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COMPLICATION FOLLOWING INADVERTENT INTRAVASCULAR INJECTION OF 2% LIGNOCAINE – EPINEPHRINE: A CASE REPORT

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ABSTRACT Local anaesthesia techniques are widely used in because of their numerous advantages, including safety. They enable procedure to be performed painlessly by reversibly blocking the conduction of the sensory nerves. Added diluted epinephrine is often locally used to provide haemostasis and improve visualization. However, rapid absorption or inadvertent intravascular injection of epinephrine can cause unexpected cardiovascular effects. A case of 47 year ASA 1 physical status female was scheduled for left ear tympanoplasty. Patient experienced uneasiness and chest discomfort following local infiltration of lidocaine 2% with epinephrine 1:200,000 combination. Severe hypertension of 200/126 mmHg, followed by ventricular tachycardia occurred within 2mins.Hemodynamics were got under control by immediate intervention. Within the next 15mins, patient developed coarse crepitation and expiratory wheeze in all lung fields .After successful treatment in the operation theatre ,patient was transferred to ICU for further management. It is important to recognize this potential cardiovascular side-effect in patients administrated with local epinephrine. Drugs used to treat hypertension and/or arrhythmia need to be understood by every physician.

KEYWORDS :

INTRODUCTION

Local anaesthetics with vasoconstrictor agents are widely used especially in ENT surgery. The most commonly used vasoconstrictor agent is Epinephrine[1-14] because it provides many advantages such as reducing toxicity, increasing the anaesthetic effect and improving hemostasis[1-10]

Epinephrine is a catecholamine type vasoconstrictor[9] which has a non-selective adrenergic profile. It causes the reduction of peripheral blood-vessel diameter and stimulates β1 receptors causing increase in heart rate and consequently, blood pressure . For these reasons, the association of a local anaesthetic with a vasoconstrictor agent might result in adverse hemodynamic effects.

Indeed, psychological and emotional factors such as anxiety and stress play significant roles in the physiological responses of patients, potentially leading to adverse hemodynamic and metabolic disturbances due to exaggerated endogenous catecholamine production.[2,6,11]

Despite the beneficial properties of vasoconstrictors, there is some concern regarding systemic consequences due to inadvertent intravascular injection and the induction of adverse cardiovascular effects, primarily in patients with CVD[15,16] . In addition, pain, stress, fear and anxiety during dental treatment that are caused by lack of pain control and poor anaesthesia may be responsible for the systemic endogenous release of catecholamines, particularly norepinephrine[17] which may lead to autonomic responses such as hypertension and arrhythmias[18,19,20]. Hence, the occurrence of most alterations may be attributed to inappropriate applications such as high dose injections, intravascular accidental injections and drug interactions.[19] Thereafter, endogenous or exogenous cathecolamines may cause or contribute to haemodynamic and cardiac changes .

A systematic review has shown that most complications that arise while using local anaesthetics with vasoconstrictors are clinically insignificant arrhythmias and that the use of the anaesthetic agent lidocaine associated with epinephrine in the recommended dosage seems to be relatively safe even for patients with CVD.[20]

The maximum recommended strength of the vasoconstrictor is 1:100,000 epinephrine equivalency for routine nerve block anaesthesia. Systemic absorption is marked by signs of anxiety, elevated blood pressure, increased heart rate, and occasional arrhythmias. These effects can be extremely serious in a patient with cardiovascular disease.

Lidocaine with epinephrine combination is most widely used by surgeons and anaesthesiologists. 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.[21]

We present a ASA I physical status patient who had transient recurrent ventricular tachycardia and acute congestive heart failure after subcutaneous lignocaine epinephrine infiltration in a scheduled tympanoplasty.

Case Report

A 47-year-old woman (height 162 cm, weight 67 kg, body mass index 25.5 kg/m^2) was scheduled for a routine left ear tympanoplasty for left ear CSOM under monitored anaesthesia care (local anaesthesia with intravenous sedation). Her physical status was deemed American Society of Anaesthesiology (ASA) class I, indicating no systemic disease or any smoking habits. Her medical history was otherwise unremarkable and there were no abnormalities in her preoperative electrocardiogram (EKG), blood tests, or chest radiograph images. She denied any allergies and she reported no prior surgeries.

