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

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.

Keywords: epinephrine, lignocaine, ventricular tachycardia

Introduction
Vasoconstrictors have been used in combination with local anaesthetic (LA) agents to reduce intraoperative bleeding, reduce the risk of systemic toxicity of local anaesthetics, and prolong their duration of action. 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 ribbon soaked in a solution of 30 ml 4% topical lidocaine and four ampoules of epinephrine 1:1000 (4 μg/ml). 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 88% 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

Systemic actions of epinephrine are produced by effects on alpha and beta adrenergic receptors. The systemic actions can be classified into five broad types. 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 and the gut. Systemic actions of epinephrine include vasocostrictror effect, desired while in combination with an local anaesthetic.

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. The signs and symptoms of vasoconstrictor toxicity include hypertension (sharp systolic), tachycardia, tremors, headache, perspiration, palpitations from arrhythmia, and rarely ventricular fibrillation by direct effect on the myocardium. 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 1 patient. One explanation to this could be the shorter half life of 1–3 minutes for epinephrine. 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. 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. 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 vasocostrictror 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 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.

Plasma concentrations of lignocaine producing signs of central nervous system toxicity range between 5–10 μg/ml. 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. 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.
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. 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). Extensive studies have been done to measure
plasma catecholamine levels after administering LA with
epinephrine, and correlating them with haemodynamic
response.6,7 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.8 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. 9

The above studies also revealed that the threshold level of
epinephrine for increase in heart rate was 50–100 pg/ml,
since 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
Resuscitation (CPR) 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 VF 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.10 In two case
series, precordial thump has been found to be effective at
reverting VF or pulseless VT to sinus rhythm.10,10 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
tachyarrhythmias after use of vasoconstrictors, hence must be used with the utmost
care 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, 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.

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