Anaesthetic management in a patient with Lennox-Gastaut syndrome

Sir,

Lennox-Gastaut syndrome (LGS), is an age specific childhood epileptic encephalopathy characterised by triad of multiple and frequent epileptic seizures resistant to anti-epileptic drugs, a characteristic electroencephalogram (EEG), psychomotor delay and behaviour disorder. It occurs between 2 and 8 years of age. Diagnosis requires assessment of both clinical and EEG features, to distinguish LGS from other childhood epilepsy syndromes. “Drop attacks” are common resulting in recurrent injury. About 60% of children progress to status epilepticus. Management options include anti-epileptic drugs, ketogenic diet, surgery (corpus callostomy) and vagus nerve stimulation.

A 7-year-old (21 kg) patient was brought to the emergency room with fracture of distal radius and ulna. A fixation was planned. The parents gave a history of LGS, indicating that she had multiple seizures in a day. The fracture was due to a fall during a “drop attack.” Her current anti-epileptic medications included valproic acid and lamotrigine 3 times a day. The morning dose of the drugs had been already administered to the patient. Previous investigations showed an unremarkable magnetic resonance imaging brain, biotinidase deficiency, and a positive 2,4-dinitrophenylhydrazine test. All other metabolic tests and routine lab investigations were normal. The EEG showed a frequent paroxysmal spike and wave discharges of <2.5 Hz.

An intravenous (IV) access was secured with the patient in her mother’s lap. Pre-medication of 1 mcg/kg of fentanyl and 0.5 mg of midazolam helped separation from her mother. Routine monitors were setup and the patient induced with 5 mg/kg of thiopentone sodium. Anaesthesia was maintained throughout the 90 min of the surgery with sevoflurane, oxygen and nitrous oxide mixture on spontaneous ventilation through a laryngeal mask airway with a Jackson-Rees circuit. Incremental dose of 0.5 mcg/kg of fentanyl were administered 60 min into the surgery. After the surgery, the paediatric consultant requested that the patient be taken for her routinely scheduled EEG. Since the patient had regained consciousness in the post-anaesthesia care unit a bolus of 0.5 mcg/kg of dexmedetomidine was given to calm her down. The patient was then transported to the EEG room with the supporting monitoring equipment on an adequately padded transport trolley. On reaching the EEG room a continuous infusion of 0.3 mcg/kg/h of dexmedetomidine was established. The EEG was recorded uneventfully. The patient was observed in the post EEG recovery room until awake and then released to her parents. During the entire process, she had no obvious convulsion and post-operative period was uneventful.

Concerns for the anaesthesiologists in such cases include: (i) ability of the anaesthetics to modulate or potentiate seizure activity; (ii) interactions of anaesthetic drugs with anti-epileptic agents; (iii) perioperative care of epileptic patients; (iv) associated co-morbid conditions. (v) difficult
IV access due to contractures, (vi) communication difficulties due to mental regression (vii) airway abnormalities due to long term use of anti-epileptic drugs.

Many anaesthetic agents can be pro-convulsant or anticonvulsant or both. Some anti-epileptic drugs especially phenobarbital and carbamazepine may potentiate the effects of anaesthetics.

Thiopentone was used for induction as it is safe for induction of anaesthesia, as well as treatment of status epilepticus. Among IV anaesthetics etomidate, ketamine, methohexital and local anaesthetics have documented EEG evidence of epileptogenic activity in non-epileptic individuals.

Amongst inhalational agents, enflurane and occasionally sevoflurane have documented EEG and clinical evidence of epileptogenic activity in non-epileptic individuals. Desflurane, isoflurane, halothane and N\textsubscript{2}O are considered as safe. We used sevoflurane for maintenance which is well tolerated in terms of airway complications (causing minimal breath-holding, coughing, excitement or laryngospasm) and is an agent of choice for induction and maintenance of anaesthesia in paediatrics. The maximal concentration was limited to $<1.5$ minimum alveolar concentration with the use of adjunctive agents like nitrous oxide and opioids which ensured early and complete awakening which was essential for a rapid examination of neurological status and also reduced the risk for regurgitation and aspiration, and the sympathetic response to extubation and emergence. Adequate post-operative analgesia was ensured to avoid cerebral excitation which may be a potential trigger for a convulsive episode.

For procedural sedation like EEG, trichlorphos is commonly used in the paediatric age group to induce sleep, has anticonvulsant property and is considered as safe, but has a risk of respiratory depression and hypoxemia.

In this case, we used dexmedetomidine, an $\alpha$2 agonist, sedative, anxiolytic and analgesic showing a pattern similar to stage II sleep with no respiratory depression. Although studies examining the effect of dexmedetomidine on EEGs have been documented, its effect on EEG in patients with LGS has not been documented. Dexmedetomidine has an effect on EEG frequency, spectral power as well as spike activity. Bolus injection of high dose dexmedetomidine (100 mcg/kg) exerts pro-convulsant effects during anaesthesia with volatile anaesthetic, due to increased concentration. On the other hand, target controlled infusion did not show any pro or anticonvulsant activity, allowing an adequate mapping of epileptic foci. Thus, we could manage a patient of LGS for non-epileptic surgery under general anaesthesia with sevoflurane and EEG with dexmedetomidine, a uniquely useful agent with early and complete awakening.

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