



Unusual Case Report : Ventricular Tachycardia, Pulseless Electrical Activity (PEA) Due to Inadvertent Intravascular Injection of Lignocaine With Adrenaline During Tympanoplasty

KEYWORDS

Epinephrine , ventricular tachycardia , PEA , CPR

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ABSTRACT A 33yr old female , a case of CSOM was posted for elective surgery for tympanoplasty under GA in ASA Grade I . The patient after induction of GA was given Inj. Lignocaine 2% with adrenaline as infiltration at the site of surgery who developed Ventricular tachycardia(VT) followed by pulseless electrical activity .

INTRODUCTION :

Vasopressors have a long history of use as a haemostatic . Infiltration of adrenaline with lignocaine has been frequently used in Ear and Nasal surgeries as a routine along with general anaesthesia. It act as a vasoconstrictor when used alongwith local anaesthetic (LA) thereby reduces the absorption of LA prolonging the duration and intensity of block and also prevent toxicity .

CASE REPORT :

A 33yr old female with a H/O hearing loss for one yr , mucopurulent discharge diagnosed as bilateral CSOM was taken up for Rt. Tympanoplasty in ASA Gr I under GA alongwith local infiltration of 2% lignocaine with adrenaline. Pt. was premedicated with glycopyrrolate 0.2 mg midazolam 1mg, ondansetron 4mg & fortwin 30 mg . Pt was put on monitor for NIBP, Pulse oximetry, ECG , ETCO₂ ,before induction of GA . After preoxygenation for 03 min .GA was induced with Thiopental sod. 250 mg. suxamethonium 100mg. , orotracheal intubation with 7.0 mm cuffed tube, . Maint. was done with vecuronium 4.0 mg, O₂ , N₂O , and sevoflurane 1.5% . After painting , drapping surgeon infiltrated 5 -6 ml of lignocaine2% with adrenaline (1:80000) preformed sol at the site of surgery. Within 5-7 min. of infiltration pt. developed monomorphic ventricular tachycardia of 220- 230/ min & BP of 220/130 mm of Hg. Immediately pt. was given a bolus of xylocard 2% 60 mg , midazolam 2mg and ventilation contd. with 100% oxygen. Pt. continued to have tachycardia of 180-200/min , BP of 200/106 with ST depression,rhythm changing to sinus. IV metoprolol 1mg . Ringer lactate 1lit was given . saturation was maintained at 99-100%. Within 8 - 10 min. pulse was not felt , ECG showing sinus rhythm, No saturation, No ETCO₂. PEA was diagnosed and cardiac compression of atleast 100/min started . Radial Pulse gradually returned with BP of 60/40 mmof Hg. Dopamine infusion of 5 microgm. /kg/ min started. Consciousness returned with return of spontaneous resp. efforts with BP of 100-120/80-90 mmof Hg.

In view of a cardiac event (PEA) it was decided to electively ventilate the pt. . Pt was shifted To ICU with ventilatory support and advised 12 lead ECG , ABG, Trop T, & physcian opinion. ECG showed signs of global ischaemia , and 2D ECHO showed hypokinesia of LV apex , IVS, ant. Wall, LVEF -40%, No clots/ vegetation. Pt was extubated after 36 hrs. On extubation pt was fully conscious, p 100/ min BP 110/70 mm of Hg. with no inotropic support. Pt remained asymptomatic for almost 30hrs when she devel-

oped difficulty in breathing restlessness, and developed Left ventricular failure with saturation of 60-70% , p 150/ min, BP 140/90,resp. rate of 40/min . chest showed bil. Creps . . Pt was reintubated , ventilated and LVF was treated.

Pt. was shifted by relatives to other hospital where she was on ventilatory and cardiac support for 2-3 days. At the time of discharge 2D ECHO showed mild dilated LV , thinning of ant. Wall, IVS, severely compromised LV systolic function with EF of 25% . No pulm. Hypertension, no thrombus/pericardial effusion. Subsequently discharged with Diagnosis of Post -Adrenaline PEA with Cardiogenic shock with Left Ventricular Failure due to Anteroseptal Myocardial Infarction

DISCUSSION

Ventricular tachycardia (1, 2, 3) / ventricular fibrillation , PEA after accidental intravascular injection of adrenaline is a known entity, . Recommended dilutions are 1: 200000 to 1:400000 . Sudden tachycardia leads to polymorphic VF/ VT , PEA . Management of tachycardia may require midazolam , lignocaine 2% IV , Betablockers (ismolol , Metaprolol) . Or if not responding to pharmacological means then D C Shock . Ones effect of adrenalie wears of hypotension becomes predominant and may require inotropic support as in our case .

For the purpose of improving operative vision, a local infiltration of epinephrine is often carried out by mixing it with physiological saline or local anesthetic, and a side effect rarely takes place with the clinical dose. However, as in this case report, there are a few other cases where cardiovascular collapse took place by a little amount of epinephrine local infiltration.

