

Seizure-induced demargination predicts peri-ictal respiratory decompensation

Jose L. Vega^{1,4}, Prabhu Emmady¹, Christina Roels², John Conforti³, Mehmet T. Dorak⁵, Catalina Ramirez⁶

¹Neurosciences and Stroke, ²Pharmacy, ³Critical Care, Novant Health, Forsyth Medical Center, ⁴TeleNeurologia SAS, ⁵School of Life Sciences, Pharmacy & Chemistry, Kingston University London, ⁶Dinamica-Grupo Sura

Objective:

To investigate whether seizure-induced demargination (SID) reflects the evolution of respiratory abnormalities during generalized tonic-clonic seizures (GTCS).

Background:

Apnea and airway obstruction can occur during GTCS, and can cause respiratory compromise. In addition, both prolonged apnea, and forceful chest excursions against a closed airway, can induce demargination responses. Thus, we hypothesized that GTCS that result in respiratory compromise also exhibit SID responses.

Design/Methods:

We investigated the frequency with which SID and seizure-induced respiratory compromise (SIRC) coexist in patients who presented to our hospital for acute treatment of GTCS between January 1, 2017 and August 23, 2018. Patients with evidence, or history, of recent infections, steroid use, or immune disorders were excluded. SID was defined as a postictal white blood cell (WBC) count $\geq 11,000$ cells/mm³. SIRC was defined as any of the following: 1) physical signs of peri-ictal respiratory distress, 2) objective evidence of peri-ictal hypoxemia (SpO₂ < 90% or PaO₂ < 60mm Hg), or 3) a requirement for peri-ictal intubation. We also analyzed earliest available post-ictal vital signs, and chest X ray (CXR) data.

Results:

SIRC was observed in 30.1% (46/153), and SID in 32.6% (50/153) of patients. SID was present in 71.7% (33/46) of SIRC patients, but only in 15% (17/107) non-SIRC patients (OR = 13.4; 95% CI = 5.9 to 30.7; P < 0.0001), suggesting that SID and SIRC stem from a common physiopathological response. Importantly, SID correlated with a higher postictal heart rate (106.7 \pm 4.0 vs 91.4 \pm 2.5; P < 0.0002), a higher peri-ictal intubation rate (28% vs 6.8%; OR = 13.7; 95% CI = 3.5 to 53.8; P = 0.0002), and a higher frequency of CXR abnormalities (53.2% vs 25.2%; OR 3.37; 95% CI = 1.6 to 6.8; P < 0.001) by comparison with non-SID patients.

Conclusions:

SID reflects an ictal physiopathological process that causes respiratory decompensation in a subset of GTCS.