EPINEPHRINE SYNDROME, AWARENESS BELL FOR PROSTHODONTISTS : A CASE REPORT

Dr. Niranjan Kumar Chugh *, Dr. Shivam Singh Tomar, Dr. Sushen Sharma*****

Abstract

Epinephrine is one of the most widely-used vasoconstrictors in dental treatment. However, the systemic safety of epinephrine has been in debate for many years because of its potential risk to cause cardiovascular complications. Prosthodontic work on vital tooth requires direct application of epinephrine into the oral cavity, and the amount is reported to be much larger than other dental surgeries. Besides, pre-existing cardiovascular complications or drug interactions can enhance its systemic influence, resulting in increased susceptibility to cardiovascular complications. Epinephrine is a dose-sensitive drug, and its hypersensitivity reaction can be fatal to patients when it is related to cardiovascular complications.The purpose of this case report was to make dentists more aware of epinephrine overdose, its cause and side effects with management.

Key Words Cardiovascular diseases, Drug interactions, Epinephrine, Vasoconstrictor agent

Key Messages Epinephrine syndrome is a less known fact amongst the dentists, which actually can cause life threatening conditions in our day-to-day practice, if not known.

INTRODUCTION

Local anesthetics are the most common drugs used by dentists in clinical practice. A reversible anesthetic effect and a wide margin of safety when administered in proper doses to obtain anesthesia are hallmarks of this drug class. However, high blood concentrations obtained via intravascular injections or frank overdoses may produce significant toxic effects to the central nervous system and the cardiovascular system.[1,2] Other physiologic changes caused by the administration of local anesthetics may be due to the drug's direct and indirect effects on the heart and blood vessels. It must be emphasized that the role of vasoconstrictors, which are included in small concentrations in anesthetic solutions for the purpose of improving both the quality and the duration of anesthesia, as well as decreasing the systemic toxicity of the local anesthetic agent. In general, toxic effects caused by vasoconstrictors develop before the toxic effects produced by local anesthetics and, consequently, may constitute a limiting factor when considering the anesthetic solution dosage and volume in adults.[3,4] Individual susceptibility plays a significant role in determining the toxicity potential of a local anesthetic.

Epinephrine is commonly used in health care and has multiple applications. Two frequent and often life-saving uses are the management of anaphylaxis and cardiac arrest. The variation in response of the drug depends in part on the number and predominant type of adrenergic receptors present in the target organ and on the physiologic reflex

ABOUT THE AUTHORS

***Assistant Professor**, Department of Dentistry, Sri Ramakrishna Institute of Medical Sciences, Durgapur
****Senior Lecturer**, Manav Rachna Dental College, Faridabad
*****Private Practitioner**

response that attempts to minimize the effects of sympathetic stimulation. As an adjunct to anaesthetic, the effect this drughas on heart rate, stroke volume, cardiac output, heart rhythm, myocardial oxygen demand and peripheral vascular resistance must be appreciated.

Therefore, it is important to use appropriate anaesthetic techniques to avoid intravascular injections.[5,6] Administration speed is particularly important since a higher rate of injection of the local anaesthetics solution leads to both a greater systemic blood level of the injected drug and greater potential toxic effects.[5] Patients who are on antidepressants or beta-adrenergic blocking agents need a more careful preoperative evaluation of the maximum permissible dosages of local anaesthetics and vasoconstrictors.[7-9] In these patients, the use of phenylephrine is not recommended due to the higher risk of hypertension as a result of increased peripheral resistance. Felypressin is a noradrenergic vasoconstrictor that would be recommended in these patients. [10] These concerns become more relevant when we consider the possibility of accidental intravascular injection, especially when the local anaesthetic is combined with adrenergic vasoconstrictors. [5]

CASE HISTORY:

A 38 year-old lady visited to the Department of Prosthodontics, S.R.I.M.S & Hospital, Durgapur, West Bengal, to have a crown preparation performed on her lower right first molar. The patient was apparently fit and well with no previous medical history. The patient had a dental history of an amalgam restorations and root canal treatment performed under local anaesthesia (lignocaine) 2% with adrenaline without any complications (fig 1-5, treatment photographs of patient). Before starting the dental procedure infiltration and inferior dental and lingual block with 2% lignocaine and adrenaline (1:100000) was administered.

