



Management of Severe Paroxysmal Sympathetic Hyperactivity Following Hypoxic Brain Injury

Hipoksik Beyin Hasarı Sonrası Gelişen Paroksizmal Sempatik Hiperaktivite Tedavisi

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Dear Editor,

Paroxysmal sympathetic hyperactivity (PSH) is the autonomic dysfunction characterized by tachypnea, tachycardia, hypertension, hyperthermia, sweating and motor features such as dystonia. It is the leading cause of higher morbidity and mortality in an intensive care unit setting. The main etiologic factors of PSH are traumatic brain injury (79%), hypoxic ischemic encephalopathy (10%) and stroke (1, 2). Presence of hypoxia, diffuse axonal injury and younger age facilitates the development of PSH (3). Treatment of PSH is comprised of reducing sympathetic and motor hyperactivity. Opioids, gabapentin, benzodiazepines, bromocriptin, β -blockers and centrally acting α_2 -agonists are recommended (1, 4). We described our management for a case of severe PSH, which occurred after hypoxic brain injury, and orally administered baclofen is effective in motor symptoms of PSH, contrary to that reported before (1).

A 19-year-old boy with a history of hypoxic brain injury due to cardiopulmonary resuscitation after a thoracic penetrating trauma was accepted in our intensive care unit. He was unconscious and the Glasgow Coma Score was 5/15; thus, informed consent was obtained from his parents. He experienced episodes of increased body temperature ($>38.5^\circ\text{C}$), blood pressure ($>140/90$ mmHg), heart rate (>100 m^{-1}) and respiration rate (>35 m^{-1}) with decerebrate posturing and excessive sweating. Morphine 2 mg intravenous (IV), dexmedetomidine 1 μg kg^{-1} h^{-1} IV infusion and remifentanyl 0.5 μg kg^{-1} h^{-1} IV infusion were administered during episodes; symptoms were controlled for a while, but not totally. On the other hand, blood, tracheal aspirate and urine sample cultures were collected and appropriate antibiotics were administered. Blood leukocyte count, CRP and procalcitonin values decreased following antibiotherapy; however, fever could not be controlled despite antipyretic therapy (such as paracetamol and metamizole). Moreover, liver enzymes were

increased; therefore, antipyretic drugs were stopped and intravenous cooling was performed by the Alsius Coolgard 3000[®], USA. Fever was controlled, but tachypnea, tachycardia, hypertension and extensor contractions were persistent. In PSH cases that were managed in our clinic before, we observed limited episodes, including one extremity or one half of the body on the opposite side of the lesion and one half of the head on the lesion side. In this case, episodes occurred in the whole body, including four extremities and the head. We used β -blocker propranolol (160 mg d^{-1}) and α_2 -agonist clonidine (0.2 mg d^{-1}); thus, the frequency and the intensity of sympathetic episodes were diminished. It is suggested that baclofen cannot cross the blood-brain barrier efficiently when administered orally (5). Although it has been reported that oral baclofen is ineffective to treat motor spasticity in PSH, we observed that the intensity of spasticity was reduced from the Modified Ashworth Scale (MAS) 5 to MAS 2 following oral baclofen therapy (90 mg d^{-1}) (1, 5).

In conclusion, β -blocker treatment is efficient in cases that present only sympathetic hyperactivity. In severe cases like our patient, β -blockers, centrally acting α_2 -agonists and gamma-amino butyric acid (GABA)-B agonist baclofen should be incorporated in PSH therapy. We emphasize that oral baclofen administration could be effective to treat spasticity, contrary to the earlier reports.

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