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Title of the Article: STATUS EPILEPTICUS FOLLOWING LOCAL ANAESTHESIA IN A PREVIOUSLY HEALTHY INFANT.

Short Title: STATUS EPILEPTICUS FOLLOWING LOCAL ANAESTHESIA.

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Background: Lidocaine Hydrochloride is a widely used local anaesthetic agent in almost all medical specialties that is well tolerated. But Systemic or local anaesthetics are studied to have proconvulsant activities and seizures could occur as a side effect even with local anaesthesia.

Case presentation: I present the case of a 6-and-half-month-old infant who developed intermittent tonic–clonic convulsions immediately after the instillation of local anaesthetic agent before performing incision & drainage of an abscess. Naranjo probability scale categorized the relationship between the drug and the event as probable. The patient was stabilized and referred to higher centre.

Conclusion: This case is reported to acquaint medical practitioners to possibility of systemic toxicity by local anaesthesia although uncommon; so that such complications can be avoided or managed promptly if occurs, to optimize patient outcome.

Keywords: Status epilepticus, Local anaesthesia, Infant, India.

Introduction

Local anaesthetics are substances that produce a reversible loss of sensation or analgesia when applied to body tissues. Local anaesthesia is used widely in most branches of medicine and is generally considered to be safer than general anaesthesia.[1]

Lidocaine is the most common local anaesthetic used in almost all medical specialties.[2,3] It is in the class of the local amide anaesthetics, which, compared to the ester-type local anaesthetics, is usually well tolerated with only rare occasions of allergic reactions.[4]

Systemic toxicity to local anaesthetics are extremely rare.[5,6] However, seizures, dysrhythmias, cardiovascular collapse, and transient neuropathic symptoms have been reported.[5,7,8] Infants have a much higher free serum concentration of local anaesthetics than older children and adults, therefore they are more prone to the deleterious effects of local anaesthetics.[5,7,9] Children have been reported to have convulsions even with serum lignocaine concentrations within the therapeutic range.[10]

Herein, I report a probable case of lignocaine-induced seizure in an infant presenting in a Government District Hospital in a remote district of Arunachal Pradesh, India.

Case Presentation

A 6-and-half-month-old female child of 7kg weight was presented in the ER with history of multiple abscesses since a week with history of mild fever the previous day.

The patient was previously healthy with no significant personal or familial medical history. She had no known drug allergies.

On examination, the infant was afebrile, active, vitals were stable. Local examination revealed multiple small abscesses on scalp and forehead and a large abscess of around 5cm in diameter on the left shoulder joint.

The infant was taken up for incision and drainage of the abscess on the shoulder under LA. 0.5ml of 4% lignocaine hydrochloride was administered. Immediately after the instillation of the local anaesthetic agent, the child became unresponsive and experienced several intermittent tonic–clonic convulsions. The seizure lasted for about 20 minutes with respiratory depression. The child was managed with intravenous diazepam, oxygen support and IV fluids.

After around an hour, the patient regained consciousness. Her saturation was 91% at room air, heart rate was 154bpm and Glasgow coma scale (GCS) was 12. No investigations were possible and the patient was referred to higher centre.

Using Naranjo probability scale the relationship between the drug and the event was categorized as probable.

Discussion

Local anaesthetic agents are widely used in most branches of medicine and are generally considered to be safe. Lidocaine is the most commonly used local anaesthetic because of its inherent potency, rapid onset, and moderate duration of action.[11] Applied either by injection, inhalation, or as a topical agent, lidocaine has a good safety margin before reaching toxic blood levels.[4]

What is less known is the occurrence of systemic toxicity which though negligible, may occur due to either over dosage, rapid absorption into blood from highly vascular spaces or accidental intravascular injection leading to increased plasma levels of these agents.[6]

In addition, toxic doses appear to be additive. Hence, any other local anaesthetics that may have been administered to the same patient should be taken into account.[4]

Systemic toxicity has been found to affect brain and/or myocardium in a similar mechanism, affecting their excitable membranes.[4.9.12] However, central nervous system toxicity usually precedes the cardiovascular effects since it occurs at lower plasma concentrations.[12]

