treatment of traumatic brain injuries is complex, and should continue to evolve with evidence-based medicine. Improvement in outcome is not based on one intervention; rather it is the additive effect of multiple interventions. Our patient had rapid access to 23% NaCl, craniectomy, and ventriculostomy placement with intracranial pressure and cerebral perfusion pressure monitoring that guided management in a stepwise approach, allowing us to limit secondary brain injury to the best of our ability with the tools we had. It is possible that the addition of multimodality monitoring could have further changed his outcome, and more studies need to be done to answer this. In addition, daily multidisciplinary rounds with Neurocritical Care, Trauma Critical Care, Infectious Disease, Pharmacy, Respiratory Therapy, Physical Therapy, Occupational Therapy, Social Services, Chaplain Services and Dietary Services provided optimal medical management in a team based approach. Further studies need to be conducted to explore the effect that daily multidisciplinary rounds have on the outcome of severe TBIs.

REFERENCES