Almost immediately after administration of local

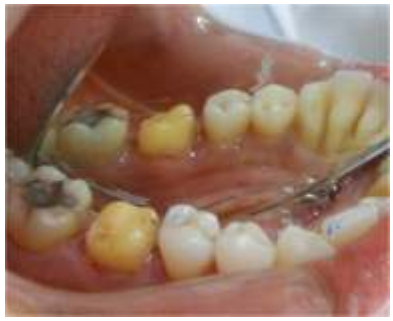


Figure 1

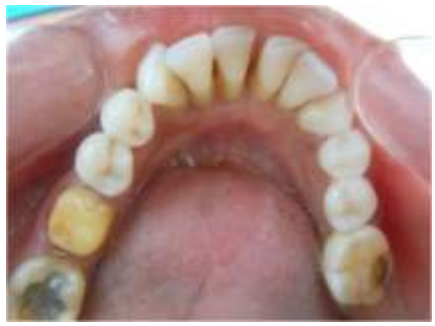


Figure 2

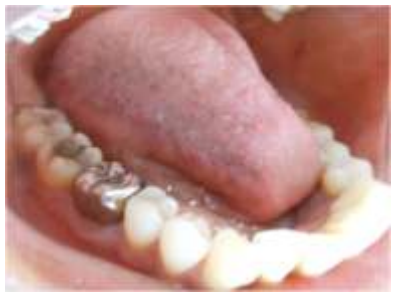


Figure 3

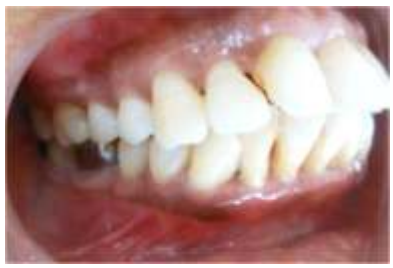


Figure 4

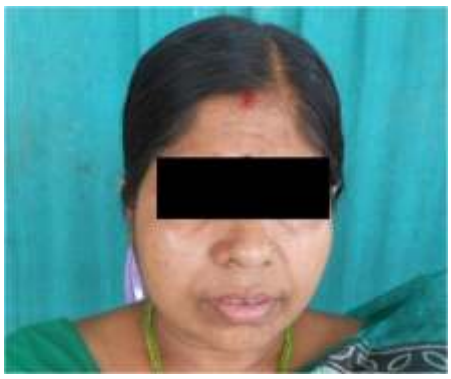
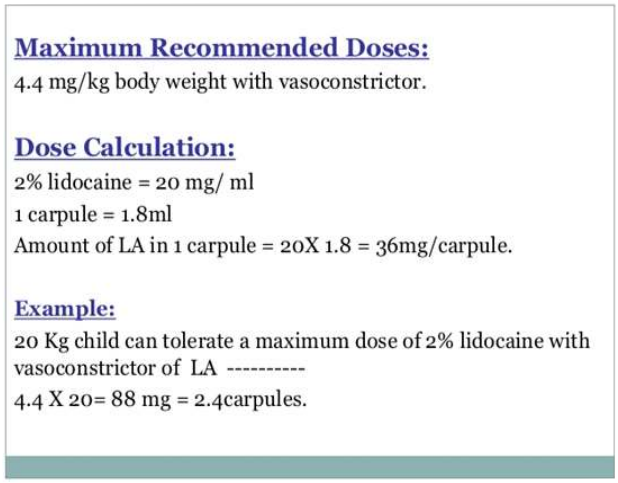


Figure 5

COMPOSITION

1	Local anesthetic agent -	21.3 mg\ml
2	Vasoconstrictors	0.005 mg\ml
3	Reducing agents like Sodium metabisulphite	0.5 mg\ml
4	Preservatives like Methyl paraben	1 gm\ml
5	Fungicide Thymol	
6	Vehicle - Ringers lactate	6mg\ml

Figure 6



Maximum Recommended Doses:

4.4 mg/kg body weight with vasoconstrictor.

Dose Calculation:

2% lidocaine = 20 mg/ ml

1 carpule = 1.8ml

Amount of LA in 1 carpule = 20X 1.8 = 36mg/carpule.

Example:

20 Kg child can tolerate a maximum dose of 2% lidocaine with vasoconstrictor of LA ----------

4.4 X 20= 88 mg = 2.4carpules.

Figure 7

anaesthesia the patient was feeling ill. She was complaining of being very apprehensive and was holding her chest complaining of palpitations.

The patient was managed immediately, was encouraged to breath regularly. She was made to lie down in supine position with head slightly down. Peripheral pulse was measured, pulse was 105 per minute, tachycardia was present. Oxygen was given using face mask (5 litters/ min). Blood pressure was monitored to be 144/90 mm of Hg. Patient was checked for rash/urticaria/pallor/change or difficulty in respiration—no findings were found.

The patient was diagnosed provisionally with vasovagal attack, or adverse reaction to local anaesthesia, or allergic response to local anaesthesia and/or myocardial infarction or angina attack.

