Dawood Nasir*, Babatunde Ogunnaike and Charles Whitten

Department of Anesthesiology and pain management, University of Texas Southwestern Medical Center, Dallas, Texas, USA

*Corresponding author: Dawood Nasir, Department of Anesthesiology and pain management, University of Texas Southwestern Medical Center, Dallas, Texas, USA, Tel: 972-804-8049; E-mail: dawood.nasir@UTSouthwestern.edu

https://www.peertechz.com

Case Report

Case Study: Local Anesthetic Toxicity After Bier Block. Was intralipid Therapy the Correct Remedy

Abstract

Introduction: Intralipid therapy is recommended for patients with local anesthetics like bupivacaine, but it is not advisable for patients with short acting local anesthetic toxicity like lidocaine.

Case Presentation: 42 years old healthy female was brought to operating room for release of carpal tunnel syndrome of left hand. It was decided to perform Bier block. After placement of successful Bier block, surgery was completed in twenty minutes and after tourniquet release, patient was noted to have some shaking movement. Local anesthetic toxicity was suspected due to the tourniquet being released too soon, before the recommended time of forty-five minutes. ACLS protocol was initiated by providing oxygen and breathing assistance to the patient and taking care of airway. Patient was initially confused and agitated, but she recovered after a few minutes becoming awake and alert very soon afterwords. She was observed for few hours and was discharged.

Could intralipid therapy have been beneficial to this in patient with lidocaine toxicity?

Conclusion: Intralipid therapy has been shown to be beneficial for amide local anesthetic-toxicity such as like bupivacaine. Bupivacaine is cardio-toxic and has a long duration of action. For less cardiotoxic local anesthetics or local anesthetic's which are less cardio toxic, following ACLS protocol with symptomatic support may be sufficient for successful resuscitation.

Historical facts: The Bier Block was first introduced by August Bier in 1908 but C. McK. Holmes reintroduced it in 1963. Today, the technique is common as it is economical, it has a rapid recovery, it is reliable and is simple to use.

Intravenous regional anesthesia or Bier block anesthesia is an anesthetic technique for surgical procedures on the body's extremities especially the upper extremity where a local anesthetic is injected intravenously. The technique usually involves exsanguination which forces blood out of the extremity, followed be the application of pneumatic tourniquets to safely stop blood flow. The anesthetic agent is introduced into the limb and allowed to set in while tourniquets retain the agent within the desired area.

After some time, the tourniquet is depressurized to start back the circulation.

Introduction

Bier block is a valuable technique for short procedures, but as the local anesthetic is being injected intravenously, it is important to make sure that the tourniquet is not released for at least forty five minutes which will give local anesthetics enough time to be absorbed slowly and to prevent any systemic toxicity.

Case

Description: In this case we detailed a case for a procedure of carpel tunnel release in an obese patient utilizing Bier Block.

Some indications for Bier block include release of carpel tunnel syndrome, incision and drainage of an extremity, ganglion excision, open reduction and internal fixation of hand, wrist and forearm [1-4].

Equipment needed include esmarch bandage, double blood pressure cuff, intravenous catheter, syringe, & 0.5% Lidocaine 50 milliliters (Figure 1).

Technique of Bier block

Insert intravenous catheter as distal as possible on the operative extremity, place a double blood pressure cuff around

the upper arm. Raise the arm and wrap the esmarch bandage from distal to the proximal portion of the upper extremity.

After exsanguination of the blood from the upper extremity, inflate the upper cuff 50 mm Hg above the systolic blood pressure, remove the esmarch bandage, inject 50 ml of 0.5% lidocaine in distal intravenous catheter. Onset of block is within 5 minutes. Remove the distally placed IV catheter. Duration of block should be 45 minutes. If tourniquet pain arises, the lower cuff should be inflated before deflating the upper cuff [4-8] (Figure 2).

After placement of successful block, surgery was performed. Tourniquet was released at approximately thirty minutes of injection of local anesthetic. Soon after release of tourniquet patient started shaking. Patient was obese but did not have any other medical problem

It was suspected that patient may have had local anesthetic toxicity.

Complications from local anesthetic which could be systemic initially and affecting the central nervous system include, metallic taste in the mouth, ringing in the ears, light headedness, dizziness, convulsions or seizures and even coma.

Late symptoms include prolonged P-R interval, A-V block, Cardiac arrest.

