**Results:** During the course of disease, 21 of 46 children with PME (45.65%) experienced total of 50 episodes of SE. The etiology was: neuronal-ceroid lipofuscinosis (7), Gaucher disease 2 (2), Nieman-Pick C (4), mitochondrial disorders (4), Lafora disease (2), Krabbe disease (1), KCNC1 (1). The age was 0.2-18 (mean 8.4) years. Nine patients experienced SE during the first year of disease, and in four cases, SE was the first epileptic event. All episodes had prominent motor symptoms: convulsive (25), myoclonic (13), focal including epilepsia partialis continua (12). Response to the treatment was variable, with common side effects. Most effective drug was midazolam in intravenous infusion with mean dosage of 0.35 mg/kg/h. The artificial ventilation was necessary in 7 episodes, in 4 together with circulatory support. Refractory SE was in 62% episodes, including nine SRSE. Recurrence rate was nearly 50%.

**Conclusion:** Children with PME frequently experience SE. Episodes are mostly convulsive, refractory to AEDs with high recurrence rate. SE appearance in later phase of disease contribute to prominent drug adverse effects. Managing SE in children with PME is challenge and requires rational approach in order to stop the seizure, and, on the other hand, to prevent side effects and worsening of general and neurological patient`s condition.

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### Refractory and super-refractory Status epilepticus- analysis of etiological factors

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**Background:** Status epilepticus (SE) can be a life-threatening condition associated with multiple complications, including death, and can progress to refractory and super-refractory SE. Treatment guidelines recently published partially addresses to the treatment of refractory and super-refractory SE.

**Materials and Methods:** We conducted a retrospective study of patients diagnosed as having SE- for a period of 2 years (01/01/2017-12/31/2018), according to the clinical presentation and EEG findings, also who had appropriate instrumental diagnosis included cerebral neuroimaging with CT and MRI to identify which condition are leading to refractoriness. The following data were analysed: age, sex, SE clinical type (convulsive and non-convulsive (NCSE)), neurological presentation, EEG features, etiology according to instrumental findings, neuroimaging study (brain CT and MRI), and/or blood examinations, and response to staged applied treatment protocol.

**Results:** Data are presented in raw numbers, data were analysed using Excel, student's t-test, we identified 78 patients, 29 of whom were female and 49 male, with a mean age of 42.2 $\pm$ 18.4 years. Regarding clinical SE type, convulsive SE was observed in 68 patients and non-convulsive in the remaining 10/78. As regards the SE type according to age patients with convulsive SE were older than patients with NCSE. According to EEG – focal SE was in 49 cases and generalised in 29, no difference for gender, for age focal SE more characteristic for older. Lesional SE was in 52 cases, toxic-dysmetabolic in 11 patients, anoxic SE in 3 and AED non- adherence in 12 from 78 patients. The oldest patients were those with lesional SE due to posttaumatic injuries, poststroke, brain metastasis, neurosurgical interventions followed by patients with non-adherence and toxico-dysmetabolic. Also it was observed among young patients the predominance of infectious and autoimmune underlying etiology (autoimmune/ viral encephalitis), and this cases proven to be refractory to first and second- line AEDs treatment (6/10 patients with refractory SE). 22 of patients have complete SE regression after being treated with benzodiazepines, 46 needed administration of second-line drugs like- phenytoin and phenobarbital and 10 patients – 12,8 % required anesthetic drugs to control the epileptic activity (3 patients- 3,8 % developed super-refractory SE (2 case herpetic encephalitis and 1 case anoxic brain injury).

**Conclusion:** Predictors of refractory status epilepticus were new diagnosis of SE and nonconvulsive SE. The etiology of refractory status epilepticus appears to be similar overall to that of nonrefractory status epilepticus, but more likely associated with encephalitis (viral encephalitis, in particular) and hipoxico-anoxic brain injuries. We also can conclude that good adherence to staged treatment as well as treatment of the underlying etiology is the key to success in controlling refractory seizures.

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### Long term follow-up of recurrent Status Epilepticus and Stroke-Like Episodes in a MELAS family

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**Background:** Mitochondrial encephalomyopathy, lactic acidosis and stroke-like episodes (MELAS) is a disorder commonly caused by the A3243G/tRNALeu mutation of mitochondrial DNA (mtDNA). MELAS patients are at high risk of developing status epilepticus (SE) during stroke-like episodes (SLEs). We describe the long-term follow-up of 2 affected members of a MELAS family with recurrent focal SE associated with SLEs. SE treatment is discussed.

**Method:** A complete clinical work-out including clinical, biochemical, neuroradiological and EEG assessment was performed over the years in both patients.

**Results:** The mother developed since 33 years of age focal epilepsy (auditory and visual simptoms, hemiclonic seizures), sensorineural deafness, migraine, intestinal dysmotility, severe cognitive impairment, right homonymous hemianopsia and hemiparesis. She presented recurrent parieto-occipital SE associated with SLEs leading to death at 40 years of age.

Her son suffered photosensitive epilepsy since 17 years of age. He presented 7 episodes of occipital SE (elementary visual hallucinations and oculo-clonic seizures) associated with hemianopia, lactic acidosis and parietal-occipital SLE and refractory epilepsia partialis continua (EPC) in one occasion. SE became progressively more difficult to treat and complicated by lactic acidosis and rhabdomyolysis. During one SE propofol was used and the patient suffered multiple organ failure (propofol infusion syndrome-PRIS). Iv high-dosage midazolam was the most effective treatment of SE.

Both patients carried the mtDNA A3243G/tRNALeu mutation with a similar degree of heteroplasmy (80%).

