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Short Communication

Paroxysmal Sympathetic Hyperactivity and the Need for Individualized Care with Regular Monitoring

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DESCRIPTION

Paroxysmal Sympathetic Hyperactivity (PSH), also known as autonomic storming, results from sympathetic nervous system dysregulation [1,2]. PSH can follow any type of severe acquired brain injury but most commonly occurs after Traumatic Brain Injury (TBI). Specifically, whereas the prevalence of PSH following TBI is 33%, the prevalence is only 6% with other conditions [3]. Those who survive TBI have up to a 10% chance of developing PSH [4,5].

PSH usually occurs within a week of a TBI, with the likelihood decreasing over time as the brain recovers [4,6]. Poor neurological outcomes occur when PSH develops earlier following severe traumatic brain injury and when the PSH symptoms are more severe [7].

The condition presents as excessive sympathetic activity that occurs in recurrent episodes, which are frequently triggered by certain types of stimulation [8,9]. These episodes eventually cease, often once an abortive medication has been administered.

Challenges in the diagnosis and treatment of PSH jeopardize the health of those with the condition and necessitate consideration of aid and attendant care for these patients. Here we summarize key features of PSH and highlight how its proper management requires specialized care and regular monitoring to prevent life-threatening complications.

The pathogenesis of PSH is not well understood, obfuscating proper and rapid diagnosis

The diagnostic tool best used at present for identifying PSH in those who have suffered a TBI is the Paroxysmal Sympathetic Hyperactivity-Assessment Measure (PSH-AM) scale, which assesses the presence of certain clinical features as well as their intensity [2,7,10]. However, PSH diagnosis is challenging because the pathogenesis of the condition is not entirely understood. As a result, there is a lack of education amongst healthcare providers around how to identify PSH [11]. Misdiagnosis also frequently occurs due to this lack of education, as well as the similar clinical features of PSH and other serious diseases, such as hydrocephalus and septicemia [12,13].

The bulk of healthcare provider knowledge regarding PSH focuses on the six core symptoms associated with the condition and to a lesser extent, the comorbidities that tend to accompany PSH. Most patients with PSH display some but not all the six core symptoms [14]. These six symptoms include tachycardia, tachypnea, hypertension, hyperthermia, hyperhidrosis and posturing [15]. According to one study, tachycardia is the most common of these symptoms [13]. Common comorbidities include weight loss, cardiovascular changes and immunodepression [16-19].

There are clinical and demographic risk factors that predict PSH development

Though the pathogenesis of PSH is not entirely understood, certain predictive factors have been identified. For instance, certain types of anatomical damage with TBI, such as focal neural parenchymal, increases the likelihood of developing PSH [20]. Similarly, damage to the deeper areas of the brain is associated with PSH risk. Accordingly, brainstem, corpus callosum and periventricular white matter injuries are more likely to lead to PSH than injuries closer to the cortex [21]. Lesions that are scattered and axonal injuries that are diffuse are also associated with PSH [22].

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In addition to anatomical risk factors, age, sex and Glasgow Coma Score (GCS) are also correlated with PSH risk. Among adults, older people are less likely to suffer PSH [23]. Women are also less likely than men to develop the condition [24]. Patients with higher GCS also appear to be somewhat protected from PSH compared to those with severe TBI and GCS lower than 8 points [25,26]. Further, GCS is observed to decrease during PSH episodes and to rise between episodes.

Lack of precise treatment paradigm necessitates individualized care

Conventional interventions for PSH include sedation, muscle relaxation and analgesia [27]. β-blockers and opioids are pharmaceuticals commonly used to treat the condition [28,29]. Most people with PSH require a combination of medications for treatment and to prevent problematic episodes [30].

It is critical that PSH is recognized and diagnosed rapidly, as untreated PSH is likely to worsen and can lead to serious complications. Compounding the risk of undiagnosed PSH is the potential for unnecessary interventions to treat symptoms or misdiagnosed conditions.

Because there tend to be several symptoms present upon diagnosis of PSH, it is also vital that the urgency of treating each symptom be clarified before treatment is initiated [19]. For instance, in the case of hyperthermia, interventions for cooling are a priority, whereas hydration is potentially more essential in patients with hyperhidrosis [31].

Despite a general standard of care for PSH, significant individual differences in PSH manifestation have prevented the development of highly effective and precise therapies for the condition [31]. Individualized care focused on the unique conditions and pathophysiology of each patient is therefore required to optimize outcomes across the PSH patient population.

Aid and attendant care can improve outcomes in PSH

Because people with PSH suffer events, the onset of which may not be entirely predictable, these patients need ongoing monitoring so that complications can be avoided or rapidly addressed, and catastrophic outcomes can be prevented. In addition to intervening in potentially life-threatening situations, there are several other ways in which the appropriate level and amount of aid and attendant care may support PSH patients.