On the morning of surgery, after confirming NBM status and written consent, the patient was taken to the operating room. Monitors were connected to the patient for continuous monitoring of heart rate, oxygen saturation (SPO2) and non-invasive blood pressure (NIBP). Prior to the procedure his heart rate (HR) was72/min, NIBP – 126/84 mmHg, SPO2 – 99%, respiratory rate – 16/min with normal cardiovascular and respiratory system examination. Intravenous access was secured with 20 gauge cannula and intravenous fluid Ringer lactate was started at 80 ml/hr. Intravenous Midazolam 1 mg

and Fentanyl 50mcg were administered for anxiolysis and analgesia respectively.

Dexmedetomidine infusion was started for intraoperative sedation in the operating room. It was infused at 1μg/kg/h for 10 min for loading then at 0.5μg/kg/hr for maintenance.

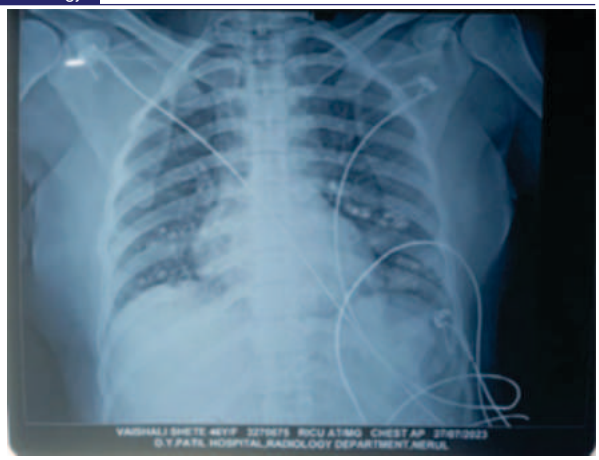
Figure 1After cleaning, painting and draping, the surgeon injected 80 μg of adrenaline (3 mL of 1:80,000 freshly prepared solution by diluting 0.8 mg of adrenaline in 30mL 2% lidocaine) subcutaneously at the incision site behind the pinna after careful negative aspiration of the needle. Within 5-7mins of the injection, the heart rate increased from 76 bpm to 120bpm and blood pressure increased from 124/80 to 155/112 mmHg, respectively (figure 1).

ECG monitor showed a short run of ventricular ectopic. Injection lignocaine hydrochloride (20 mg/ml) 4ml was given and sinus rhythm was present for a short time. Patient went into ventricular tachycardia with HR- 200 bpm. Bp-190/120mmhg. The procedure was stopped and the patient was administered 100% O_2. Dexmedetomedine infusion stopped immediately and an emergency call for a defibrillator and help was made.

Ventricular tachycardia persisted. Injection Amiodarone 150 MG was given IV slowly over 10 mins. Patient responded to amiodarone with intermittent sinus rhythm and alternate premature ventricular ectopics. As her peripheral oxygen saturation decreased to 90%, we assisted the patient's breathing with 100% O2 via bag and mask. Patient's heart rate started falling to 60-65 bpm with a feeble pulse on palpation. Glycopyrolate 200mcg was given intravenously following which heart rate raised to 118bmp with sinus tachycardia. Once stabilized, patient was moved to the post-anaesthesia care unit (PACU) and monitored by standard monitoring procedures. Lead II in ECG monitor showed sinus tachycardia.

Within 10 mins of shifting to PACU, patient had sudden onset of cough and shortness of breath but maintained normal sensorium. Auscultation revealed coarse crepitations and expiratory wheeze in all lung fields. There were no signs of airway obstruction. O_2 saturation (SpO_2) was 90-95% at 10L O2 via face mask. BP decreased to 75/40 mmHg with a HR of 160-165 bpm.

A provisional diagnosis of acute congestive heart failure was made. Inj. furosemide 20mg IV bolus was given. A continuous infusion of Injection noradrenaline at 0.5□g/kg/min and Injection furosemide 4ml/hr were initiated. In the meantime, a urinary catheter was inserted and blood samples were drawn for blood gas analyses with electrolytes and cardiac markers. Patient was immediately shifted to the intensive care unit for further management. Immediate post stabilization chest xray showed signs of pulmonary edema in bilateral lower lung fields (figure 2). Cardiac markers were negative for MI and ECG was normal.



