Case Report

Hypotension in Cath Lab, a Rare Cause…

Vivek Singh Guleria[1], DM; Prafull Sharma[1], DM

[1]Department of Cardiology, Army Hospital Research and Referral, New Delhi, India

Received: 15-04-2020
Revised: 07-05-2020
Accepted: 30-05-2020
Published: 26-09-2020

Abstract

Sudden onset of hypotension during a procedure makes interventionalist perturbed. In cath lab, hypotension is a red flag sign, and the causes are diverse, ranging from vasovagal, cardiac tamponade, or a significant bleed somewhere, keeping the operators on their toes. We had a similar ordeal recently when a patient developed prolonged hypotension requiring inotrope and vasopressor support after local anesthesia. This case will act as a sensitizer to practitioners and make them more vigilant for the symptoms of toxicity after local anesthetic administration.

Keywords: *Adverse effect, hypotension, lignocaine*

Introduction

Lignocaine, is an amide local anesthetic agent and a Class 1b antiarrhythmic. For the past five decades, it has been commonly used in various clinical settings. In interventional cardiology, it is widely used before every procedure. Despite its widespread use, most physicians are not familiar with the life-threatening manifestations of lignocaine toxicity and its treatment. Allergic reactions to drugs are common and are divided into four types: Type I (immediate hypersensitivity), II (cytotoxic), III (immune complex), and IV (contact dermatitis). The conjugation of the offending antigen (drug) with IgE in the surface of mastocytes (in tissues) or of basophils (circulating), results in anaphylaxis mediators release, especially histamine which, in turn, produces vasodilation, and increased capillary patency.

Anesthetics are classified as either ester- or amide-type local anesthetics. Ester-type local anesthetics generate Para-aminobenzoic acid as a metabolite that can induce hypersensitivity, while amide-type local anesthetics seldom cause hypersensitivity.[1]

Lignocaine is an essential drug on the World Health Organization's essential drug list, considered efficacious, safe, and cost-effective for any health-care system. Type I hypersensitivity to lignocaine by specific IgE antibodies against the drug has been reported by previous studies.[2-4] This study is aimed at presenting a case of allergy to local lignocaine.

Case Report

60-year-old male with no comorbidities, had been operated for hernia 3 years back under general anesthesia, was taken up for electrophysiological studies in view of recurrent palpitations and presyncope. He had a blood pressure of 140/80 mmHg and pulse of 84/min at the start of the procedure. Groin area was cleaned with betadine, 10 ml of lignocaine was infilterated, and three femoral venous lines were secured. Coronary sinus catheter was placed in the coronary sinus. Right at this time patient started complaining of paresthesia in the perianal region spreading to the groin and whole body. He developed erythematous rash over the chest, face and started sweating profusely. His blood pressure dropped down to 60/30 mmHg and his pulse rate increased to 120/min. Two-dimensional echography was done, and cardiac tamponade was ruled out. Intravenous crystalloids were rushed through the femoral venous line, but hypotension persisted. There was no evidence of retroperitoneal hemorrhage on ultrasonography either. With this, a provisional diagnosis of anaphylactic reaction to lignocaine was made. Anti-histaminics and a steroids were administered through parenteral route, but nothing helped. The patient was started on adrenaline and noradrenaline infusion, and later vasopressin was added. He was shifted from cath lab to the intensive care unit once his systolic blood pressure was 90 mmHg. He continued to be hypotensive for the next 6 h and thereafter, his vasopressors were gradually tapered off. After 2 days, the skin prick test (SPT) was administered using lidocaine. SPT results were read after 15 min and were positive [Figure 1].

Discussion

One of the most widely used local anesthetic agent, lignocaine is safe and effective for almost every possible type of local anesthetic procedure. It has a rapid onset, and it is effective for about 30–60 min in its plain form (or up to 90 min when used with a vasoconstrictor).

Address for correspondence: Dr. Vivek Singh Guleria, DM, Department of Cardiology, Army Hospital Research and Referral, New Delhi - 110 010, India.
E-mail: viveksguleria@gmail.com

Access this article online

Quick Response Code:

Website: www.jcpconline.org

DOI: 10.4103/JCPC.JCPC_19_20

How to cite this article: Guleria VS, Sharma P. Hypotension in cath lab, a rare cause…. J Clin Prev Cardiol 2020;9:121-2.

