Confronting 'paroxysmal sympathetic storming' in traumatic brain injury

Volume 7 Issue 4 - 2017

Arun Oommen

VPS Lakeshore Hospital, India

Correspondence: Arun Oommen, Consultant Neurosurgeon, VPS Lakeshore Hospital Kochi, India, Email oommenarun@yahoo.co.in

Received: July 19, 2017 | **Published:** September 20, 2017

Editorial

Dysautonomia or Paroxysmal sympathetic 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 diencephalon 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 mimic 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 dysautonomia.¹⁶⁻²⁵

Acknowledgments

None.

Conflicts of interest

None.

Funding

None.

References

1. Baguley IJ, Nicholls JL, Felmingham KL, et al. Dysautonomia after traumatic brain injury. A forgotten syndrome? *J Neurol Neurosurg Psychiatry*. 1999;67(1):39-43.
2. Blackman JA, Patrick PD, Buck ML, et al. Paroxysmal autonomic instability with dystonia after brain injury, *Arch Neurol*. 2004;61(3):321-328.
3. Baguley IJ, Cameron ID, Green AM, et al. Pharmacological management of dysautonomia following TBI. *Brain Inj*. 2004;18(5):409-417.
4. Fernández-Ortega JF, Prieto-Palomino MA, Muñoz-López A, et al. Prognostic Influence and computer tomography findings in dysautonomic crisis after traumatic brain injury. *J Trauma*. 2006;61(5):1129-1133.
5. Boeve BF, Wijdicks EF, Benarroch EE, et al. Paroxysmal sympathetic storms (Diencephalic seizures) after DAI. *Mayo clinic Proc*. 1998;73(2):148-152.
6. Benarroch EE. The central autonomic network, functional organization, dysfunction and perspective, *Mayo Clin Proc*. 1993;68(10):988-1001.
7. Rossitch E Jr, Bullard DE. The autonomic dysfunction syndrome, aetiology and treatment. *Br J Neurosurg*. 1988;2(4):471-478.
8. Pranzatelli MR, Pavlakis SG, Gould RJ, et al. Hypothalamic- Midbrain dysregulation syndrome; Hypertension, hyperthermia, hyperventilation, and decerebration. *J Child Neurol*. 1991;6(2):115-122.
9. Jiang JY, Gao GY, Li WP, et al. Early indicators of prognosis in 846 cases of severe traumatic brain injury, *J Neurotrauma*. 2002;19(7):869-874.
10. Natale JE, Joseph JG, Helfaer MA, et al. Early Hyperthermia after Traumatic brain injury in children, risk factors, influence on length of stay, and effect of short term Neurologic status. *Critic Care Med*. 2000;28(7):2608-2615.
11. Suz P, Vavilala MS, Souter M, et al. Clinical features of fever associated with poor outcome in severe paediatric brain injury. *J Neurosurg Anaesthesiol*. 2006;18(1):5-10.
12. Clifton GL, Zeigler MG, Grossman RG. Circulating catecholamines and sympathetic activity after head injury. *Neurosurgery*. 1981;8(1):10-14.

13. Moore R, Najarian MP, Konvolinka CW. Measured energy expenditure in severe head Trauma. *J Trauma* . 1989;29(12):1633-1636.
14. Biffi WL, Moore EE, Haenel JB. Nutritional support of trauma patient. *Nutrition*. 2002;18(11-12):960-965.
15. Wilson RF, Tyburski JG. Metabolic response and nutritional therapy in patients with severe head injury. *J Head Trauma rehabil*. 1998;13(1):11-27.
16. Bullard DE. Diencephalic seizures; responsiveness to bromocriptine and morphine. *Ann Neurol*. 1987;21(6):609-611.
17. Sneed RC. Hyperpyrexia associated with sustained muscle contractions, an alternative diagnosis to central fever. *Arch physio medical rehabilitation*. 1995;76(1):101-103.
18. Robertson CS, Clifton GL, Taylor AA, et al. Treatment of hypertension associated with TBI. *J Neurosurg*. 1983;59(3):455-460.
19. Sandel ME, Abrams PL, Horn LJ. Hypertension after brain injury, case report. *Arch phys med rehabilitation*. 1986;67(7):469-472.
20. Do D, Sheel VL, Bromfield E. Treatment of paroxysmal sympathetic storm with Labetolol. *J neurol Neurosurgery Psychiatry*. 2000;69(6):832-833.
21. Russo RN, O'Flaherty S. Bromocriptine for the management of autonomic dysfunction after severe TBI. *J paediatric child health*. 2000;36(3):283-285.
22. Chioléro RL, Breitenstein E, Thorin D, et al. effects of propranolol on resting metabolic rate after severe head injury. *Crit Care Med*. 1989;17(4):328-334.
23. Meythaler JM, Stinson AM III. Fever of central origin in traumatic brain injury controlled with propranolol. *Arch phys rehabil*. 1994;75(7):816-818.
24. Cuny E, Richer E, Castel JP. Dysautonomic syndrome in the acute recovery phase after traumatic brain injury: relief with intrathecal baclofen therapy. *Brain Inj*. 2001;15(10):917-925.
25. François B, Vacher P, Roustan J, et al. Intrathecal baclofen after traumatic brain injury; early treatment using a new technique to prevent spasticity. *J Trauma*. 2001;50(1):158-161.