Management of Paroxysmal Sympathetic Hyperactivity Guideline

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Background:

Paroxysmal sympathetic hyperactivity (PSH) is commonly also referred to as dysautonomia or “storming” and is a syndrome consisting of paroxysmal simultaneous increases in sympathetic nervous system (SNS) function. Neurological trauma is a common precursor to PSH. More commonly seen in younger patients, these episodes consist of an exaggerated stress response (often unprovoked) that may occur in up to a third of patients with severe traumatic brain injury (TBI). The incidence rate is 15-30% of comatose patients (GCS≤8) with severe TBI. PSH also occurs with other conditions (i.e, hypoxia, stroke, hydrocephalus, tumor, NMDA receptor encephalitis) The onset can be within 24 hours up to weeks following injury and may persist into the rehabilitation phase for weeks to months after injury in patients that remain in a low-response state. The identification of PSH is reliant on clinical observation of the specific symptom cluster, and the diagnosis remains one of exclusion.

Storming is theorized to be due to an increase in activity of the SNS created by a disassociation between the sympathetic and parasympathetic nervous systems. The increased activity of the SNS with resultant elevated levels of circulating catecholamines can have long term implications including permanent cardiac and skeletal muscle fiber damage and is an independent predictor of poor outcome after traumatic brain injury. Early recognition and treatment of storming has the potential to minimize secondary brain injury.

The increased metabolic rate and diaphoresis that occurs with persistent storming can lead to hypernatremia, renal insufficiency as well as thickening of pulmonary secretions. The increased metabolic rate can also result in an elevated core body temperature, hyperglycemia and an increased risk of muscle wasting/weight loss. Energy needs can be increased by 100-200% in the presence of storming. Persistent hypertension resulting from prolonged increases in catecholamines can increase bleeding risk in the setting of intracranial hemorrhage and potentiate arrhythmias.

Clinical Features/Hallmark Symptoms:

Hyperthermia (>38.5 C)

Tachycardia (HR>120 bpm)*

Hypertension (SBP>160 mmHg)*

Tachypnea (>20 breaths per min)*

Pupil dilation

Posturing during episodes

Dystonia

Altered LOC

Diaphoresis

Common Triggers:

Non-noxious stimuli

Pain

Environmental stimuli

Suctioning

Hyperthermia

Constipation

Urinary retention

Passive movements (repositioning, bathing, stretching)

***** *acute (within 15 minutes) change from baseline*

Scoring Tool (taken from J Neurotrauma 2014

Treatment Algorithm:

Presence of ≥ 4 hallmark symptoms

or CFS + DLT score >17

Pharmacologic Measures:

* utilize lower dose and extended interval if significant renal dysfunction

ŧ check LFTs prior to initiation; do not use if active liver disease; do not use in combination with calcium channel blockers as this may result in severe hyperkalemia and cardiovascular collapse

Non-Pharmacologic Interventions:

- Minimize triggers or group timing of common triggers together
- Cooling measures

Opioids

- Considered an abortive therapy
- Widely distributed opioid receptors in the central and peripheral sympathetic pathways inhibit catecholamine release and modulate their effect on peripheral sympathetic afferents
- Morphine specifically aborts the allodynic response. If allergy to morphine, consider hydromorphone.

Antihypertensives

- If storming episodes are associated with severe hypertension and tachycardia, a nonselective β-blocker (i.e, labetalol or propranolol) should be initiated.
- Antihypertensives decrease catecholamine effects by nonspecifically blocking alpha and beta receptors.
- Clonidine stimulates alpha 2-adrenergic receptors in the brain resulting in reduced sympathetic outflow from the CNS and decreased peripheral resistance, renal vascular resistance, heart rate, and blood pressure

Gabapentin

- Centrally acting
- GABA agonist
- Treats spasticity and allodynic response

Bromocriptine

- Acts at the hypothalamic level to lower temperature threshold, diminish diaphoresis, & lower blood pressure
- Binds to D2 receptors to inhibit sympathoexcitatory structures and affect motor control
- Effects may be modest and delayed
- *Use caution in patients with epilepsy or at risk for seizures as may lower seizure threshold*

Dantrolene

- Added if contractures or persistent dystonia are noted
- Alternative to baclofen if baclofen produces oversedation
- Suppresses calcium release → Promotes relaxation of skeletal muscle
- May help control hyperthermia

References

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