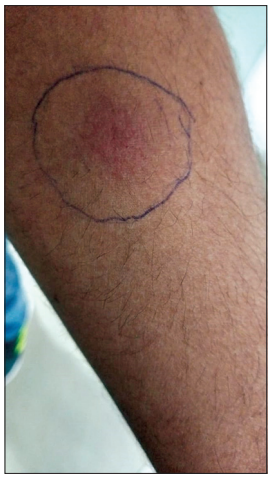

Figure 1: Positive skin prick test done after 2 days

It takes 10–25 min for lignocaine to reach peak blood levels after injection. At this time, toxic effects are most likely to be observed. The onset of symptoms is faster if accidental intravascular injection has occurred. The first symptoms and signs of local anesthetic toxicity are usually neurological with numbness of the mouth and tongue. Shortly afterward, there is tinnitus, confusion, seizures, and potentially coma. Cardiovascular toxicity usually manifests itself as tachycardia and hypertension but with increasing toxicity, bradycardia and hypotension occur. Ventricular arrhythmias and cardiac arrest are also known side effects.[5]

Characteristics of arrhythmias induced by Local anesthetics (LAs) are prolonged PR, QRS, and QT intervals potentiating reentry. Aberrant conduction may result in cardiac arrest. The treatment of local anesthetic toxicity is essentially supportive. The airway should be maintained, and oxygen should be administered. If convulsions occur, they should be controlled with benzodiazepines along established guidelines. The symptoms of toxicity persist as long as the plasma concentrations remain above the therapeutic index. Seventy per cent of the dose is metabolized in the liver and less than 10% is excreted unchanged in the urine. Diagnosis of lignocaine toxicity is mainly clinical. Therapeutic concentrations of lidocaine can be up to 5.5 mg/L, whereas a plasma level of 8–12 mg/L and above is associated with central nervous system and cardiotoxicity.[6] SPTs have low negative predictive value.

The American Society of Regional Anesthesia guidelines recommend starting lipid emulsion therapy at the first signs of systemic toxicity from LAs, after airway management . It is proposed that lipid infusion creates a lipid phase that extracts the hydrophobic molecules of LA from the aqueous plasma phase.[7] Other considerations in cardiac life support for patients with local anesthetic-induced cardiac arrest are as follows; epinephrine is recommended, small initial doses (10–100 μg boluses in adults); vasopressin is not recommended; avoid calcium channel blockers and beta-blockers; if ventricular arrhythmias develop, amiodarone is preferable and in patients who do not respond to standard resuscitative measures, cardiac pacing, and cardiopulmonary bypass are recommended.[7]

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.

Financial support and sponsorship

Nil

Conflicts of interest

There are no conflicts of interest.

References

1. Baluga JC, Casamayou R, Carozzi E, López N, Anale R, Borges R, *et al*. Allergy to local anaesthetics in dentistry. Myth or reality? Allergol Immunopathol (Madr) 2002;30:14-9.
2. Helfman M. An unusual symptom of lidocaine allergy. Report of a case. N Y State Dent J 2002;68:24-5.
3. Ogunsalu CO. Anaphylactic reaction following administration of lignocaine hydrochloride infiltration. Case report. Aust Dent J 1999;44:64-8.
4. Kokubu M, Oda K, Shinya N. Detection of serum IgE antibody specific for local anesthetics and methylparaben. Anesth Prog 1989;36:186-7.
5. Brown DL, Skiendzielewski JJ. Lidocaine toxicity. Ann Emerg Med 1980;9:627-9.
6. Brosh-Nissimov T, Ingbir M, Weintal I, Fried M, Porat R. Central nervous system toxicity following topical skin application of lidocaine. Eur J Clin Pharmacol 2004;60:683-4.
7. Neal JM, Mulroy MF, Weinberg GL; American Society of Regional Anesthesia and Pain Medicine. American Society of Regional Anesthesia and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2012 version. Reg Anesth Pain Med 2012;37:16-8.