In this patient oxygen was administered by face mask. Endotracheal intubation equipment was prepared. In the meantime, the patient stopped shaking. Vital signs remained

Figure 1:

Figure 2:

stable with a pulse rate of 70 beats per/ minute. Blood pressure was 107/66. Oxygen saturation stayed above 95%, and the patient never lost consciousness. After few minutes, patient was responsive and answering questions appropriately.

Intralipid therapy was considered but not administered. Intralipid therapy was readily available but will have had have been collected from the Pyxis machine where is it is normally stored.

After few minutes of keeping the patient in the operating room, she was taken to the recovery room where she was observed for a couple of hours, during which she did not showed any signs of local anesthetic toxicity and was discharged after meeting the Aldrete criteria for discharge.

Mechanism of action of local anesthetic

Local anesthetics produce anesthesia by inhibiting excitation of nerve endings or by blocking conduction in peripheral nerves. This is achieved by reversibly binding to and inactivating sodium channels. Sodium influx through these channels is necessary for the depolarization of nerve cell membranes and subsequent propagation of impulses along the course of the nerve. When a nerve loses depolarization and capacity to propagate an impulse, the individual loses sensation in the area supplied by the nerve [9,10].

Mechanism of action of intralipid

Three postulated mechanisms have been brought forward to explain the beneficial effect of lipid. The first suggests establishment of a new pharmacokinetic equilibrium within an expanded plasma lipid phase, reducing free drug levels and thereby toxicity. Secondly, bupivacaine is known to inhibit carnitine acyltransferase, essential in transport of fatty acids across the inner mitochondrial membrane. ILE may overcome this inhibition and the metabolic effects of other similar metabolic poisons through "mass effect" alone or via some as yet unknown mechanism on preferred myocardial substrate. Third, fatty acids are known to increase cardiac myocyte calcium levels and may act via a direct inotropic action through increasing intramyocyte calcium concentration. This hypothesis may be of particular relevance to overdose with calcium channel blocking agents. All postulates permit the cogent hypothesis that the beneficial effect demonstrated in local anesthetic toxicity may generalize to agents of similar lipophilicity and/or inhibition to mitochondrial lipid metabolism.

Dosing for 20% intralipid therapy

Administer 1.5 mL/kg as an initial bolus; the bolus can be repeated 1- 2 times for persistent asystole.

Start an infusion at 0.25 mL/kg/min for 30-60 minutes; increase infusion rate up to 0.50 mL/kg/min for refractory hypotension.

Conclusion

In most of the cases, if local anesthetic toxicity is suspected, ACLS protocol should be initiated with support of the circulation and airway management.

Intralipid therapy should be considered if long acting more cardio toxic local anesthetic is the culprit.

References

1. Weinberg G, Ripper R, Feinstein DL, Hoffman W (2003) Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. Reg Anesth Pain Med 28: 198-202. **Link:** https://goo.gl/59wsDQ

2. Rosenblatt MA, Abel M, Fischer GW, Itzkovich CJ, Eisenkraft JB (2006) Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. Anesthesiology 105: 217-218. **Link:** https://goo.gl/XAneds

3. Weinberg G (2009) Lipid rescue in the literature. **Link:** https://goo.gl/4mXTP0

4. Weinberg GL, Ripper R, Murphy P, Edelman LB, Hoffman W, et al. (2006) Lipid infusion accelerates removal of bupivacaine and recovery from bupivacaine toxicity in the isolated rat heart. Reg Anesth Pain Med 31: 296-303. **Link:** https://goo.gl/vs28Ii

5. de Jong RH (2007) Lipid infusion for cardiotoxicity: promise? Yes-panacea? Not. Anesthesiology 106: 635-636. **Link:** https://goo.gl/HZV5EG

6. Corman SL, Skledar SJ (2007) Use of lipid emulsion to reverse local anesthetic-induced toxicity. Ann Pharmacother 41: 1873-1877. **Link:** https://goo.gl/yJRTdd

7. Corcoran W, Butterworth J, Weller RS, Beck JC, Gerancher JC, et al. (2006) Local anesthetic-induced cardiac toxicity: a survey of contemporary practice strategies among academic anesthesiology departments. Anesth Analg 103: 1322-1326. **Link:** https://goo.gl/k3StMD

8. Weinberg GL (2008) Lipid infusion therapy: translation to clinical practice. Anesth Analg 106: 1340-1342. **Link:** https://goo.gl/WZgFc3

9. Brull SJ (2008) Lipid emulsion for the treatment of local anesthetic toxicity: patient safety implications. Anesth Analg 106: 1337-1339. **Link:** https://goo.gl/Iustqy

10. Weinberg G (2009) Treatment regimens. **Link:** https://goo.gl/YVriew