WEBTABLE I TREATMENT OF ONGOING COMPLICATIONS AND PRECIPITATING FACTORS

Condition	*Management*	*Comments*
Hypoglycemia	If present or if blood glucose cannot be measured, dextrose boluses with 2ml/kg of 25% dextrose, maintain blood glucose >110 mg/dL [12]	Both hypoglycemia and hyperglycemia are deleterious to brain during SE. Hyperglycemia with resultant lactic acidosis may cause neuronal injury [13]
Hyperglycemia	During the initial compensatory phase (due to excess catecholamines) may normalize with control of seizures. If it is persisting, insulin infusion is required to maintain the blood glucose in the range of 140-180 mg/dL[12]	
Hypocalcemia	IV calcium gluconate: 1 mL/kg of 10% calcium gluconate diluted in 1mL/kg of 5% dextrose; infuse over 20-30 min under close cardiac monitoring	Hypocalcemia in the context of SE should be treated with IV calcium
Hypomagnesemia	Magnesium: IM or slow IV at dose of 50-100 mg/kg per dose	If present, may make hypocalcemia refractory to treatment
Fever	Intravenous/intramuascular or rectal anti-pyretics. Other method of reducing temperature quickly is infusion of refrigerator-cold saline (4^0C) boluses (20 mL/kg), although its effect is short-lived (30 minutes) [15]	May be due to continuing seizures, or infections (either CNS infection or nosocomial infections). Uncontrolled, it may exacerbate neuronal injury [14]
Hypernatremia	o Rapid fluid resuscitation is the first step, fluid of choice is normal saline; gradual correction is calculated based on standard formulas. o Hyperglycemia is often associated with hypernatremia, and requires treatment by change of IV fluids to dextrose-free solutions and/or insulin infusions	
Hyponatremia	o Rapid correction should be avoided except in cases with rapidly developing hyponatremia causing seizures and acute encephalopathy o In symptomatic acute hyponatremia, boluses of hypertonic saline (3%) should be given at 1ml/kg over one hour, and repeated till serum sodium reaches at least 125 meq/L o In chronic hyponatremia, the correction has to be more gradual (over at least 48 h, and at rate of not more than 10-12 meq/L per day)	Causes include mannitol infusions, capillary leak associated with sepsis, SIADH, excessive diuretics usage, and external losses (diarrhea and vomiting)
Hypotension and cardiac dysfunction	o Early inotrope support with dobutamine or milrinone should be started in cases with cardiac-associated hypotension [12] o In patients with cardiomyopathy or arrhythmias, invasive monitoring of blood pressure and central venous pressure for accurate fluid balance and titration of inotropes	
Metabolic acidosis	o pH <7.2: Treat with fluid boluses and maintain systemic perfusion, along with aggressive control of seizures; it is infrequently associated with life-threatening arrythmia [23]. o More severe acidosis (pH <6.8), Sodium bicarbonate [12].	Due to excessive muscle contraction, hypovolemia, distributive shock (with barbiturates), and propylene glycol toxicity [12,21,22]
Cerebral edema	o No benefit of empirical mannitol or corticosteroids in children with SE in the absence of features of raised intracranial pressure (ICP) o Clinical features of raised ICP (decorticate posturing, hypertension associated with bradycardia, irregular breathing): Mannitol 5ml/kg bolus dose, followed by 2-3 mL/kg 6 hourly; in children with hypotension, 3% saline can be used at rate of 1mL/kg/hour till serum sodium reaches to 165 meq/L	Recent experimental studies have suggested the possibility of vasogenic edema mediated by tumor necrosis factor-alpha (TNF-α) and endothelin-1 with resultant breakage of blood-brain barrier (BBB) during SE [26].
Infection	Appropriate culture specimens should be obtained, and empiric antibiotics should be started in those with high suspicion of nosocomial infection	Risk of infections is increased in children with SE especially those with prolonged SE.