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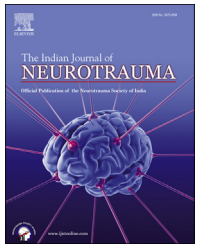

Letter to the Editor

Role of autoantibodies in the pathophysiology of hypopituitarism following traumatic brain injury

We read with interest the review 'Endocrine manifestations of traumatic brain injury' written by Agrawal et al.[1] The authors have broadly described the causes of post-traumatic brain injury (TBI) hypopituitarism to be ischemia–hypoxia, direct mechanical injury, compression injury and vascular injury. It is worthwhile to mention here that recent studies have shown the presence of anti-pituitary antibodies (APAs) and anti-hypothalamic antibodies (AHAs) following TBI as an important potential cause of hypopituitarism. Preliminary studies on animals have shown the presence of APAs and AHAs, similar to those seen in Sheehan's syndrome of postpartum pituitary necrosis.[2,3] Up to 44% patients with TBI showed the presence of APAs in a study by Tanriverdi et al.[3] These APAs may have distinct selective bias toward the growth hormone (GH) releasing hormone secreting cells as compared to the vasopressin secreting cells, as suggested by Tanriverdi's study, which showed that all AHA-positive cases were accompanied by at least some GH deficiency but no diabetes insipidus.[4] Damage to the blood–brain barrier allowing escape of brain proteins into the circulation and thus evoking an immunological response[5] or, inflammation triggering a vascular cascade causing histopathological changes may be responsible.[3] Spontaneous disappearance of these APAs from the circulation, allowing functional recovery of the pituitary may be the cause of transient hypopituitarism seen in a number of TBI patients.[4] De Bellis et al[6] suggest that if detectable in high titer (>1.8) these antibodies may be used as markers of hypopituitarism. Research on the mechanism of formation of these autoantibodies after TBI may have promising rewards in preventing post-TBI hypopituitarism-induced morbidity and mortality in the future.

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