Case Report

Generalized seizure following lignocaine administration: Case report and literature review

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Abstract

Local anaesthetics (LAs) are considered the most effective drugs for prevention and management of pain associated with dental procedures. Lignocaine is the most preferred LA worldwide. Adverse drug reactions reported with lignocaine use are usually mild, however severe complications have been encountered. This article reports a case of lignocaine-induced seizure in a child. We also reviewed similar cases encountered over the last 10 years. The possible explanations could be placement of the needle in a small vein or hypersensitivity to lignocaine. We hereby reinforce the fact that seemingly safe LA's can cause life-threatening complications and rapid identification of clinical symptoms can drastically change the clinical course. Hence it is vital that primary care physicians and other healthcare professionals should to be aware, alert and be able to diagnose and manage these reactions immediately.

Keywords: Adverse drug reactions, dental procedures, lignocaine, local anaesthetics, seizure

Introduction

Local anesthetics (LA's) are used as pain relief measures in dental procedures and are the most widely used drugs in routine dentistry.[1] Lignocaine is commonly used in infiltration anesthesia, extremity blocks, topical anesthesia, intravenous regional anesthesia and in general anaesthesia.[2] Toxic reactions, though negligible, may occur due to over dosage, rapid absorption into the blood from highly vascular spaces or accidental intravascular injection leading to increased plasma levels of these agents.[2] Young children are certainly more vulnerable to experience a toxic reaction than adults. Herein, we report a probable case of lignocaine-induced seizure in a child.

Case Presentation

An 8-year-old healthy male patient (30 kg weight and 130 cm height) with no history of epilepsy visited the dentist for tooth extraction. The patient had no known drug allergies. During his visit for extraction of deciduous second molar, the dentist injected 1.8 ml of 2% lignocaine (36 mg) with adrenaline (1:200,000) after prior aspiration at upper left maxillary vestibule with respect to deciduous second molar followed by 0.5 ml at left greater palantine nerve block. The total 46 mg lignocaine lasting over 2 min was administered. Immediately following the nerve block, the child presented with tonic-clonic convulsions. Patient was placed in supine position with elevated legs, and care was taken so as to protect from injuries. Injection diazepam was given. The vital signs and all laboratory investigations were normal. Neurological examination was unremarkable. MRI scan of the head and electroencephalogram were normal. There was no recurrence of seizures and the child was discharged after 1 day. Using Naranjo probability scale the relationship between the drug and the event was categorized as probable.

Discussion

Central nervous system (CNS) and the cardiovascular system are more prone to the toxic effects. CNS toxicity is due to unopposed

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excitatory nerve activity resulting from blockade of inhibitory cortical synapses and increase of glutamate.[3] The symptoms include light-headedness, tinnitus, confusion, circumoral numbness, etc., which culminates in generalized seizures. Excitation is followed by CNS depression.[4] Onset of toxic symptoms usually begins within 10-20 minutes after injection. However, if the local anaesthetic is injected intravenously, the effect may be instant.[5]

At lower concentration, lignocaine has anticonvulsant properties, whereas definite CNS excitation and seizure is observed between at 4 to 10 ug/ml concentration. Further increase causes CNS depression.[6] Seizures induced by lignocaine in experimental models are focal in nature, invariably starting in the amygdala. However, the seizures occurring in patients after lignocaine administration are usually generalized.[7]

Neurologic toxicity has been frequently observed with IV use, occasionally with topical use and oral ingestion also.[3,8] Susceptibly to toxic effects of local anaesthetics depends on various factors like patient age, maximum dose, site and speed of administration, presence or absence of concomitant disease, etc.[1]

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 90 mg for our patient. The total dose used in present case was 46 mg which is within the safe dose range. The likely explanation for low dose adverse reactions is low tolerance to LA. Additionally the threshold of toxicity may differ with factors such as medication, hypercarbia, electrolytes abnormalities, carnitine, alpha-1-acid glycoprotein or albumin deficiencies.[4]

Similar to the present study, Ayas *et al.* (2014) reported a case of seizure with lignocaine within the safe dose range in a child.[2] A summary of case reports of lignocaine induced seizures in children's during the last 10 years have been depicted in Table 1.

