Neurosurgical forum

Letters to the editor

Concussion

TO THE EDITOR: We read the review of Zuckerman et al.⁵ on structural brain injury in sports-related concussion (Zuckerman SL, Kuhn A, Dewan MC, et al: Structural brain injury in sports-related concussion. *Neurosurg Focus* 33(6):E6, December 2012), which suggests a number of interesting issues. In spite of the uncertainty that has arisen from the difference in the perspectives, it is a worthwhile contribution consistent with the literature. Aside from reviewing published cases of isolated head injuries, probably the most valuable contribution is inclusion of comments about unpublished cases from the authors' own institutional experience.

The paper offers a different vantage point from which subarachnoid hemorrhage (SAH), subdural hemorrhage (SDH), epidural hemorrhage (EDH), malignant edema, and second-impact syndrome can be viewed vis-à-vis the blood-brain barrier damages of various intensity, mechanism, or location. These are all manifestations of extravasation, regardless of whether focal or diffuse, per rhexis or per diapedesis bleeding took place. The term edema, in the context of malignant cerebral edema, is generally used to indicate an increase in brain volume due to an increase in tissue water.⁴ Brain swelling and cerebral edema are the common reactions of the brain to injury of whatever etiology. While generally considered to occur most commonly in infants and children, malignant cerebral edema also occurs in adults.

In practice the most common type of edema is vasogenic edema, in which there is a breakdown of the blood-brain barrier. This occurs locally around mass lesions and also more diffusely in the cerebral white matter. The second most common type of edema is cellular edema. It is most often seen in association with ischemic conditions and is more prominent in gray matter, as cellular components are found in this location.¹ It seems that blood hyperdensity could not be the only criterion that the authors used, taking into account the edema's hypodensity and the loss of normal gray-white matter differentiation. As concussion is a functional rather than structural change, we believe that it primarily disturbs endothelial functioning of the blood vessel's wall. This is involved in the formation of the blood-brain barrier. Tight junctions of the nonfenestrated endothelium and the thick basement membrane restrict the diffusion of microscopic objects and large or hydrophilic molecules into the CSF. Its disruption causes extravasation of blood elements.

Therefore, we suggest that edema and hemorrhage should be regarded as two extremes in a broader spectrum, where disruptions of endothelial continuity can be found microscopically or as evident to the naked eye.

Since the literature recognizes a concussion as physiological (functional) rather than a morphological disruption,

its severity is not measured by CT or MRI findings but by the duration of transient memory loss or disorders that interfere with the qualitative character of consciousness. The long-term effect of these functional disorders sometimes results in posttraumatic chronic encephalopathy (a combination of motor, cognitive, and psychiatric symptoms).² While CT and MRI sometimes suggest encephalopathy or cerebral atrophy, confirmation of a diagnosis is not possible until postmortem. Thus, we doubt the appropriateness of the use of radiological criteria in assessing mild traumatic brain injury (TBI). This only highlights the importance of bridging full clinical details including radiological results with results of other investigative tests. We would like to clarify that we are responding to follow up in a constructive manner and hopefully produce a creative debate on the subject. Zuckerman and colleagues were apparently looking at isolated head injuries, where the structural injury is the focus of the presentation. However, it is possible that there is an exponentially greater number of more dramatic injuries that present first, and the mild TBI is ignored.³

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Disclosure

The authors report no conflict of interest.

References

1. Ho ML, Rojas R, Eisenberg RL: Cerebral edema. *AJR Am J Roentgenol* 199;W258–W273, 2012
2. McCrory PR, Berkovic SF: Concussion: the history of clinical and pathophysiological concepts and misconceptions. *Neurology* 57:2283–2289, 2001
3. Sosa I, Grubescic A, Bosnar A: Mild traumatic brain injuries and their sequelae. I: the need for screening. *J Rehabil Med* 44:988, 2012
4. Yang Y, Rosenberg GA: Blood-brain barrier breakdown in acute and chronic cerebrovascular disease. *Stroke* 42:3323–3328, 2011
5. Zuckerman SL, Kuhn A, Dewan MC, Morone PJ, Forbes JA, Solomon GS, et al: Structural brain injury in sports-related concussion. *Neurosurg Focus* 33(6):E6, 2012

RESPONSE: We greatly appreciate the thoughtful response from Dr. Sosa and Dr. Bosnar. They clearly share our passion for understanding the impact of sports-related head injuries, ranging from functionally disruptive concussive insults to severe, structural injuries.

