

Introduction :

Intractable status epilepticus not responding to conventional pharmacotherapy after residual tumor debridement and cranioplasty. Such refractory status epilepticus has led to the use of anesthetic drugs. Tumor location and histology influences the risk for refractory seizure. Clinically tumor related seizures manifest as simple or complex partial seizures with or without secondary generalization and in more than 50% cases are pharmacoresistant. When uncontrolled, tumor related epilepsy affects patient's quality of life, causes cognitive deterioration and may result in significant morbidity. Tumors involving frontal temporal and parietal lobes are more commonly associated with seizures than an occipital lesion. The frequency of seizure differs widely according to tumor type. Glioneuronal tumors such as gangliogliomas and dissembryoplastic neuroepithelial tumor are typically associated with chronic pharmacoresistant epilepsy in up to 90-100%.

Case Study:

A 38 year old male admitted in hospital for cranioplasty with p/h/o craniotomy for ganglioglioma. Patient was chronic alcoholic and had h/o of seizure disorder since 2012. Earlier he underwent craniotomy in march 2016. He was on tablet levetiracetam 500 mg BD. Sample biopsy of lesion was suggestive of active gliotic lesion. Its CT finding showed temporal gliotic area with central necrosis and hemorrhagic in nature. Patient underwent cranioplasty on 14/12/16. Two days later a witness GTCS was reported. Initially infusion medazolam was started @ 2mg/kg/hr. but focal partial seizures continued. Other antiepileptic drugs such as T. Levetiracetam 750 mg, T. Sodium valproate 500mg T Frisium 10 mg, and T Librium was added. The regimen was continued for next 2 days, but there was no reduction in frequency of seizures. Thereafter higher anesthetic drugs were introduced. Initially treatment started with infusion propofol @ 3mg/kg followed by inj Lorazepam 4mg IM 6 hrly. Infusion continued for next 48 hours. No relief achieved and partial focal seizure continued. Infusion propofol was stopped and Thiopental was added to the regimen @ 3mg/kg/hr for next 24 hours. Reduction in seizure frequency was observed. Infusion Dopamine was added to maintain hemodynamic stability. Post SOL resection was done on 21/12/16. There after thiopentone infusion was continued. Total reduction of seizures were observed. Slowly Thiopentone was tapered and stopped on 26/12/16. Infusion Medazolam was started as maintenance. No event of seizure was reported thereafter. Patient was extubated on 31/12/16 and was put on oral antiepileptic. With inj. phenobarbitone 100mg BD and inj. Levetiracetam 1gm TDS.

Discussion :

Thiopental is an intravenous anesthetic agent used for refractory SE. It is more commonly used in Europe for treating status epilepticus, is loaded at 2 to 4 mg/kg and then infused at 3 to 5 mg/kg/h. The half-life is 3 to 11 hours at serum levels < 30 mg/L, but is significantly prolonged up to 60 hours at higher serum concentrations. Hypotension appears to be most profound with barbiturate therapy. Once seizures have been controlled for 12-24 hours, continuous intravenous therapy should be gradually tapered off if the drug being administered is midazolam or propofol. Gradual tapering is probably not necessary with pentobarbital or thiopental sodium. During withdrawal of anaesthetic therapy, intravenous phenytoin/fosphenytoin or valproate should be continued (these agents having been administered during earlier phases of GCSE) to ensure an adequate baseline of antiepileptic medication so as to prevent the recurrence of status epilepticus. These agents have several inhibitory effects on lymphocyte and leukocyte functions causing increased infection rates in patients treated with barbiturate coma. Routine surveillance cultures of blood, urine, and sputum are recommended for patients on long-term therapy.

Conclusion :

We conclude that tightly controlled by serum levels and carefully monitored for therapeutic efficiency, initiating and tapering of Thiopentone infusion in the ICU setting with mechanical ventilation and hemodynamic monitoring will allow the physician to establish the therapeutic serum levels of anesthetic agents. It will help to reduce the relapse rate and to avoid the mortality and long term morbidity associated with this life threatening medical emergency.

References:

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