Intracranial pressure monitoring for brain injury

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Zeng and colleagues examine the impact of treatment based on monitored intracranial pressure (ICP) versus that delivered without monitoring on both 6-month neurologic outcome (Glasgow Outcome Scale score) and renal function. They report significantly better recovery and less renal dysfunction in monitored patients managed in accordance with the Brain Trauma Foundation guidelines.

Unfortunately, these authors do not present data sufficient to support their claim of improved outcome. Neither do they securely establish that ICP monitoring would be associated with less renal failure if this study were repeated elsewhere.

Patients were parsed into monitored versus non-monitored groups according to the Brain Trauma Foundation’s recommendations as to whom published evidence suggests should be monitored. These criteria include CT imaging characteristics, age, admission Glasgow Coma Scale [GCS] motor score, and history or presence of hypotension. By definition, then, these are dissimilar groups, and it is the authors’ responsibility to quantify and control the differences in their analysis. Unfortunately, these data were not presented; rather, the groups were deemed demographically similar based on examination of age, total admission GCS score, and sex alone. It remains unproven, and highly unlikely, that these groups are similar enough in terms of outcome-relevant demographic characteristics to allow attributing differences in outcome to ICP-monitor–based management. In our recent randomized controlled trial (RCT), when such variables were studied and controlled, we found no difference in outcome between monitored and non-monitored groups.

Regarding renal toxicity, the major risk in trauma patients, particularly those receiving hyperosmotic agents, is hypovolemia. Given that Zeng et al. found (as did we in our RCT) that ICP monitoring is associated with significantly decreased use of brain-specific therapies, including mannitol, patients managed without monitoring were at increased risk of negative fluid balance and renal hypoperfusion. In contrast to our findings, this risk was associated with what they called increased acute kidney injury (AKI). Unfortunately, they did not clearly define AKI and their reference to that definition does not provide one. The currently accepted definition of AKI would not support their reported AKI incidences. Furthermore, lack of information as to daily fluid balance or fractional excretion of sodium make it difficult to believe that the group differences in the renal data that they present are not simply due to lack of volume replacement to balance iatrogenic diuresis. In our RCT, the management protocol for all included patients required close attention to maintaining normovolemia, and our incidence of renal dysfunction was globally low and not related to treatment group (3% and 4%, for monitored versus non-monitored patients [p = 1.0]), presented in Table 10b in the online supplements to the RCT available at NEJM.org).

The use of ICP monitoring remains a critical aspect of the care of patients with severe traumatic brain injury despite the body of recent evidence suggesting that our interpretation and management algorithms need refinement based on clinical research. However, the risk of confounding is very high in such studies and requires meticulous prospective study design and data collection, shortcomings in which greatly inhibit the value of this study.

Disclosure
The author reports no conflict of interest.

References
Response

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We are grateful to Dr. Chesnut’s comments and criticisms regarding our article and offer the following response.

Regarding the neurological outcome and AKI in our enrolled patients, according to this clinical study, we only reached the conclusion that the AKI incidence rate was reduced in traumatic brain injury (TBI) patients whose treatment was based on ICP monitoring. In addition, we cannot draw the conclusion easily that the ICP monitoring can improve the neurological outcome in TBI patients because of the small statistical power, insufficient data, and nonrandom nature of our study. Therefore, in our conclusion, we only focused on the protection against kidney injury, rather than the neurological outcome.

As for the balance of 2 groups (ICP monitoring or control), as we mentioned in the article, some patients in the control group who had low GCS scores and continuous comas did not undergo ICP monitoring (which means the control group was not treated strictly in accordance with the guidelines). In fact, 27 patients in the control group had GCS scores in the 3–8 range and did not undergo ICP monitoring.

Regarding the neurological outcome again, a large retrospective study concluded that ICP monitoring was associated with significantly decreased mortality.4 In contrast, other studies have not demonstrated benefits from ICP monitoring.5-7 Furthermore, a few studies have shown that ICP monitoring was associated with worsened survival.2,8 So this assessment index is quite variable. However, differences in the monitoring site might have contributed to the problem. Some surgeons prefer intraparenchymal monitoring, while others use intraventricular monitoring. The latter is able to drain the CSF and further decrease the ICP in a safe way.

To avoid negative fluid balance and renal hypoperfusion, we maintained the urinary volume and osmotic pressure at a normal level. It is a weakness of this paper that we did not present the daily fluid balance and the level of several important electrolytes: Na+, K+, and Cl−. In a future study, we would include these important measures to support our conclusions.

As for the definition of AKI, we are very regretful: we cited the wrong reference. As we mentioned in the manuscript, “according to the clinical guidelines,” and apparently, the cited reference should not have been a literature review of animal TBI models. Actually, we adopted the AKIN criteria. This definition of AKI is based on an absolute increase in the level of serum creatinine more than 26 mmol/L. Therefore, the cited reference (Marklund N, Hillered L: Animal modelling of traumatic brain injury in preclinical drug development: where do we go from here? Br J Pharmacol 164:1207–1229, 2011) should actually have been the Renal Association Clinical Practice Guidelines on acute kidney injury.9

When it comes to the incidence rate of AKI in patients with TBI, a recent report by Corral et al. showed it was 8% in patients with severe TBI.1 In our study, the rate was 6.5% in the ICP-monitoring group, as compared with 13.2% in the control group. Admittedly, the quantity of mannitol used could be blamed for the high incidence rate. Obviously, our paper is not focused on the ICP monitor itself as having the ability to protect kidney function in patients with TBI; the point is, rather, that it is beneficial to give a reference when administering mannitol or other ICP-lowering treatment to patients with TBI. We admit that there are several shortcomings in our paper. However, there are few reports focused on the strategies for kidney protection in TBI patients. We hope that this paper might add a small amount of evidence that might help support ICP monitoring in the management of TBI.

References


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