Since the patient was slightly hypertensive with tachycardia present, the possibility of syncope decreased. The patient had a dental history with previous multiple exposure to local anaesthesia and has no symptoms of hypersensitivity such as urticaria, dyspnoea, low blood pressure etc. thus the probability of type 1 hypersensitivity decreased too. The symptoms of myocardial infarction is crushing central chest pain, sometimes radiating to the arm or neck, dyspnoea and possibility of vomiting, which was this patient was absent. The patient remains conscious but is nervous and agitated. She gradually calms and says that the palpitations have reduced. She takes few more breaths of oxygen but refuses more after a few minutes and says she is feeling better. Pulse and blood pressure were measured again which were reduced as compared to the previous readings.

Definite Diagnosis:

Intravascular injection was the most likely diagnosis because the patient's symptoms were being caused by the vasoconstrictor component of local anaesthesia (fig 6). The solution contains 1:100000 epinephrine which caused tachycardia, palpitations and apprehension after the administration of the block because of high vascularity of the injection site. Anxiety can itself cause a significant level of adrenaline but very slowly and the patient would have to be very nervous, positively phobic to generate endogenous adrenaline to the levels found in intravascular injection of local anaesthetic.

DISCUSSION:

Although theoretically possible, overdose of local anaesthesia is actually quite difficult of administer in dentistry. It is important to realize that advice based on a recommended number of cartilages or fixed dose does not take into account different formulations. Current recommendations are expressed in the form of the maximum safe dose per body weight given over a period of treatment of one hour (fig 7). This case was classical presentation of epinephrine syndrome where the symptoms were caused by intravascular injection of epinephrine. Good injection technique is the key to reducing the risk of intravascular administration. The solution should be injected slowly, reducing the risk of a bolus injection into the vessel. An aspiration technique is always good though it does not always guarantee success; the narrow needle diameters used in dentistry aspirate relatively poorly.

It is important to completely avoid the tip of the needle entering a vessel. Indeed, in some vascular areas penetration of the needle into a vessel is not the cause because the solution can be absorbed into the blood almost as rapidly as it can be injected. Nothing can guarantee the prevention of intravascular injection.

Conclusion: Clinicians should be aware of the nature of epinephrine, and should pay attention to its potential risk when treating patient with endodontic microsurgery, especially for medically compromised patients.

REFERENCES:

1. Abraham-Inpijn LAL, Borgmeijer-Hoelen AB, Gortzac RAT. Changes in blood pressure, heart rate and electrocardiogram during dental treatment with use of local anesthesia. Am Dent Assoc. 1981;116(4):531–531. [PubMed]

2. Yagiela JA. Local anesthetics. In: Yagiela JA, Neidle EA, Dowd FJ, editors. Pharmacology and Therapeutics for Dentistry. 4th ed. St Louis, Mo: Mosby; 1998. pp. 217–234.

3. American Dental Association Council on Scientific Affairs. ADA Guide to Dental Therapeutics. 2nd ed. Chicago, Ill: American Dental Association; 2000.

4. USP DI Updates On-line, United States Pharmacopeial Convention Inc. http://library.dialog.com/bluesheets/html/bl0461.html#top (accessed March 16, 2004)

5. Malamed SF. Handbook of Local Anesthesia. 5th ed. St Louis, Mo: Mosby; 2004. pp. 159–167.

6. Kiyomitsu Y, Sugiyama K, Joh S. The effects of catecholamines added to lidocaine on cardiac function. Anesth Prog. 1989;36:198–200. [PMC free article] [PubMed]

7. Yagiela JA. Local anesthetics. Anesth Prog. 1991;38(4):128–128. [PMC free article] [PubMed]

8. Mito RS, Yagiela JA. Hypertensive response to levonordefrin in patient receiving propranolol. report of case. Am Dent Assoc. 1988;166(1):55–55. [PubMed]

9. Foster CA, Aston SJ. Propranolol and epinephrine interaction: a potential disaster. Plast Reconstr Surg. 1988;72(1):55–55. [PubMed]

10. Hargreaves KM, Neidle EA. Adrenergic drugs. In: Yagiela JA, Neidle EA, Dowd FJ, editors. Pharmacology and Therapeutics for Dentistry. 4th ed. St Louis, Mo: Mosby; 1988. pp. 85–99.

11. Malamed SF. Local anesthetics: dentistry's most important drugs, clinical update 2006. Calif Dent Assoc. 2006;34:971–976. [PubMed]

12. Van der Bijl P, Victor AM. Adverse reactions associated with norepinephrine in dental local anesthesia. Anesth Prog. 1992;39(3):87–87. [PMC free article] [PubMed]