It bears mentioning that Drs. Sosa and Bosnar, as forensic neuropathologists, bring a unique perspective to the discussion of functional and structural brain injuries. From our neurosurgical viewpoint, we are familiar primarily with the acute and long-term medical and sur-

gical management of structural head injuries. However, neurosurgeons rarely engage in any form of postmortem examination, and any lessons gleaned from such an examination are often missed by the practicing neurosurgical clinician. The perspective of the forensic pathologists is invaluable and sheds new light on a well-known clinical problem.

Our colleagues from the Rijeka School of Medicine in Croatia provide a succinct review of the two different types of edema, vasogenic and cellular. We agree that pathophysiologically, brain hemorrhages (such as SDH, EDH, and SAH) are categorized by extravasation, and edema is altogether a different pathophysiological entity. However, the reason for grouping these two abnormalities as structural injuries stems from their appearance on neuroimaging. Blood, whether acute or chronic, can easily be seen on a noncontrast head CT scan. Malignant cerebral edema can be similarly recognized with loss of sulci, cisternal effacement, and possible herniation. The commonality of positive neuroimaging findings was the essential justification for grouping these injuries together in our review.

To our colleagues' second point, note is made that a concussion, or mild TBI, is a functional (as opposed to structural) injury not evident on routine neuroimaging. Furthermore, they "doubt the appropriateness of the use of radiological criteria in assessing mild traumatic brain injury (TBI)." We wholeheartedly agree that concussions, by definition, are not diagnosed through imaging techniques. As the Concussion in Sport Group (CISG) asserts, "No abnormality on standard structural neuroimaging studies is seen in concussion."²

However, there is a fundamental misunderstanding at the root of one of the points made by Drs. Sosa and Bosnar, and this was due in fact to an error of omission in our review. Approximately 75% of patients with moderate TBI have extraaxial blood present on CT, and severe TBI is categorized by any intracranial contusion, hematoma, or brain laceration—all structural injuries.¹ We believe that any structural injury elevates a concussion or mild TBI to at least a moderate TBI, while implying simul-

taneously that a functional injury has also taken place. We make the assumption that the presence of a structural injury inherently includes a functional brain injury. For example, if a child presents to the emergency department after striking his head against the hardwood basketball court to a degree sufficient enough to produce a small SDH (where extravasation has occurred), the assumption is that enough force was also provided to induce a concussion. When treating patients with structural injuries, it is our routine practice to counsel the patient and family that a functional injury has also occurred. A full clinical history is equally important in cases with positive imaging.

Finally, we wish to add that the unpublished cases mentioned in our review article and highlighted by our colleagues in their letter have been submitted for editorial review.

Again, we thank Drs. Sosa and Bosnar for taking the time to elucidate further a nebulous area. Their comments have prompted us to crystallize our thinking, and for that we are appreciative. We hope our response serves to clarify an area ripe with gray and vague terminology. It is only through constructive discussion and debate that difficult-to-define concepts can be refined and explicated.

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References

1. Decuyper M, Klimo P Jr: Spectrum of traumatic brain injury from mild to severe. *Surg Clin North Am* 92:939–957, 2012
2. McCrory P, Meeuwisse W, Johnston K, Dvorak J, Molloy M, Cantu R: Consensus Statement on Concussion in Sport—The 3rd International Conference on Concussion in Sport Held in Zurich, November 2008. *PM R* 1:406–420, 2009

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