Over a period of 2hr, the crepitations and wheeze gradually improved. Blood pressure was now maintained at 110/75 mmHg with noradrenaline support. Saturation was maintained at 97% on 8L O2 via face mask and urine output was 70-80ml/hr. Her ABG and electrolytes were normal. Echocardiography revealed a normal ejection fraction of 55% and a normal study. Over next 24hours , infusions were slowly tapered and stopped. Patient was discharged after 2days in a vitally stable condition.

DISCUSSION

Vasoconstrictors are widely often used in local anaesthetic solutions. The vasoconstrictor most commonly used is epinephrine.

Epinephrine offers many advantages, such as reducing toxicity, increasing the anaesthetic effect, and improving haemostasis, making it a useful tool for intra-operative bleeding control.[1-9,11]

It is a common practice to infiltrate skin and subcutaneous tissue with adrenaline prior to incision in an attempt to decrease the vascularity of the tissues, which helps reduce blood loss and improve the surgical field view while operating on a vascular field like head and neck surgeries. The maximum recommended dose of adrenaline for infiltration is 5-10 μg/kg. However, in surgical procedures where local tissue haemostasis is needed, such as in ENT surgery, clinicians may choose to use a local anaesthetic solution containing 1:100,000 - 1:200,000 epinephrine to achieve better haemostasis.

However, it must be taken into consideration that this haemostatic effect has been associated with delayed wound healing, an increased risk of infection and potential harmful effects on soft tissue flaps due to decreased blood flow.[22] Liu W, et al.[10] produced a systematic review that focused on adverse drug reactions (ADRs) associated with the use of local anaesthetics .They found that ADRs related to local anaesthetics with epinephrine are 2.16 times higher than those associated with plain local anaesthetics, suggesting the need to be cautious when using a vasoconstrictor agent. They also explained that adrenergic reactions are often misdiagnosed as allergic reactions and that psychological stress due to the local anaesthetic (LA) injection could be a contributory factor. This stress can lead to sympathetic activation, which is correlated with increased levels of stress hormones and metabolites in the blood.

Systemic absorption of epinephrine from local anaesthetic solutions can manifest with signs of anxiety, elevated blood pressure, increased heart rate, and occasional arrhythmias. These effects can be particularly serious in patients with cardiovascular disease or those taking medications that affect adrenergic activity, potentially exacerbating

cardiovascular symptoms or complications. Thus, careful monitoring is required during infiltration.

A proposed possible mechanism of cardiac arrest or pulmonary edema -systemic hypertension that resulted from absorbed topical vasoconstrictor could increase left ventricular afterload and decrease cardiac output, leading to cardiac congestion and left heart failure.

Epinephrine's effect on plasma potassium levels is biphasic, with an initial transient rise followed by a more prolonged decrease. Understanding these effects is crucial for managing patients who receive epinephrine, particularly in emergency or surgical settings where close monitoring and rapid intervention might be necessary.[23] Acute severe hypokalaemia can indeed lead to serious cardiac arrhythmias, including ventricular tachyarrhythmia , even in patients without pre-existing heart disease [24], due to its effects on cardiac repolarization, conduction, and pacemaker activity.[25]

Careful injection technique and knowledge of the anatomy of the area to be anesthetized should minimize the occurrence of overdose reactions. Factors necessary to prevent this complication include the use of an aspirating syringe, use of a needle no smaller than 27-gauge, aspiration in at least two planes before injection and slow administration. Several studies have demonstrated that it is not possible to aspirate consistently with a needle gauge smaller than 25.[11] Small-gauge needles occlude more easily with tissue plugs or with the wall of the blood vessel than do larger-gauge needles, leading to false-negative aspirations.

In our case, the surgeon slowly injected 80 μg of adrenaline for infiltration. It is indeed concerning when an adverse cardiovascular event occurs following the administration of a low dose of adrenaline, especially when it is within the recommended dosage guidelines. It could be due to accidental intravascular placement of the drug. Repeated aspirations while injecting are recommended. Also, 1:80,000 adrenaline was used as compared with the standard recommended concentration of 1:100,000-1:200,000.

In conclusion, the complex cardiovascular interactions of lidocaine and epinephrine, must be appreciated in order to prevent severe complications such as pulmonary edema and cardiac arrest. Both surgeons and anaesthesiologists should be familiar with this possible cardiovascular side effect in patients administrated with local infiltration of epinephrine. Hence, one should be cautious while adrenaline is being injected.

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