



Accidental Ingestion of 2% Viscous Lidocaine Hydrochloride Leading To Toxicity: A Case Report

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Abstract

For the past 50 years, local anesthetics such as lidocaine hydrochloride have been commonly used in various clinical settings. Its use is not just limited to anesthesia and surgery but is also frequently utilized in internal medicine and in primary care setting for bedside procedures. Our case demonstrates a successful resuscitation in a 30-year-old female with systemic lidocaine toxicity after accidental ingestion of 2% viscous xylocaine.

Keywords: lidocaine-induced systemic toxicity, local anesthetic toxicity, intravenous lipid emulsion

INTRODUCTION

Local anesthetics are widely used in everyday practice. Their application ranges from use in outpatient medicine clinics to emergency departments and operation theaters.⁽¹⁾ Lidocaine is an amide-type local anesthetic and a class Ib antiarrhythmic agent. Systemic exposure to large amounts of it leads to adverse effects on the central nervous systems (CNS) and cardiovascular systems (CVS). Parenteral form of lidocaine is the best-known source of poisoning, but poisoning could also happen with topical spray formulation.⁽²⁾ We present a case of lidocaine-induced systemic toxicity in 30-year-old female after accidental ingestion of 2% viscous xylocaine posted for an elective procedure. We also intend to create an understanding on the use of intravenous 20% lipid emulsion as the treatment of choice to reverse the symptoms.

CASE REPORT

A 30 year old female k/c/o Atrial septal defect (ASD) admitted under cardiology in Heart command center for ASD device closure under local anaesthesia. Prior to procedure patient was asked for xylocaine gargles for insertion of transesophageal echocardiography (TEE) probe. Pt had accidentally ingested approx 170 ml of 2% of viscous xylocaine. After 30 mins of ingestion patient became unconscious followed by tonic clonic generalized seizures. Anesthesia call was sent immediately, meanwhile patient was intubated by cardiologist, and intravenous midazolam 2mg and levetiracetam 1gm loading dose given followed by gastric lavage.

On reaching the heart command center, we observed patient was unconscious, intubated on mechanical ventilation, her Glasgow Coma Scale (GCS) was 5/15, blood pressure (BP): 72/48 mmHg, pulse rate (PR): 86/minute, auxiliary temperature (TA): 37.7°C, and respiratory rate (RR): 14/minute and pupils were mid dilated and sluggishly reactive. Oxygen saturation was 97%.

Patient was given bolus of 250 ml crystalloid for hypotension and started with 75 ml (1.5 ml/kg) of 20% lipid emulsion bolus followed by infusion of 0.25 ml/kg/min. Arterial Blood Gas (ABG) showed severe metabolic acidosis of PH- 7.023, PCO₂- 51.8 mmHg, PO₂- 344 mmHg, HCO₃⁻- 12.8 mmol/L, SO₂- 99%. Correction was given accordingly. Chest Xray, electrocardiography (ECG), and brain Computed Tomography Scan (CT scan) were advised. In her past medical history, she had no underlying diseases like as epileptic disorders, any drug history or co-ingestion.

Neurology call was sent which advised for Midazolam infusion 0.1 mg/kg/hr and samples for Calcium / Magnesium with routine investigations for complete blood count (CBC) / liver function test (LFT) / kidney function test (KFT) were sent.

Patient became conscious but drowsy after 2 hours and was put on CPAP mode with pressure support of 8 and Fio₂ of 40% and subsequent ABG were done to look for increase in met Hb and acidosis.

The treatment was supportive and symptomatic. No dysrhythmia was detected during hospitalization. She remained seizure-free and was successfully extubated the next day and transferred to the ward.

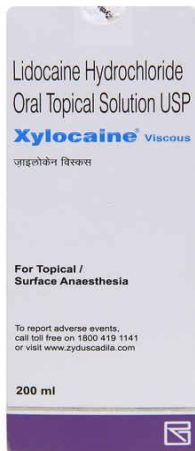


Figure 1:Xylocaine Viscous

DISCUSSION

Lidocaine hydrochloride has become the most commonly used local anesthetic in the outpatient setting. According to a report published in 2013 by the US Poison Control Center, there were 1454 reports of lidocaine exposure with five cases ending up as a fatality.⁽³⁾Xylocaine viscous 2% contains 20 mg of lidocaine hydrochloride and is administered topically.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.⁽⁴⁾The rate of absorption of local anesthetic agents following topical application occurs most rapidly after intratracheal administration. Lidocaine toxicity is dosage-dependent and directly relative to its plasma concentration.⁽⁵⁾

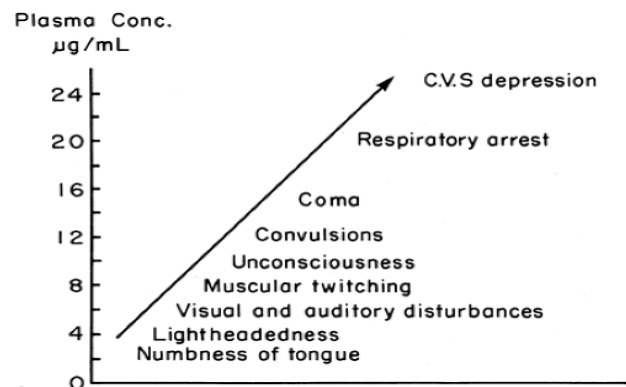


Figure 2: Local Anaesthetic Systemic Toxicity

A plasma level of 8-12 mg/L and above is associated with central nervous system(CNS) and cardiotoxicity.Hamedaminiahidashti et al, demonstrated a case study on 4 year old presented with 3 episodes of seizure after his mother mistakenly gave him 2 spoons (amount 20–25 cc) lidocaine hydrochloride 2% solution instead of pediatric gripe. Neurological examination was essentially unremarkable except for the depressed level of consciousness. 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, was managed conservatively and was discharged 2 days after admission.⁽⁶⁾In another study by Zuberi et al a young medical student developed severe toxicity, including seizures, respiratory distress, hypotension, and asystole, and died after gargling with 20 ml 4% lidocaine solution (800 mg of the drug) planned for upper gastrointestinal endoscopy.⁽⁷⁾

There are cases of ingestion of 5 to 25 mL of lidocaine (2%) in children which resulted in seizure.⁽⁸⁾ In another report, accidental ingestion of 30 mL topical lidocaine (1.2g) in a 74-year old woman caused CNS toxicity.⁽⁹⁾ In our case, hypotension, seizure was an

important clinical symptom after accidental ingestion of 2% viscous lidocaine. In spite of ingestion of high amount of lidocaine, no cardiovascular effects were detected. This may be due to the route of exposure. Lidocaine undergoes extensive first-pass hepatic metabolism with a bioavailability of about 35% via oral administration which can produce a low blood level of lidocaine. Unfortunately, lidocaine blood level could not be determined

in this case and it is the main limitation of this study.

CONCLUSION

Systemic toxicity of lidocaine hydrochloride on different concentration can be life-threatening. The rapid identification of clinical symptoms and administration of intravenous lipid emulsion is key to prevent mortality.

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