Cardiac arrest after submucosal infiltration with lignocaine 2% – epinephrine in nasal surgery: A case report

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AABSTRACT
A case of a 26-year-old ASA I physical status male undergoing septoplasty had an abrupt pulseless ventricular tachycardia following submucosal infiltration of lignocaine 2% with epinephrine 1:200,000 combination. Ventricular tachycardia associated with unconsciousness and absent peripheral pulse was transient and easily reverted by precordial thump, but was recurrent. Ventricular tachycardia was replaced by ventricular bigeminy and subsequently by sinus tachycardia.

Introduction

Vasoconstrictors have been used in combination with local anaesthetic (LA) agents to reduce intraoperative bleeding,[1] reduce the risk of systemic toxicity of local anaesthetics,[2] and prolong their duration of action.[3] Nasal surgeries like septoplasty have been performed under local anaesthesia alone or in combination with general anaesthesia. It is very interesting to know the various ways nasal cavities are decongested and anesthetised for ear, nose and throat (ENT) procedures. ENT surgeons have been following the traditional nasal packing with ribbon gauze soaked in Local anaesthetic- epinephrine solution and after pack removal applying wool pledgets soaked in LA and finally using anterior ethmoidal or maxillary nerve blocks or submucosal infiltration of the nasal cavity. Lignocaine with epinephrine combination is most widely used by surgeons and anesthesiologists. Unfortunately, the effects of epinephrine are not always beneficial. Epinephrine use may be associated with complications like pulmonary oedema, reversible cardiomyopathy, myocardial infarction, severe hypertensive crisis, cerebral haemorrhage and cardiac arrest.[4]

We present a young ASA I physical status patient who had transient recurrent pulseless ventricular tachycardia after submucosal intranasal lignocaine epinephrine infiltration in a scheduled septoplasty.

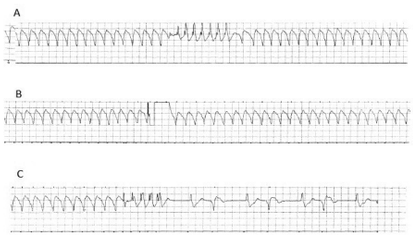
Case report

A 26-year-old, 60 kg male, ASA I physical status, was scheduled for septoplasty for deviated nasal septum under monitored anaesthesia care (local anaesthesia with intravenous sedation). The patient had an unremarkable medical history and unremarkable laboratory investigations. He denied any previous cocaine addiction and allergies.

The patient was premedicated with intramuscular atropine 0.6 mg one hour prior to surgery. After confirming starvation and consent, the patient was placed on the operating table. Monitors were connected to the patient for continuous monitoring of cardiac, pulse oximeter oxygen saturation (SPO2) and non invasive blood pressure (NIBP). Prior to the procedure his heart rate (HR) was106/min, NIBP – 130/84 mmHG, SPO2 – 99%, respiratory rate – 16/min with normal cardiovascular and respiratory system examination. Intravenous access was secured with 20 gauge angiocath and intravenous fluid Ringer lactate was started at 100 ml/hr. Intravenous pentazocine 30 mg, promethazine 25 mg diluted in 5 ml normal saline were administered for analgesia and sedation. Midazolam 1 mg was also given intravenously for sedation. Intravenous ranitidine 50 mg and ondansetron 4 mg were given. Post sedation the patient had normal vitals, was sedated but arousable and obeyed verbal commands. The surgeon packed the right nasal cavity with ribbon soaked in a solution of 30 ml 4% topical lidocaine and four ampoules of epinephrine 1:1000 (4 mg). After 15 minutes 6 ml of 2% lignocaine with epinephrine 1: 200,000 (5 μg/ml) was infiltrated submucosally after careful aspiration of the needle. At around seven minutes post infiltration the patient had a run of monomorphic ventricular tachycardia with a rate of > 200/min as seen on the cardioscope. SPO2 and NIBP were not recordable, peripherals and carotids were not felt. The patient developed pallor of face and cold pulseless extremities. The patient was also unresponsive. An emergency call for a defibrillator and help was made. The patient received a precordial thump and cardio pulmonary cerebral resuscitation was started with cardiac massage and bag mask ventilation of 100% oxygen. The patient reverted to sinus tachycardia immediately on cardiac thump, but was still not responsive to verbal commands and did not breathe. Endotracheal intubation was done with 8.5 cuffed portex tube under direct laryngoscopic vision and ventilated with 100% oxygen, as cardiac massage was continued by the surgeon. Soon the pulse oximeter recorded 89% SPO2 and NIBP of 180/110 mmHG. The patient had another run of ventricular tachycardia with a rate of 300/min. Another cardiac thump (as defibrillator arrived) reverted it back to sinus tachycardia transiently soon to be followed by ventricular bigeminy. One hundred mg lignocaine hydrochloride (20 mg/ml) was given intravenously. The patient once again reverted to sinus tachycardia.

