

was performed which revealed diffuse brain edema and then at day 4 the patient was transferred to the ICU for further management under sedation with propofol, fentanyl and midazolam continuous infusion. Mannitol was also administered and sedation was withheld after 48 h to allow for assessment of the neurologic status. Myoclonus was evident within 15 minutes, sedation was reinitiated and valproic acid as a continuous infusion was added along with an increase of levetiracetam. The following days as guided by neurologic consultation, antiepileptic medication had to be titrated until day 23 due to persistent myoclonus activity up to levetiracetam 6g, valproic acid 4g, clonazepam 3mg, lacosamide 200mg and perampanel 8mg. He also received continuous infusion of 1.5mg/kg/h ketamine for 24 hours to allow for therapeutic levels of the accompanying treatment. Management of antiepileptic medication was based on clinical manifestations of myoclonus due to lack of portable EEG. Results: On day 23 of hospitalisation the patient status allowed for his transfer to the EEG laboratory where despite demonstrating myoclonus, this was not associated with epileptiform abnormalities in the EEG. The EEG performed was asymmetrical, with θ waves in the left hemisphere, lack of basal activity was recorded in the other hemisphere and myoclonic status epilepticus of cortical origin was excluded. Gradually, the frequency of myoclonus decreased and antiepileptic treatment was reduced to 2.4g valproic acid, lacosamide 100mg, clonazepam 1mg, perampanel 8mg and levetiracetam 4g. No ECG changes were recorded during the course of treatment. The patient died after 58 days of hospitalisation due to complications of sepsis. Conclusion: PHM can be a challenging phenomenon that occurs after cardiac arrest. EEG data may not be indicative of clinical manifestations while thorough, step by step titration is necessary to manage such demanding and persistent cases.

Cardiac conduction disturbances following high-dose antiepileptic therapy for refractory status epilepticus: a case report

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Background: The management of refractory status epilepticus (RSE) often requires high doses of sodium- channel blocking antiepileptics. While effective for seizure control, these agents, particularly phenytoin, are associated with significant cardiotoxicity. We report a case of severe cardiac conduction disturbances and hemodynamic instability following the administration of a multi-drug antiepileptic regimen. Methods: A 79-year-old female with a history of meningioma resection presented with focal-to-bilateral tonic-clonic SE. Initial management included 4g of levetiracetam, 20mg of diazepam, and 250mg of phenytoin. Due to persistent seizure activity, the patient was intubated and started on propofol and remifentanyl sedation. Upon Intensive Care Unit (ICU) admission, an additional dose of phenytoin was administered, and maintenance

therapy with levetiracetam (1g BID) was initiated. Empirical coverage with ceftriaxone and acyclovir was provided due to a recent history of a vesicular rash. Results: Within hours of ICU admission and following antiepileptic loading, the patient developed nystagmus and significant QTc prolongation, progressing to long QT syndrome accompanied by acute hemodynamic instability. Despite initial stabilization, the following day the patient manifested transient Mobitz Type II atrioventricular block. A temporary transvenous pacemaker was inserted; however, sinus rhythm was restored within the next hour. The patient was successfully weaned from mechanical ventilation and vasopressors, and subsequently transferred to the neurology ward. Maintenance therapy continued with phenytoin (125mg TID) and levetiracetam (1.5g BID). Regrettably, on the 38th hospital day, the patient died due to septic shock and multi-organ failure caused by a multidrug-resistant Gram-negative infection. Conclusion: This case highlights the critical need for continuous cardiac monitoring during the aggressive treatment of SE. The synergy of high-dose antiepileptics can trigger life-threatening arrhythmias and conduction blocks, even in patients without prior cardiac history. Clinicians must remain vigilant for iatrogenic cardiotoxicity, as these complications may necessitate invasive interventions.

Predicting the impact of mechanical ventilation on central venous pressure

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Background: Mechanical ventilation could affect the central venous pressure via changes in intrathoracic pressures. Objective: To predict the transmission ratio of the airway pressures changes on central venous pressure (CVP). Methods and analysis: A retrospective analysis of mechanically ventilated patients from the AmsterdamUMC ICU database was conducted. For each patient, variables describing mechanical ventilation (PEEP, DP, PPlat, I:E ratio, RR, VT, PAW mean) and CVP were extracted. These sub-sets were used to determine optimal combinations of input features for identifying nonlinear patterns. Exclusion criteria for the factors compromising the accuracy of central venous pressure measurements were applied. We applied XGBoost as a predictor, for its interpretable decision tree characteristics that captures non-linear relations well, as it harnesses methods to mitigate overfitting while training. It optimizes a global objective function combining a loss term and regularization to control model complexity. Using second - order gradient, tree splits are selected to maximize loss reduction while maintaining regularization. Trees are sequentially added with a learning rate to ensure stable model improvement and strong generalization. As a cross-reference means to study the generalization properties and mitigate overfitting with XGBoost, a two-stage predictor is set in a series of four ensemble based learners: XGBoost, LightGBM, CatBoost, and Random Forest to lend insights for change in performance and narrow the best performing method. Results and Discussion: The evaluation benchmarks