Survival after cerebral herniation

To The Editor: I would like to thank Dr. Stiver and colleagues for their recent case report (Stiver SI, Gean AD, Manley GT: Survival with good outcome after cerebral herniation and Duret hemorrhage caused by traumatic brain injury. Case report. J Neurosurg 110:1242–1246, June, 2009). While I am in agreement that the patient received extraordinary care and excellent management in the hospital, I would like to address several key points in the early management and care of this patient with traumatic brain injury (TBI).

It is noted that the patient’s initial Glasgow Coma Scale (GCS) score was M1 E1 V1 at the time of her TBI, but upon presentation to the emergency department, the patient’s score improved to M4 E1 V1. This does not describe a “hopeless situation” as indicated. It is well known that the best predictive value when dichotomizing the GCS is the motor response component, in this case a 4. In addition, it is important to mention that the patient’s GCS score was possibly compromised by the presence of an important secondary brain insult, such as hypotension related to the left humeral fracture. It is very unlikely that the intracranial lesion can be the source of such severe secondary brain insult in the absence of ongoing scalp hemorrhage.

Additionally, the airway management provided prior to admission at the receiving hospital warrants discussion. Further information regarding her ventilation and respiratory condition in the field, during transport, and on arrival to the receiving hospital would illustrate if hypoxia contributed to the patient’s initially lower GCS score. This is an important factor as the combination of hypoxia and hypotension can increase the mortality rate by 70% after TBI, and may contribute to increased intracranial pressure, cerebral edema, and herniation syndrome. It may be possible with early interventions to control and prevent these insults. Early airway management and intubation in the field, control of the hemorrhage, stabilizing the fracture, and intravenous fluid administration provided by emergency personnel could have contributed to a reduction of secondary brain insults during the so-called “golden hour.” Perhaps this sequence of events reduced the risk for uncontrollable cerebral edema that can lead to herniation syndrome.

The centered ventral pons location of the secondary Duret hemorrhage without involvement of the pontine and medullary reticular formation in the dorsal pontine tegmentum was definitely an important factor in promoting this patient’s survival. I thank Drs. Stiver, Gean, and Manley for such descriptive information about the brainstem displacement during herniation syndrome in patients with TBI.

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References


Response: We thank Dr. Defillo for his comments and critique and for the kind remarks on the excellent care provided to this patient at our hospital. As pointed out by Dr. Defillo, it is important to remember that the GCS score of record should be obtained after resuscitation. Many patients with TBI, including those with a mild head injury, incur a transient episode of unresponsiveness at the time of impact. Secondary insults such as hypoxia and hypotension do suppress the GCS score and may lead one to overestimate the severity of the primary impact. Dr. Defillo raises 2 important questions. First, the patient’s GCS score on arrival to the emergency department was 6. He notes that this is not a “hopeless situation,” and questions the attention drawn to her survival. The second question he asks is whether continued resuscitation efforts would have allowed her GCS score to improve even further. Both points suggest that perhaps this was not a devastating injury, but rather was compounded by sec-
ondary injury, and that the patient’s eventual good recovery was not as remarkable as we have indicated.

Details of the patient’s course in the emergency department corroborate that the patient’s neurological condition was deteriorating and that her condition was, in fact, consistent with extremis and life-threatening cerebral herniation in the emergency department. The mechanism of injury was a high-speed, rollover motor vehicle accident with a 10-minute extrication. Her blood pressure in the field was 80/palp with a pulse of 80 beats per minute. An \( O_2 \) saturation was not recorded in the field. However, as Dr. Defillo points out, given the difficult extrication one can presume that she may have incurred hypotension and hypoxic secondary injuries due to the delayed resuscitation. She arrived at the emergency department with blood pressure 100/palp, pulse of 106, and with agonal respirations. Her pupils were bilaterally blown. She was noted to have an initial GCS score of 6 with withdrawal of all 4 extremities to pain, but her condition quickly deteriorated. She was intubated within the first 5 minutes of arrival. Just prior to intubation, her GCS score had dropped to 4. Her condition declined from a motor examination of withdrawal to extension within this short time. Upon suctioning for the intubation, cough and gag responses were noted to be absent. As is common during resuscitation in the emergency department, intubation is a very early step and the pharmacological agents used obscure further neurological testing.