Arterial blood gas (ABG) with electrolytes sample was collected for urgent lab investigation. The patient was now responsive to verbal commands and had SPO2 – 100%, NIBP – 140/86 mmHG, HR – 140/min, clear chest with normal first and second heart sounds (S1 S2). The patient remained stable and free of any arrhythmias as monitored in intensive care for that day. His ABG and electrolytes were normal. A 12 lead electrocardiogram taken was normal. Echocardiography revealed a normal ejection fraction of 60%, and a normal study.

Figure 1: ECG of ventricular tachycardia



A: Ventricular tachycardia interrupted by precordial thump transiently.
B: Ventricular tachycardia monomorphic type rate>200/min.
C: Ventricular tachycardia interrupted second time with thump followed by ventricular bigeminy.

Discussion

Infiltrative local analgesia combined with moderate sedation is a well accepted and safe anaesthetic method for nasal surgeries like septoplasty.[5] Various local anaesthetics used are cocaine, benzocaine, tetracaine and lignocaine, but lignocaine has the widest margin of safety and is most common in use. Epinephrine remains the most commonly used vasoconstrictor, despite efforts to popularise phenylephrine, nor-epinephrine, vasopressin analogues like octapressin and felypressin. Although the main purpose of vasoconstrictors is to reduce systemic absorption and potential systemic toxicity of a local anaesthetic agent, there is systemic absorption of the vasoconstrictor itself.[6,7] In addition, there is an added chance of rapid systemic absorption of both the local anaesthetics and the vasoconstrictor, following inadvertent intravascular injections like in our patient [8]

Systemic actions of epinephrine are produced by effects on alpha and beta adrenergic receptors. The systemic actions can be classified into five broad types.[9] First of all it has a cardiac excitatory action resulting in an increase in heart rate, force of contraction and stroke volume. The second type is central nervous system excitation. Metabolic actions include increased glycogenolysis in liver and hypokalemia. Peripheral excitatory actions are seen in smooth muscles, like those in blood vessels supplying mucous membrane and the skin. This action provides the vasoconstrictor effect, desired while in combination with an LA agent. Finally, it has a peripheral inhibitory action on the smooth muscles of the gut wall and bronchial tree.

Inadvertent intravascular administration, high volumes or high concentrations used or injections into inflamed tissues may potentiate the systemic uptake of vasoconstrictors along with local anaesthetics and produce toxic manifestations.[10] The signs and symptoms of vasoconstrictor toxicity include hypertension (sharp systolic), tachycardia, tremors, headache, perspiration, palpitations from arrhythmias, and rarely ventricular fibrillation by direct effect on the myocardium.[9] According to Malamed, most instances of true epinephrine over-dosage are of such short duration that little or no formal management is required in an ASA I patient.[8] One explanation to this could be the shorter half life of 1–3 minutes for epinephrine.[11] Further epinephrine is largely eliminated from the blood within 10 minutes due to its metabolism by catechol-O-methyl transferase in the blood, liver, lungs and other tissues.[9] This is also an explanation of the 10 minutes duration during which two transient episodes of ventricular tachycardia followed by ventricular bigeminy occurred in our patient.