Managing medication is one way aid and attendant care can improve outcomes for those with PSH. For instance, they can help to minimize unwanted side effects and prevent overdosing, which are priorities of PSH. They can also contribute to a positive impact on the progression of PSH recovery through careful dose titration and initiating the switching of medications and therapies as appropriate [31].

Modifying the patient's environment and diet are other critical ways aid and attendant care can benefit those with PSH [17,32]. For instance, it is important to prevent environmental stimuli that may trigger episodes, including variations in room temperature. Proper nutrition can also contribute to reduced rates of long-term mortality from PSH. The introduction and management of mineral supplementation and enteral feeding represent ways care providers can support nutrition [33].

CONCLUSION

Through mechanisms that are not entirely clear, dysregulation of the sympathetic nervous system following severe TBI can lead to PSH. Though this condition does not manifest in a uniform or highly predictable manner, it is associated with several symptoms and comorbidities, including tachycardia and weight loss, respectively. It can lead to serious complications, and unfortunately, there is not currently a highly effective treatment paradigm for PSH.

The array of clinical features and potential treatment options across the PSH population highlights the critical importance of individualized care to ensure the best outcomes for each patient. Additionally, as PSH is associated with relatively unpredictable episodes that must be addressed in real-time, regular monitoring is vital to prevent catastrophic events. Several aspects of a PSH patient's daily routine, including their environmental stimuli and nutrition, must also be personalized and regularly managed. As such, the appropriate amount and level of aid and attendant care should be recommended to improve outcomes in those with PSH.

REFERENCES

1. Perkes I, Baguley IJ, Nott MT, Menon DK. A review of paroxysmal sympathetic hyperactivity after acquired brain injury. Ann Neurol. 2010;68(2):126-135.
2. Meyfroidt G, Baguley IJ, Menon DK. Paroxysmal sympathetic hyperactivity: The storm after acute brain injury. Lancet Neurol. 2017;16(9):721-729.
3. Thomas A, Greenwald BD. Paroxysmal sympathetic hyperactivity and clinical considerations for patients with acquired brain injuries: A narrative review. Am J Phys Med Rehabil. 2019;98(1): 65-72.
4. Pozzi M, Conti V, Locatelli F, Galbiati S, Radice S, Citerio G, et al. Paroxysmal sympathetic hyperactivity in pediatric rehabilitation: Clinical factors and acute pharmacological management. J Head Trauma Rehabil. 2015;30(5):357-363.
5. Fernandez-Ortega JF, Prieto-Palomino MA, Garcia-Caballero M, Galeas-Lopez JL, Quesada-Garcia G, Baguley IJ. Paroxysmal sympathetic hyperactivity after traumatic brain injury: Clinical and prognostic implications. J Neurotrauma. 2012;29(7):1364-1370.
6. Baguley IJ, Slewa-Younan S, Heriseanu RE, Nott MT, Mudaliar Y, Nayyar V. The incidence of dysautonomia and its relationship with autonomic arousal following traumatic brain injury. Brain Inj. 2007;21(11):1175-1181.
7. Zheng RZ, Lei ZQ, Yang RZ, Huang GH, Zhang GM. Identification and management of paroxysmal sympathetic hyperactivity after traumatic brain injury. Front Neurol. 2020;11:81.
8. Baguley IJ, Nott MT, Slewa-Younan S, Heriseanu RE, Perkes IE. Diagnosing dysautonomia after acute traumatic brain injury: Evidence for overresponsiveness to afferent stimuli. Arch Phys Med Rehabil. 2009;90(4):580-586.
9. Baguley IJ, Heriseanu RE, Nott MT, Chapman J, Sandanam J. Dysautonomia after severe traumatic brain injury: Evidence of

persisting overresponsiveness to afferent stimuli. Am J Phys Med Rehabil. 2009;88(8):615-622.