In the interests of brevity for publication, we did not detail her emergency department course and deterioration. It has been argued that the GCS score that immediately precedes operative intervention is more important than that on admission. The patient’s last neurological examination prior to paralysis showed a GCS score of 4, with fixed and dilated pupils and absent cough and gag responses. With expeditious transfer to the operating room, it is impossible to know whether the patient’s GCS score would have improved with ongoing efforts to resuscitate her hypotension and hypoxia. Given the precipitous decline in her GCS score prior to intubation and the ominous appearance of her CT scan, we think this was unlikely. Thus, we do not believe that the patient’s survival was predictable based on her initial GCS score of 6.

Independent of the initial GCS score and whether or not further resuscitation might have improved that score, the patient did go on to show radiological signs of brainstem herniation. Thus, one important point of the report is that the patient did sustain a Duret hemorrhage from which she not only survived but that she did so with a good outcome. Many regard a Duret hemorrhage as an irreversible sign of medical futility with cause to withdraw medical care. We agree that the patient’s initial GCS score of 6 may have contributed to our motivation to persist with aggressive care in this case. Differentiating patients with low GCS scores due to primary impact injuries who may be at high risk for persistent vegetative outcomes from those with potentially reversible secondary insults is extremely difficult in the acute setting. As Dr. Defillo points out, it is critically important to remember the role of resuscitation. Patients with TBI can present with GCS scores of 3 or 4 as a result of secondary injury that is both treatable and reversible. Ultimately, this is what makes TBI care in the emergency department both challenging and rewarding. (DOI: 10.3171/2009.6.JNS091009)

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Venous anomaly

To The Editor: We read with great interest the article by Walsh et al. (Walsh M, Parmar H, Mukherji SK, Mamourian A: Developmental venous anomaly with symptomatic thrombosis of the draining vein. Report of 2 cases. J Neurosurg 109:1119–1122, December, 2008). We would like to take this opportunity to comment on the first case that presented with seizures and imaging features consistent with thrombotic occlusion of the collecting vein of a large frontal lobe developmental venous anomaly (DVA). Remarkably, thrombosis of the collecting vein did not result in hemorrhagic or ischemic infarction within the drainage territory of the DVA. As the authors mention in the discussion, the essential role played by DVAs in the normal cerebral venous drainage has been well illustrated in the literature by cases of venous ischemic and hemorrhagic complications secondary to either surgical removal or spontaneous thrombosis of a DVA. It is not clear in the article by Walsh and colleagues how the confirmed thrombosis of the collecting of the DVA did not result in venous infarction.

Cerebral DVAs represent a purely venous entity, an anomalous venous disposition due to the absence of normal pial or subependymal veins. They may be understood as a variation of the venous angioarchitecture. When the DVA serves as an anomalous pathway for a deep venous territory, it can either drain centrifugally toward the cortical veins or directly into a dural venous sinus. Alternatively, where a DVA serves as an anomalous pathway for the cortical and subcortical venous territory, it drains centripetally toward the network of deep subependymal veins, tributaries of the deep venous system. In some cases, however, the venous system that the DVAs are supposed to compensate for remains patent although hypoplastic, and connections between the two may be observed. The result is a dual drainage toward the deep and superficial venous systems, with either the superficial or the deep venous system being clearly dominant over the other (personal observations; Fig. 1). Walsh et al. likely refer to this in their statement “the drainage vein can empty into the superficial venous system such as the cortical veins, the dural venous sinus, the subependymal veins, or some combination.”

Though it is not possible to ascertain from the figures that illustrate Case 1, we suppose that a connection similar to the one demonstrated in Fig. 1 between the superficially draining collecting vein of the DVA with the subependymal veins of the left frontal horn could have been retained during intrauterine life, allowing for additional centripetal drainage of the caput medusa of the DVA. Hence, the existence of this connection could account for