**Conclusion:** We report the long term follow-up of 2 members belonging to a MELAS family with recurrent SE and SLEs. SE became refractory to treatment in both patients leading to death in the mother. Based on the occurrence of PRIS and evidence of mitochondrial toxicity,

we suggest to avoid the use of propofol for SE treatment in patients with mitochondrial encephalopathy. Midazolam is well tolerated and is a therapy of choice for SE in MELAS.

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## Post-anoxic status epilepticus: A review of our experience in the last five years

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**Background:** Status epilepticus (SE) after cardiac arrest (CA) is associated with high mortality despite an appropriate antiepileptic treatment. The aim of our study is to determine which factors could contribute to a better prognosis in this patients, taking into account the duration of the status, EEG pattern and drugs used.

**Methods:** We retrospectively analysed our patients who suffered SE following CA in the last five years (2013-2018). EEG background patterns of SE, duration of SE, presence and characteristics of clinical seizures and use of antiepileptic drugs (AD) were assesed. Outcome was evaluated using the Cerebral Performance Category (CPC).

**Results:** We collected 34 patients admitted to the ICU after CA who presented anoxic encephalopathy. 14 patients (41.17%) developed a myoclonic status after finishing therapeutic hypothermia and onset of awakening. All were treated with a combination of AD, including valproic acid, levetiracetam, anesthetics (midazolam and propofol), occasionally phenytoin, and in two cases, ketamine and lacosamide. Five patients presented a flat encephalographic pattern while six patients showed continuous generalized paroxysmal discharges. Two patients survived (CPC: 2 and 3, respectively). Median survival of the deceased patients was 14.4 days.

**Conclusions**: In post-anoxic encephalopaty, SE is a frequent complication and it normally correlates with poor outcome. A continuous EEG and multimodal prognostication approach to adapt pharmacologic treatment would be necessary in order to improve the prognosis.

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## Eucalyptus Oil Ingestion Induced Status Epilepticus: A Short Case Series

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**Introduction:** Eucalyptus oil is an essential oil derived from leaf of Eucalyptus tree and is widely used as an over the counter remedy for common ailments. Here we report a small case series of 3 adults with Eucalyptus oil induced status epilepticus.

**Case Presentations:**

**Case 1**

24-year-old young man was admitted to the critical care intensive care unit of our hospital with status epilepticus of unknown cause. History was clarified from father who narrated the following incident. He (Father) had bought a bottle of eucalyptus oil at home few weeks back. As the bottle was leaking he transferred the contents from the eucalyptus bottle to another empty cough syrup bottle. His son who was apparently healthy came home in the afternoon and drank 5 ml of the liquid (Eucalyptus oil) from the cough syrup bottle as he was having some cough and cold from the morning. 5 min after drinking the syrup he had an episode of generalized tonic-clonic seizures and had multiple episodes after that. He was taken to a near by hospital for the initial treatment and was later transferred to our hospital in a critical state. In our critical care unit he was intubated and was treated for status epilepticus with lorazepam, phenytoin, levetiracetam and midazolam infusion. His CT Brain showed diffuse cerebral edema while EEG showed diffuse slowing in the delta range. He developed multiorgan dysfunction and died on the 5$^{th}$ day of admission.

**Case 2**

31-year-old man used to take few drops of eucalyptus oil mixed with water occasionally for abdominal pain for the past 3 years. He drank 2-3 tea spoon full (10-15 ml) of Eucalyptus oil for abdominal pain and had multiple episodes of generalized tonic clonic seizures, 20 minutes after the intake. He was taken to a near by hospital and was treated with intravenous antiepileptic drugs and was discharged after 5 days on Tab levetiracetam 500 mg bid. His hematological, biochemical and neurological investigations were unremarkable.

**Case 3**

38-year-old man was a case of post traumatic occipital lobe epilepsy and was well controlled on Tab phenytoin 300 mg per day. He had headache and cold for which he applied various balms and oils containing eucalyptus and camphor and had multiple episodes of visual aura in the form of flashes of light with altered sensorium on going for hours suggestive of a complex partial status. His MRI brain showed occipital gliosis and EEG showed slowing from the occipital regions. He was discharged with an advice to avoid these essential oils which provoke seizures.

**Discussion:** Essential oils like eucalyptus have epileptogenic potential which is rarely recognized by public and physicians. These essential oils are kept in the houses in open places within the reach of everyone including toddlers as they are generally perceived as safe. Here we have described three cases of Eucalyptus oil induced status epilepticus in three young adults. The young man who expired following status epilepticus and multiorgan dysfunction had consumed it accidentally while others took as remedy for abdominal pain and upper respiratory tract infection. The epileptogenic properties of plant derived essential oils are known for centuries but public and physicians are equally ignorant of these serious complications. The essential oils which are epileptogenic are eucalyptus, camphor, thuja, sage, spike lavender and terpentine. 1 The route of exposure, the type and amount taken all may be important in causing these complications. The essential oil of eucalyptus and camphor are the ones which are commonly used and abused. These contain an aromatic monoterpene called 1,8 Cineole, which is epileptogenic compound. 1,8 Cineole has a mechanism of action similar to known anti- convulsant pentylenetetrazole. 2 Although there have been no explicit studies outlining the mechanism by which eucalyptus oils can precipitate seizures, studies on rat models show it may be secondary to loss of tissue sodium/potassium gradient leading to increased cellular hyperexcitability. 3 In cases of so called de novo status epilepticus exposure to essentials oils need to be sought. Public and physicians should be made aware of the epileptogenic potential of these essential oils.

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