There appears to be a general agreement that 2% lignocaine with 1:200,000 epinephrine should be used whenever possible specially while using volatile anaesthetics like halothane. Various studies were done comparing varying doses of epinephrine in combination with local anaesthetics and studying their effects on their efficacy, duration of action and toxicity. Kennedy et al concluded in their study that increasing the amount of epinephrine above 1:200,000 does not increase the duration of the block.[12] In fact, lower doses of epinephrine can have same beneficial effects desired of a vasoconstrictor and can also avoid unwanted haemodynamic changes.

Is it LA toxicity or vasoconstrictor toxicity? Ventricular arrhythmias and cardiac arrest are also known side-effects arising from unexpected high plasma levels of LA agents. One must carefully distinguish local anaesthetic toxicity from vasoconstrictor toxicity. Lidocaine has a rapid onset and has a short duration of action of 60–120 minutes after infiltration in plain form.[13] Accidental intravascular injection of local anaesthetics is the most common mechanism of production of excess plasma concentration of local anaesthetics leading to toxicity. Less often, plasma levels of LA can rise from systemic absorption of the LA agent. Factors influencing the systemic absorption of a local anaesthetic from its site of injection are dosage used, site of injection, use of vasoconstrictors and pharmacologic characteristics of the drug.[13] Plasma concentrations of lignocaine producing signs of central nervous system toxicity range between 5–10 μg/ml.[13]

The toxic manifestations arise in a dose dependent manner. The first symptoms and signs are usually neurological with numbness of the mouth and tongue, followed by tinnitus, confusion, seizures and potential coma. Cardiovascular toxicity manifests as tachycardia and hypertension but with increasing toxicity bradycardia and hypotension occurs. The cardiovascular system is typically more resistant to the effect of local anaesthetics than the central nervous system (CNS); that is, the CNS toxic responses occur at lower blood levels than the cardiovascular system toxic responses.[14] In our patient, local anaesthetic toxicity could not be completely ruled out on the basis of absent central nervous system signs and symptoms. One reason for this was that the patient was under heavy sedation of midazolam and promethazine, both of which can raise the threshold for CNS toxicity signs. It is also possible that CNS symptoms were masked due to heavy sedation.

In our patient, the initial event was a cardiac arrest presenting as pulseless ventricular tachycardia, apnoea, unconsciousness and facial pallor. There were no signs and symptoms of CNS excitation like perioral twitching or tinnitus, seizures in our case. The occurrence of cardiac arrest was within a ten minute period after submucosal infiltration of 6 ml lignocaine 2% (120 mg) and 1:200,000 epinephrine (30 μg). Further, the rapid onset and offset of symptoms would likely correlate with epinephrine and not

serum lignocaine levels. The maximum recommended single dose for a 70 kg patient is 500 mg of lignocaine with epinephrine and 300 mg of plain lignocaine.[15] Maximum recommended doses serve as the guidelines in clinical practice to avoid use of excessive doses leading to systemic toxicity, but are of value only if the local anaesthetic is not injected intravascularly.

It is also worth knowing the maximum dosage of epinephrine as recommended by the New York heart association is 0.4 mg (400 μg).[16] Extensive studies have been done to measure plasma catecholamine levels after administering LA with epinephrine, and correlating them with haemodynamic response.[6,7,17] It is interesting to note that after 1.8 ml of 2%lidocaine with 1:100,000 epinephrine (18 μg), there was two-three fold rise in plasma catecholamine levels over baseline without causing any significant haemodynamic response.[6,7] Dionne et al in their studies revealed that increased plasma epinephrine levels five times their baseline values (34 ± 31 pg/ml) were associated with haemodynamic changes.[17] The above studies also revealed that the threshold level of epinephrine for increase in heart rate was 50–100 pg/ml, increase in systolic blood pressure was 75–125 pg/ml and at 150–200 pg/ml it caused decrease in diastolic pressure.