Susceptibly to toxic effects of local anaesthetics depends on various factors like patient's age, maximum dose, site and speed of administration, presence or absence of concomitant disease, etc.[13]

All sexes are affected equally but patients at the extremes of age and pregnant women are likely to be more susceptible to local anaesthetic toxicity.[4]

We could not estimate the serum concentration of lignocaine in our patient. The recommended maximum dose of lignocaine in children is 3 mg/kg, which amounts to 21 mg (0.49ml) for our patient.[9] The total dose used in present case was 21.35 mg (0.5ml) which is 0.35mg higher than the safe dose range. This could have been due to difficulty of titration of small dosage. It has been reported that true overdosage of local anaesthetics is quite rare, except in small children, because of the low doses employed.[12]

However, toxicity can develop even during the use of the safest LAs' in safe dose ranges and use of suitable techniques for injection. Additionally, the threshold of toxicity may differ with factors such as medication, hypercarbia, electrolytes abnormalities, carnitine, alpha-1-acid glycoprotein or albumin deficiencies.[14]

Several case reports reported toxicity at safe dose.[9,15,16] Authors have suggested low tolerance to LA by some patients to be the likely explanation.[9,15] In another report, the event of seizure following LA during a dental procedure was concluded to be provoked by reversible condition such as the phobia of needles injection.[16]

A case series found the most frequent clinical presentations to be nausea and vomiting (50%), seizure (33.3%), and loss of consciousness (16.7%).[17] The manifestations of convulsion and respiratory depression after lidocaine administration in this study has been reported by other investigations as well.[18-20]

The diagnosis is usually made clinically. The timing, dose, and site of the lidocaine injection are the main factors in considering systemic manifestations. Blood levels of lidocaine and imaging studies aid in diagnosis.[17]

However, coincidental seizures due to a seizure disorder or panic attacks with hyperventilation may confound the diagnosis.[4] Anaphylaxis, anxiety disorders, cocaine toxicity, and conversion disorders should be considered in differential diagnosis of patients.[17]

At the outset of any probable signs and symptoms, injection should be stopped immediately, patient placed in supine position with legs elevated, protected from injuries and provided ABCs of basic life support.[9]

Treatment is symptomatic by raising the seizure threshold through pharmacologic interventions such as administering benzodiazepines and/or barbiturates or propofol. Hyperventilation with high doses of oxygen reduces cerebral blood flow and also has been used to raise the seizure threshold. [21,22]

Phenytoin has potential for synergistic effect with lidocaine toward cardiac toxicity. Thus, phenytoin may be optimally avoided in such circumstances. Diazepam, however, represents the first line treatment while Phenobarbital comes second.[12]

The other mainstay of treatment is to reduce the free available local anaesthetic concentration in the plasma by the administration of lipid emulsions.[4,23]

The adverse effects can be prevented by taking detailed medical history of the patient, by administering test dose, by following dosage protocol and using minimum necessary dose, by use of Adrenaline along with lignocaine to slow vascular uptake, by aspiration before and during injection by slow injection and by maintaining verbal contact with the patient.[9,23]

The prognosis of lidocaine toxicity depends on the dose and site of manifestation. CNS toxicity is either self-limited or quite amenable to treatment with benzodiazepines, has a good prognosis without sequelae, and does not need further neurologic testing. Cardiac toxicity may require prolonged resuscitation, but the prognosis after return to spontaneous circulation is often very good. Usually, outcomes are good with prompt treatment.[24,25]

Conclusion

All medical professionals should consider possibility of toxicity to local anaesthetics, recognize early signs and treat promptly to optimize patient outcome.

It should be kept in mind that toxicity can develop even during the use of the safest LAs' in safe dose ranges and use of suitable techniques for injection. Patients at the extremes of age and pregnant women are likely to be more susceptible to local anaesthetic toxicity.

Diagnosis is done based on clinical presentation, blood levels of lidocaine and imaging studies.

Benzodiazepines, hyperventilation with high dose oxygen and administration of lipid emulsions are mainstays of treatment.

It is vital to note anatomical structures before injection and understand the possible mechanisms of systemic toxicity.

Careful administration with minimum dosage and proper local anaesthetic techniques and appropriate monitoring of the patient throughout is mandated.

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