10. Baguley IJ, Perkes IE, Fernandez-Ortega JF, Rabinstein AA, Dolce G, Hendricks HT. Paroxysmal sympathetic hyperactivity after acquired brain injury: consensus on conceptual definition, nomenclature, and diagnostic criteria. J Neurotrauma. 2014;31(17): 1515-1520.
11. Choi HA, Jeon SB, Samuel S, Allison T, Lee K. Paroxysmal sympathetic hyperactivity after acute brain injury. Curr Neurol Neurosci Rep. 2013;13(8):370.
12. Lump D, Moyer M. Paroxysmal sympathetic hyperactivity after severe brain injury. Curr Neurol Neurosci Rep. 2014;14:1-7.
13. Hughes JD, Rabinstein AA. Early diagnosis of paroxysmal sympathetic hyperactivity in the ICU. Neurocrit Care. 2014;20:454-459.
14. Mathew MJ, Deepika A, Shukla D, Devi BI, Ramesh VJ. Paroxysmal sympathetic hyperactivity in severe traumatic brain injury. Acta neurochirurgica. 2016;158:2047-2052.
15. Rabinstein AA. Paroxysmal sympathetic hyperactivity in the neurological intensive care unit. Neurol Res. 2007;29(7):680-682.
16. Woiciechowsky C, Asadullah K, Nestler D, Eberhardt B, Platzer C, Schöning B, et al. Sympathetic activation triggers systemic interleukin-10 release in immunodepression induced by brain injury. Nat Med. 1998;4(7):808-813.
17. Mehta NM, Bechard LJ, Leavitt K, Duggan C. Severe weight loss and hypermetabolic paroxysmal dysautonomia following hypoxic ischemic brain injury: The role of indirect calorimetry in the intensive care unit. J Parenter Enteral Nutr. 2008;32(3):281-284.
18. Hendricks HT, Geurts AC, van Ginneken BC, Heeren AJ, Vos PE. Brain injury severity and autonomic dysregulation accurately predict heterotopic ossification in patients with traumatic brain injury. Clin Rehabil. 2007;21(6):545-553.
19. Burton JM, Morozova OM. Calming the storm: Dysautonomia for the pediatrician. Curr Probl Pediatr Adolesc Health Care. 2017;47(7):145-150.
20. Fernández-Ortega JF, Prieto-Palomino MA, Muñoz-López A, Lebron-Gallardo M, Cabrera-Ortiz H, Quesada-García G. Prognostic influence and computed tomography findings in dysautonomic crises after traumatic brain injury. J Trauma. 2006;61(5):1129-1133.
21. Lv LQ, Hou LJ, Yu MK, Qi XQ, Chen HR, Chen JX, et al. Prognostic influence and magnetic resonance imaging findings in paroxysmal sympathetic hyperactivity after severe traumatic brain injury. J Neurotrauma. 2010;27(11):1945-1950.
22. Hinson HE, Puybasset L, Weiss N, Perlbarg V, Benali H, Galanaud D, et al. Neuroanatomical basis of paroxysmal sympathetic hyperactivity: A diffusion tensor imaging analysis. Brain injury. 2015;29(4):455-461.
23. Cuny E, Richer E, Castel JP. Dysautonomia syndrome in the acute recovery phase after traumatic brain injury: Relief with intrathecal Baclofen therapy. Brain Inj. 2001;15(10):917-925.
24. Lv LQ, Hou LJ, Yu MK, Qi XQ, Chen HR, Chen JX, et al. Risk factors related to dysautonomia after severe traumatic brain injury. J Trauma. 2011;71(3):538-542.
25. Baguley IJ, Nicholls JL, Felmingham KL, Crooks J, Gurka JA, Wade LD. Dysautonomia after traumatic brain injury: A forgotten syndrome?. J Neurol Neurosurg Psychiatry. 1999;67(1):39-43.
26. Alofisan TO, Algarni YA, Alharfi IM, Miller MR, Stewart TC, Fraser DD, et al. Paroxysmal sympathetic hyperactivity after severe traumatic brain injury in children: Prevalence, risk factors, and outcome. Pediatr Crit Care Med. 2019;20(3):252-258.
27. Pucks-Faes E, Hitzenberger G, Matzak H, Verrienti G, Schauer R, Saltuari L. Intrathecal baclofen in paroxysmal sympathetic hyperactivity: Impact on oral treatment. Brain Behav. 2018;8(11):e01124.
28. Baguley IJ, Cameron ID, Green AM, Slewa-Younan S, Marosszeky JE, Gurka JA. Pharmacological management of dysautonomia following traumatic brain injury. Brain Inj. 2004;18(5):409-417.
29. Feng Y, Zheng X, Fang Z. Treatment progress of paroxysmal sympathetic hyperactivity after acquired brain injury. Pediatr Neurosurg. 2015;50(6):301-309.
30. Blackman JA, Patrick PD, Buck ML, Rust Jr RS. Paroxysmal autonomic instability with dystonia after brain injury. Archives of Neurology. 2004;61(3):321-328.
31. Rabinstein AA, Benarroch EE. Treatment of paroxysmal sympathetic hyperactivity. Current Treatment Options in Neurology. 2008;10(2):151-157.
32. Lemke DM. Riding out the storm: Sympathetic storming after traumatic brain injury. J Neurosci Nurs. 2004;36(1):4-9.
33. Caldwell SB, Smith D, Wilson FC. Impact of paroxysmal sympathetic hyperactivity on nutrition management after brain injury: A case series. Brain Inj. 2014;28(3):370-373.