CONFERENCE ABSTRACT

Distant Organs Complications Post Traumatic Brain Injury

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Background: The central nervous system (CNS) plays an integral role in controlling body organs; there is a constant interplay of neurotransmitters between the CNS and extracranial organs. Hence, an isolated traumatic brain injury may be associated with various physiological responses of other body organs. Extracranial injuries such as polytrauma and infection may also be associated with changes in neuropathology in patients with TBI. The aim of this paper is to highlight the systematic organs complications post TBI.

Methods: A literature review of extracranial organ complications post-TBI was conducted. The findings were presented in a lecture format, using a head-to-toe systematic assessment format. The presentation started with changes in the pituitary gland, followed by heart and lung changes, then gastrointestinal and renal changes, and a brief on immune system changes. The main mechanism of these physiological changes was discussed, such as inflammation, catecholamine surge, and increased sympathetic tone affecting systematic organs.

Result: Literature identified pituitary changes in up to 47% of traumatic subarachnoid hemorrhage. Hormonal insufficiency and antidiuretic hormone secretion abnormalities are the most common forms of pituitary dysfunction in patients with TBI. Diabetes Insipidus is reported in up to 51% of TBI cases, and it is associated with a 57% to 69% mortality rate among patients with TBI. Cardiac dysfunctions are reported in up to 74% of TBI cases in which ECG abnormalities are the most common form of cardiac changes. Cardiovascular failure was reported in 18% of TBI cases. Catecholamine surge leads to structural change in cardiomyocytes due to the direct effect of norepinephrine on cardiac muscles. Severe dysregulation of catecholamine homeostasis affects pulmonary vessels, leading to increased blood pressure and massive blood shifts to the pulmonary vasculature, causing Neurogenic Pulmonary Edema (NEP). In contrast, NPE can further harm the brain due to hypoxia and inadequate oxygen supply to brain cells. Brain injury insults the renal system indirectly in the form of electrolyte disturbances such as hypo and hypernatremia. Hyponatremia is reported in 51% of TBI cases. Stress ulcer is the most common form of gastrointestinal system changes among patients with TBI. In addition, CNS injury stimulates the immune system, leaving patients with TBI highly susceptible to infection due to the release of proinflammatory cytokines such as IL-1, IL-6, and TNF-

α. The systematic inflammatory response state impacts organ hemostasis, resulting in tissue hypoperfusion.

Conclusion: Cerebral injury is not only a brain disease, but it also affects the body as a whole, leading to systematic changes that require a holistic, therapeutical approach to manage. Autonomic imbalance caused by CNS dysfunction may lead to various systematic complications. Understanding the mechanism of extracranial organ injuries is key to preventing and managing complications. Elevation of inflammatory biomarkers may predict mortality and morbidity in patients post-TBI.