Intravascular injection of lignocaine may result in toxic effects. However, adverse reactions can occur without intravascular placement of the needle and while using divided doses and repeated aspiration. A possible elucidation is that the needle tip might have been placed within a small vein, so that the negative pressure aspiration resulted in apposition of the vessel wall against the needle. This explains the present case. Increased sensitivity to lignocaine could have been another possible cause for the convulsive episode in the present case.

Management of the adverse reaction[6]

Stop injection immediately. In majority of the patients, the reaction is mild and ephemeral which usually does not require any specific treatment as the blood level of the anaesthetic decreases because of biotransformation and redistribution. The

Table 1: Summary of case reports of lignocaine toxicity reported in paediatric age group over last 10 years

Author, Year	Age (in months)/Sex	Route of administration (Total dose)	Presentation & time of onset
Alipour A (2019)[8]	16/Male	Oral 2%lignocaine	Lethargy followed by generalized tonic-clonic seizure and loss of consciousness within 15 minutes
Bathla *et al.* (2017)[9]	3/Male	6 ml i.v lignocaine (120 mg)	Focal seizures progessing to generalized tonic-clonic seizures
Alsukhni *et al.* (2016)[10]	180/Female	1.5 ml of 2%lignocaine with epinephrine (30 mg)	Status epilepticus followed by coma in 2 minutes
Syed hoda *et al.* (2015)[11]	17/Male	Polysporin kids cream containing 5%lignocaine (750 mg)	Generalized tonic-clonic seizures after 20 minutes
Doye *et al.* (2015)[12]	4/Male	Bilateral dorsal penile nerve block with lignocaine (16 mg/kg)	Generalized tonic-clonic seizure and cardiac arrest within 15 minutes
Aminiahidashti *et al.* (2013)[11]	48/Male	20-25 ml lignocaine 2%solution (50 mg)	Generalized tonic-clonic seizures after 3 hours
Ayas and Isik (2014)[2]	12/Male	1% lignocaine IV (40 mg)	Generalized seizure within 30 seconds
Ozer and Erhan (2014)[13]	4/Male	2.5 ml ampoules of 2%lignocaine (100 mg)	Loss of consciousness, generalized tonic-clonic seizures and erythematous rash
Larson *et al.* (2013)[14]	4/Female	Cream containing 2.5%lignocaine and 2.5%prilocaine (1500 mg)	Seizures and methemoglobinemia 75 minutes after treatment
Jagadish and Manjunath (2013)[15]	2/Male	1 ml of 2%lignocaine (20 mg)	Generalized tonic-clonic convulsions within few minutes
Menif *et al.* (2011)[5]	<1/Male	Subcutaneous 3.5 ml of 1%lignocaine for regional anesthesia (35 mg)	Generalized seizure after 30 minutes
	1.5/Male	Subcutaneous 1.5 ml of 1%lignocaine (15 mg)	Generalized seizure after 2 hours
	3/Male	Subcutaneous lignocaine (6 mg/kg)	Generalized seizure in 90 minutes
Sanaeizadeh *et al.* (2011)[16]	3/Male	2 ml lignocaine 2% (40 mg)	Generalized tonic-clonic seizures within 20 minutes

patient should be placed in supine position with elevated legs and protected from injuries ABCs of basic life support to be provided. Benzodiazepines for seizure control.

Prevention[4,6]

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.

Conclusion

The present case signifies that toxicity can develop even during the use of the safest LAs' by experienced professionals with all treatment standards met and use of suitable techniques for injection. Dental professionals should recognize early signs of toxicity of LAs' and should be well-trained to perform CPR in order to improve the patient outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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