Case Report

Recurrent seizures after lidocaine ingestion

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Abstract

Lidocaine has a concentration-dependent effect on seizures. Concentrations above 15 μg/mL frequently result in seizures in laboratory animals and human. We report a case of central nervous system (CNS) lidocaine toxicity and recurrent seizure after erroneous ingestion of lidocaine solution. A 4-year-old boy presented to the Emergency Department of Imam Hospital of Sari in December 2013 due to tonic-clonic generalized seizures approximately 30 min ago. 3 h before seizure, his mother gave him 2 spoons (amount 20–25 cc) lidocaine hydrochloride 2% solution instead of pediatric gripe by mistake. Seizure with generalized tonic-clonic occurred 3 times in home. Neurological examination was essentially unremarkable except for the depressed level of consciousness. Personal and medical history was unremarkable. There was no evidence of intracranial ischemic or hemorrhagic lesions in computed tomography scan. There were no further seizures, the condition of the patient remained stable, and he was discharged 2 days after admission. The use of viscous lidocaine may result in cardiovascular and CNS toxicity, particularly in children. Conservative management is the best option for treatment of lidocaine induced seizure.

Key words: Lidocaine, lidocaine ingestion, recurrent seizures

INTRODUCTION

Lidocaine belongs to the amide group of local anesthetics. It is also used as a type 1b antiarrhythmic agent. It is rapidly absorbed from the gastrointestinal tract with peak plasma level within 30–60 min. After ingestion, lidocaine undergoes extensive first-pass hepatic metabolism with a bioavailability of about 35%.[1] This may cause a false impression of the general safety of its use as an oral agent. Published reports of toxicity of lidocaine largely involved parenteral therapy.[2]

Lidocaine has a concentration-dependent effect on seizures. At lower concentrations, it has anticonvulsant properties, whereas concentrations above 15 μg/mL frequently result in seizures in laboratory animals and human. Seizures induced by lidocaine in experimental conditions invariably start in the amygdala. Despite the clear focal onset in these experimental models, the seizures emerging in patients given intravenous (IV) lidocaine are almost invariably generalized and without any clear signs of focality. Given the prevalence of partial seizures and the frequent use of lidocaine, a higher incidence of partial seizures would be expected with its use.[3]

It has a narrow therapeutic index, with only a slight difference between therapeutic and potentially toxic concentrations. Lidocaine toxicity is dose-related and proportional to its plasma level.[4] Although neurologic toxicity has been frequently observed with IV use, it has also been reported for topical use.[5]

Here, a case of central nervous system (CNS) lidocaine toxicity and recurrent seizure after erroneous ingestion of lidocaine solution is reported.

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CASE REPORT

A 4-year-old boy with 25 kg weight presented to the Emergency Department of Imam Hospital of Sari in December 2013 due to tonic-clonic generalized seizures approximately 30 min ago. 3 h before seizure, his mother

mistakenly gave him 2 spoons (amount 20–25 cc) lidocaine hydrochloride 2% solution instead of pediatric gripe. Seizure with generalized tonic-clonic occurred 3 times in home. On arrival at our Emergency Department, she was agitated and had confused speech with Glasgow coma scale score of 14/15. His initial blood pressure was 90/55 mmHg with a pulse rate of 130 beats/min.

His temperature was 36.9°C axial. Respiratory rate was 20 per min with a SpO_2 of 96% on 2 L of nasal oxygen. Twitching movement with muscle spasm was noticed in the limbs. The pupils were 4 mm in diameter, equal and reactive, and no focal neurological deficit was found. His skin was normal. Bowel sound was normal, and the bladder was not palpable. An electrocardiogram (ECG) showed sinus tachycardia with a heart rate of 139 beats/min, and QT interval was 0.89 s. Neurologic examination was essentially unremarkable except for the depressed level of consciousness. The personal and medical history was unremarkable; including the absence of birthing trauma or febrile seizures. He had no history of use of other drugs.

Laboratory studies while the patient received supplemental oxygen included an arterial blood gas with pH 7.30; pO_2 104 mm Hg; pCO_2 39 mmHg; and HCO_3 21. A complete blood count revealed white blood cells 10,400/mm^3 (31% polymorphonuclear leukocytes, 64% lymphocytes, 5% monocytes); hemoglobin 12 g; and hematocrit 35%. Blood chemistry studies were unremarkable except for an elevated glucose (219 mg %) obtained while the patient was receiving an IV line with 5% glucose. One day after admission; a repeat glucose level was 89 mg%.

