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Oral Presentation

Severe Traumatic Brain Injury Following Motor Vehicle Accidents: Pathogenesis, Pathophysiology and Prognosis

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Abstract

Over half of all reported traumatic brain injuries are the result of a motor vehicle accident. Other then the direct head impact itself, the sheer forces of the accident can cause the brain also to collide against the internal bone of the skull. In fact, when a moving head comes to a quick stop, the brain continues in its movement, striking the interior of the skull. This can cause bruising of the brain and/or bleeding form vessels tearing. The consequences are immediate, with a scarcely predictable spectrum of lesions: from simple scalp lacerations (SLs), up to the most severe types of diffuse axonal injury (DAI). SLs can bleed profusely, causing dangerous acute hypotension and may act as a conduit for infection when associated with depressed skull fractures (SFs). SFs generally correlate with severity of injury, and patients with a SFs have a much higher likelihood of having an intracranial hematoma, such as extradural hematomas. Contusions are found to have a characteristic distribution, with the frontal poles, orbital gyri, the cortex above and below the Sylvian fissure, the temporal poles, and the lateral and inferior aspects of the temporal lobes. Lacerations of the frontal and temporal lobes are frequently associated with acute subdural bleeding and/or intraparenchymal hemorrhage. DAI causes by itself 35% of all deaths after head injury, and may or may not be accompanied by intracranial mass lesion. Approximately half of severe cases of DAI exhibit focal lesions of the corpus callosum, the rostral brainstem, and microscopic evidence of widespread damage of the white matter. Urgent surgical candidates are those patients in whom a mass lesion is causing a significant mass effect. Radiographically, lesions are considerable when they are more than 25 cc of volume, and/or cause effacement of the basal cisterns or midline shift > 5mm. More important, clinically significant lesions are those causing progressive neurologic symptoms. The decision to operate is based on a combination of the history of the trauma, significance of the patient's neurologic deficits, and the significance of findings on cerebral imaging. Prognosis for victims of severe brain injury has improved in the past two decades, with a decreasing mortality. Still, however, one out of two will face permanent neurological deficits. Undoubtedly, after the urgent surgical treatment that represents the crucial action to stop the "run against the clock", a better understanding of the pathophysiology and the development of new pharmacological strategies is mandatory. Continual improvements in the training of pre-hospital medical personnel, rapid transport of head injury victims to definitive care, and the increase of the Trauma Centers and Neuroscience Intensive Care Units will play a role in the next future.

Keywords: Severe Traumatic Brain Injury, Pathogenesis, Pathophysiology, Prognosis.

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