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Identification and Management of Post-Traumatic Paroxysmal Sympathetic Hyperactivity: Role of Beta-Blockers

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Description

Sympathetic Hyperactivity Syndrome (PSH) is a syndrome characterized by hypertension, diaphoresis, tachycardia and agitation. First described by Penfield in 1929, he called it diencephalic convulsions [1]. However, electrophysiological investigations showed no electro-encephalic activity. Since then, many names have been given to this syndrome including dysautonomia, sympathetic storming, brainstem attack, autonomic dysregulation, and paroxysmal autonomic instability with dystonia. However, it is only very recently in 2014 that the International Brain Injury Association consensus has determined a nomenclature "paroxysmal sympathetic hyperactivity" as well as clear diagnostic criteria through a tool called PSH-AM(Paroxysmal Sympathetic Hyperactivity Assessment Measure). This tool consists of two parts, the first of which rates the severity of the clinical signs from 0-3 and the second part the diagnostic probability. The numerical result of each part is put together and the sum of the two parts gives the probability of a diagnosis of PSH; unlikely (score<8), possible (scores between 8-16) and probable (score ≥ 17) [2-6].

The prevalence of PSH is about 8%-33% in intensive care units, this wide range reflects the difficulties of diagnosis and admission as well as ignorance of the identification of the disease according to the causes, and the prevalence of PSH is 33% after a head injury and 6% for other causes. The aetiologies of PSH are secondary to, severe acquired brain injuries including traumatic brain injury, stroke, anoxic brain injury, tumors, infections, spinal injuries, serotonin syndrome. Most reported cases are secondary to Traumatic Brain Injury (TBI). 79.4% of PSH are secondary to traumatic brain injury, 80% of patients have a moderate-to-severe traumatic brain injury, 15%-33% has a severe traumatic brain injury. Followed by hypoxic brain injury (9.7%) and cerebrovascular accident (5.4%) [7].

Very little is known about the pathophysiology of this condition, the first theory of diencephalic seizure was abandoned for lack of empirical evidence, a disconnection theory suggests a failure of the central autonomic connection (insular cortex, amygdala, hypothalamus, medulla, the periaqueductal grey matter, the parabrachial complex and the nuclei of the solitary tract), although there is evidence to support this theory of disconnection of the cerebral inhibitory pathway, it is insufficient to account for all of the symptoms observed in patients with PSH. Currently, a widely accepted theory includes an Excitation/Inhibition Ratio (EIR). This theory describes PSH in two pathological processes, firstly, the excitation originates from the disconnection of the descending inhibitory pathway and secondly, the paroxysm is arrested by the re-establishment of inhibitory factors.

However, the anatomical basis of PSH pathogenesis remains undefined; research suggests that specific traumatic brain injuries may increase the occurrence of PSH. Therefore, the treatment of PSH is not yet clearly established. Pharmacological agents used alone or in combination are benzodiazepine, opiates, bromocriptine, gabapentin, baclofen, muscle relaxants (dantrolene), alpha-blockers, and beta-blockers [5].

Beta-blockers have a cardioprotective and neuroprotective effect due to their pharmacological properties. On the one hand, they reduce heart rate, perfusion volume and mean arterial pressure, thus limiting oxygen consumption and preventing myocardial infarction. On the other hand, they reduce cerebral blood flow, which reduces cerebral oxygen and glucose consumption, as the metabolism is reduced. In addition, non-cardioselective beta-blockers have a lipophilic property that allows them to cross the blood-brain barrier. The result is that beta-blockers decrease the effect of circulating catecholamines and attenuate basal metabolism, which is increased in cerebral subjects. Propranolol and labetalol have been shown effects against PSH, but there is still very little evidence to support the use of beta-blockers alone as a treatment for PSH [8-11].

Conclusion

There should therefore be a greater focus on understanding, identifying and managing PSH, including the role of beta-blockers in treatment, so that more than 90 years later a clear treatment is established.

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