Shortly after arrival at the Emergency Department, he developed one episode of brief generalized convulsion with upward gaze and episode lasting for around 30 s, 5 mg of IV diazepam was administered and a contrast-enhanced computed tomography (CT) scan of the brain was promptly performed. The latter showed no evidence of intracranial ischemic or hemorrhagic lesions. Serum lidocaine concentration 30 min after the onset of seizures was 5.1 μg/ml. There were no further seizures; the patient remained stable and was discharged 2 days after admission.

DISCUSSION

Lidocaine has a rapid onset and is effective for about 30–60 min in its plain form and up to about 90 min in combination with a vasoconstrictor. Therefore, most of the signs and symptoms of poisoning with lidocaine start about 10–25 min after administration.[6] Almost all types of local anesthetics including lidocaine are toxic to nerve cells. The first symptoms and signs of local anesthetic toxicity are usually neurological with numbness of the mouth and tongue. Shortly afterward, there is the onset of tinnitus, lightheadedness, numbness, disorientation, confusion, auditory and visual disturbances lethargy and potentially, coma, apnea, and cardiovascular collapse (CC).[7] Cardiovascular toxicity usually reveals with tachycardia and hypertension; but, with increasing toxicity, bradycardia, and hypotension occur. Ventricular arrhythmias and cardiac arrest may also occur. Local anesthetic toxicity is extremely rare in neonates, infants, and children.

Recurrent seizures have been previously reported in a neonate where IV lidocaine was given in conjunction with midazolam and atropine for the preoperative intubation.[8] Therefore, it seems that prolonged and recurrent seizures after lidocaine administration for ingestion have not been previously explained. It has been reported that seizures happen when the dose of lidocaine administered exceeds 3 mg/kg.[6,7] However, some cases of seizure following administration of recommended doses of lidocaine have also been reported.[8] Most of these seizures are self-resolving or resolve with conventional anticonvulsants. They may be dangerous because of the acidosis and hypoxia due to a prolonged duration and resulted in hypoxic-ischemic encephalopathy.

Oral ingestion of 5–25 ml of 2% viscous lidocaine has resulted in the seizure in children.[9] Thus, CNS toxicity provides a warning sign to cardiac toxicity. Early recognition of potential cardiac toxicity is important because if it goes unrecognized for any interval, it is more difficult to resuscitate. The earliest signs of cardiac toxicity are ECG changes which include prolonged PR, QRS, and QT intervals. Subsequently, it may progress to bradycardia, atrioventricular and intraventricular blocks, ventricular dysrhythmia, and CC that are often refractory to treatment. The level causing CC to CNS toxicity ratio (CC/CNS) has been estimated in animal studies. Local anesthetics with higher potency and lipophilicity like bupivacaine have a lower CC/CNS ratio as compared with that of lidocaine (3 vs. 7). Therefore, bupivacaine toxicity can occur with few premonitory symptoms of CNS excitation.[10]

The mainstay of management in lidocaine or other local anesthetic toxicity is supportive treatment to prevent hypoxia, acidosis, and hyperkalemia which may enhance the toxicity, especially cardiac toxicity. Gastric decontamination is of limited value because of rapid absorption after ingestion. Benzodiazepine and barbiturates can be used to control local anesthetic-induced seizure. However, they may exacerbate circulatory and respiratory depression. Propofol was found to be as effective as thiopental in the treatment of bupivacaine-induced seizure in rats and has also been used successfully to stop the seizure in patients with lidocaine toxicity.[11] However, it should be used cautiously in patients with local anesthetic toxicity as it typically lowers blood pressure and may cause significant Bradydysrhythmias and even a systole. Therefore, it is contraindicated in

local anesthetic toxicity with cardiovascular compromise. It is also proposed that propofol's effect in bupivacaine toxicity may be due to the lipid component of the propofol preparation in which 10% lipid is present.[12]

CONCLUSION

The use of viscous lidocaine may result in cardiovascular and CNS toxicity, particularly in children. Clinicians must be aware of the potential toxicity of topical or oral lidocaine and carefully review dosage schedules and potential toxicity with parents as well as considering alternate therapeutic modalities. Seizures in a patient suffering from erroneous oral ingestion should encourage parents and physicians to consider the possibility of lidocaine use and toxicity. Lidocaine-induced seizures are a warning sign for subsequent cardiac toxicity which can be lethal. Conservative management is the best option for treatment of lidocaine-induced seizure.

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