Cardiac arrest must be managed according to the Cardiopulmonary Cerebral Resuscitation (CPCR) guidelines by correct identification of presenting arrhythmia and appropriate treatment. Ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) as in our case needs defibrillation. In rare situations like our case ventricular tachycardia is transient and reverts with a cardiac thump offsetting the requirement of defibrillator which had been kept on stand by, if VT had not reverted with the thump. A precordial thump should be considered only if cardiac arrest (ventricular fibrillation or pulseless ventricular tachycardia) is confirmed rapidly, following a witnessed and monitored ECG and if a defibrillator is not immediately available.[18] In two case series, precordial thump has been found to be effective at reverting VF or pulseless VT to sinus rhythm.[19,20] Yet others have indicated that chest thump can lead to ventricular fibrillation or aggravate the rate of ventricular tachycardia and should be recommended only in settings where external defibrillation is readily available.

The short and reversible episodes of pulseless ventricular tachycardia associated with unconsciousness occurring during the 10 minutes duration period could be explained with inadvertent intravascular epinephrine and/or a larger dose in a highly vascular site of nasal septum. The most likely reason of possible cardio version with precordial thump in our patient is the episode was witnessed and treated within 30 seconds of confirming it and the underlying cause was the short duration rise of epinephrine levels in the blood, in an otherwise normal ejection fraction heart.

Pheochromocytoma[21]and thyrotoxicosis[22] are absolute contraindications to the use of epinephrine. The patients with cardiovascular disease are more prone to cardiac arrhythmias after use of vasoconstrictors, hence must be used with the utmost caution after weighing their risks versus benefit. Also the adrenergic vasoconstrictors may participate in a variety of adverse drug interactions, the most important of which involve tricyclic antidepressants, [23] non-selective beta blockers, volatile inhalational agents and cocaine.

In conclusion, this case highlights the need for vigilance for symptoms of systemic toxicity while using vasoconstrictors like epinephrine with lidocaine, especially in nasal (vascular) regions. Haemodynamic monitoring of blood pressure, ECG, SPO2 during and after infiltration is mandatory. It is essential to know the weight of a patient, as an accurate calculation using well established guidelines of doses, prior to administration, can avoid unwanted toxicity. The principle of using the lowest possible dose of vasoconstrictor to produce the desired action, while minimally affecting the physiology of the patient should be administered. Careful aspiration of the needle each time we inject drugs and injecting a small test dose is the safest method. Some remote chances of vascular exposure cannot be completely avoided as minute movement of needle could occur while injecting drugs. Finally, identifying the signs and symptoms of toxicity and their management is crucial and possible only by keeping a high level of vigilance.

Reference:

1. Sveen K. Effect of the addition of a vasoconstrictor to local anesthetic solution on operative and postoperative bleeding, analgesia and wound heeling. Int J Oral Surg 1979 Aug;8(4):301–6.
2. Braid DP, Scott DB. Effect of adrenaline on the systemic absorption of local anaesthetic drugs. Acta Anaesthesiologica Scand Suppl. 1966;23:334–46.
3. Liu S, Carpenter RL, Chiu AA, McGill TJ, Mantell SA. Epinephrine prolongs duration of subcutaneous infiltration of local anaesthesia in a dose-related manner. Correlation with magnitude of vasoconstriction. Regional Anesthesia 1995 Sep–Oct;20(5):378–84.
4. Lormans P, Gaumann D, Schwieger I, Tassonyi E. Ventricular fibrillation following local application of cocaine and epinephrine for nasal surgery. ORL J Oto-rhino-laryngology Relat Spec 1992;54(3):160–2.
5. V. Srinivasan, R.B.S. Arasaratnam, G A Jankelowitz. Day-case septal surgery under general anaesthesia and local anaesthesia with sedation. The Journal of Laryngology & Otology 1995;109:614–617.
6. Chernow B, Balestrieri F, and Ferguson CD, Terezhalmy GT, Fletcher R, Lake CR.. Local dental anesthesia with epinephrine: Minimal effects on the sympathetic nervous system or on hemodynamic variables. Arch Internal Medicine 1983;143(11):2141–2143.
7. Cioffi GA, Chernow B, Glahn RP, Terezhalmy GT, Lake CR. The hemodynamic and plasma catecholamine responses to routine restorative dental care. J American Dental Association. 1985;111(1):67–70.
8. Malamed S, edition, Handbook of local anesthesia, 1st edition, St Louis, CV Mosby publishing Co, pp 1980;27-35:222–224.
9. Weiner N. Norepinephrine, Epinephrine and sympathomimetic Amines. In; Goodman and Gilman, Eds, The pharmacological basis of Therapeutics, 6th edition, New York, Macmillan publishing Co, 1980;8:138–151.
10. James P. Casidy, James C. Phero, and William H.Grau. Epinephrine: Systemic effects and varying concentrations in Local Anesthesia. Anesthesia Progress 1986 December;33(6):289–297.
11. Guyton AC, ed, Short term regulation of mean arterial pressure. In: Textbook of Medical physiology, Philadelphia, WB Saunders, 12:256, 1981.
12. Kennedy WF, Bonica JJ, Ward RJ, Tolas AG, Martin WE, Grinstein A. Cardio respiratory effects of epinephrine when using in regional anesthesia. Acta Anaesthesiologica Scandinavica 1966;23:320–333.
13. Robert K Stoelting, Simon C Hiller. Pharmacology and physiology in Anesthetic practice. Lippincott Williams and Wilkins Third edition, 1999 section1:158–181.
14. Brendan T. Finucane. Complications of Regional anesthesia. Springer, Second edition 2007;page 61–73.
15. Paul G. Barash, Bruce F. Cullen, Robert K Stoelting, Michael K. Cahalan, M.Christine Stock. Handbook of Clinical Anesthesia, Sixth edition Wolters Kluwer, Lippincott Williams & Wilkins. 2009, section 4;page 308–322.
16. Special Committee of New York Heart Association, Inc: Use of epinephrine in connection with procaine in dental procedures. Journal of American Dental Association 1955;50:108.
17. Dionne RA, Goldstein DS, Wirdzek PR. Effects of diazepam premedication and epinephrine containing local anesthetic on cardiovascular and plasma catecholamine responses to oral surgery. Anesthesia Analgesia 1984;63:640–646.
18. Caldwell G, Millar G, Quinn E, R Vincent, Chamberlain DA. Simple mechanical methods for cardio version: Defense of the precordial version and cough version. BMJ 1985;291:627–630.
19. Volkmann HK, Klumbies A, Kuhnert H, Paliege R, Dannberg G, Siegert K. Terminating ventricular tachycardias by mechanical heart stimulation with precordial thumps. Z Kardiology, 1990;79:717–724.
20. Befeler B. Mechanical stimulation of the heart: its therapeutic value in tachyarrhythmias. Chest 1978;73:832-838.
21. Klingenstrom P, Nylen B, Westermark L. A clinical comparison between adrenalin and octapressin as vasoconstrictors in local anesthesia. Acta Anaesthesiologica Scandinavica 1967;11:35–42.
22. Jastak JT and Yagiela JA. Regional Anesthesia of the oral cavity, St Louis, CV Mosby publishing CO, 1981.
23. Yagiela JA, Duffin SR, Hunt LM. Drug interactions and vasoconstrictors used in local anesthetic solutions. Oral surgery 59:565-571, 1985.