Confronting 'Paroxysmal Sympathetic Storming' in Traumatic Brain Injury

Editorial

Dysautonomia or Paroxysmal sympathethetic storming occurs in around 10-30% of TBI [1-3]. It exerts a profound negative influence on the final outcome in affected individuals. Dysautonomia commonly occurs following severe head injury if associated with at DAI, Brain stem Injury, preadmission hypoxia or in the younger age group [4,5]. The pathophysiological basis for dysautonomia is yet to be determined but recent evidences support two recent hypothesis - first, the direct injury to the Hypothalamus causing dysautonomia and second functional disconnection of the hypothalamus and diecephalon from rest of the Central nervous system secondary to the axonal injury to the afferent and efferent pathway [6,7].

Tachycardia, Tachypnoea, hypertension, fever, Diaphoresis, rigidity and posturing are the common clinical findings in Dysautonomia. Presence of 5 out of any of the above can be considered diagnostic of Dysautonomia [6-8]. Infection or agitation can also mimick Dysautonomia. Correct identification of the syndrome and optimal management is highly detrimental in the management of head injuries [9].

Phase 1 which lasts during the initial 1 week where patient is on ventilator and sedated with or without paralysis. It's in phase 2 where the above said symptoms presents and occurs on stopping sedation. Phase 3 represents the burnt out Dysautonomia which begins with stoppage of diaphoresis and leaves patient in variable dystonia/spasticity [1].

Goal of managing dysautonomia is to prevent secondary injury caused by the Paroxysmal sympathetic storming. Dysautonomia aggravates secondary brain injury by basically 3 mechanisms, first Hyperthermia, which needs very aggressive control. Second the rigidity and posturing that markedly increases the energy expenditure to upto 200% and resulting in a relative malnutrition. Third there is an elevation of circulating catecholamines which is an independent predictor of poor outcome [10-15].

Pharmacological interventions to treat Dysautonomia are plenty. Benzodiazepine and Narcotic combination reduces the number and frequency of paroxysms probably by reduction of brain activity. Other medications that have shown success include Clonidine (alpha 2 adrenergic agonist) which decrease central sympathetic outflow. The hypertensive and tachycardia component can be controlled through Labetolol. Bromocriptine (Dopamine D2 agonist) is effective in controlling fever and diaphoresis. Intrathecal Baclofen has been found to be of some help in managing autonomic dysutonomia [16-25].

Editorial

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