Wanamaker et al. [4] reported the case where severe hypertension and tachycardia took place after the hypodermic injection of 1% lidocaine 3 ml mixed with 1 : 100,000 epinephrine for tympanoplasty, followed by ventricular tachycardia and ventricular fibrillation, and thus cardioversion was required. Woldorf and Pastore [5] also reported the case where pulmonary edema, as well as severe low blood pressure with the systolic blood pressure lower than 30 mmHg and tachycardia, took place after the infiltration of 1% lidocaine 5 ml mixed with 1 : 200,000 epinephrine to the gingivobuccal fold mucosa. In our case report, a hypodermic injection of 2% lidocaine 4 ml mixed with 1 : 80000 epinephrine was carried out, dividing the

total amount into several times, into the part around the retroauricular mastoid, . The total amount of epinephrine used in the quoted cases was 30 µg and 25 µg, respectively. The maximum allowable dose for adults is the maximum therapeutic dose of 0.5-1 mg and the minimum lethal dose of 4 mg for hypodermic injection, which are a little amount compared to the maximum allowed dose of 7-8 mg (6). In addition, considering that the maximum safe dose of epinephrine can be increased due to the endogenous vasoconstriction and the protective effect of lidocaine that is jointly used [7], the amount in this case was insufficient to cause cardiovascular collapse, even though it is assumed that all the injected epinephrine was immediately absorbed in the linear fracture at the temporal bone or the vein.

A few factors were presumed as the causes of the electrocardiographic disorder by the tiny amount of epinephrine in this case report.

The possibility that epinephrine caused hypersensitization in the body cannot be ruled out. Carter et al. [8] measured the discharged epinephrine and the metabolites after an epinephrine hypodermic injection and reported that the detected amount was 2 times more than the expected value. They mentioned the possibility of endogenous catecholamine hypersensitization by the externally injected catecholamine. However, this explanation might be valid only for the clinical pattern when hundreds of small quantity epinephrine units are injected.

On the other hand, the studies that compared the vasoconstriction effect depending on the ratio of local anesthetic revealed that there was no significant clinical difference, although a wide range of concentrations were tested from 1 : 50,000 to 1 : 400,000. If so, the use of a lower concentration of epinephrine for the vasoconstriction effect can be helpful in reducing various complications.

Inhalation anesthetics can also affect the occurrence of arrhythmia by a little amount of epinephrine injected from outside. However, this can hardly be the cause of the ventricular arrhythmia since sevoflurane, which was used in this case report, shows a low epinephrine-induced arrhythmia incidence rate, although it has a negative myocardial inotropic effect similar to that of isoflurane and desflurane [12].

To prevent the epinephrine-induced cardiovascular crisis, a patient's family and personal history regarding cardiovascular diseases, cryptorrhea, and medication should be thoroughly investigated before the operation and attention should be paid to the ventilation, blood pressure, heart rate, and the heart rhythm during the operation.

The therapy for epinephrine-induced cardiovascular crisis is symptomatic and similar to the therapy for pheochromocytoma. For the treatment of severe hypertension, α-adrenergic blockers such as phentolamine are recom-

mended, and β-adrenergic blockers are recommended for the treatment of tachycardia. Esmolol, the short acting β₁-selective blocker, is preferred because it has a short half-life; it can be used by volume titration depending on the heart rate; and it can reduce the risk of hypertension and coronary spasm due to the excessive α-stimulation that is found in nonspecific β-blockers [10]. Calcium-channel blockers, such as verapamil and diltiazem, are also used for hypertension, tachycardia, and arrhythmia. The cardiopulmonary resuscitation algorithm is practiced for the treatment of arrhythmia and cardiac arrest. In this case, since ventricular arrhythmia was found a few seconds after tachycardia and hypertension, lidocaine, which has been used for a long time with a less immediate adverse reaction, Amiodarone affects the sodium channel, potassium channel, and calcium channel, and it has a blocking effect on α and β sympathetic nerve receptors. It is injected in ventricular fibrillation or ventricular tachycardia patients who are not responsive to cardiopulmonary resuscitation, electroshock, or vasoconstrictor by an initial intravenous injection of 150 mg for 10 minutes, and the daily maximum allowance is 2.2 g.

Pulseless electrical activity (PEA) (11) is a clinical condition characterized by unresponsiveness and lack of palpable pulse in the presence of organized cardiac electrical activity. Pulseless electrical activity has previously been referred to as electromechanical dissociation (EMD). PEA can occur in many different forms- along with sinus brady /tachycardia . . Absence of carotid/femoral pulsations with ECG rhythm on monitor can identify PEAs . Treatment includes cardiac compressions which was started immediately as in our case and adrenaline if required. PEA occurs when a major CVS , respiratory, metabolic derangements results in inability of cardiac muscle to generate sufficient force . It is always caused by profound CVS insult . The initial insult weakens cardiac contractions which is increased by worsening acidosis , hypoxia and increased vagal tone and leads to inadequate mechanical activity even though electrical activity is present . Overall prognosis is poor and poorly understood entity. (12)

Pt. with PEA as first documented rhythm has lower rate of survival to discharge than pts. Who had VT/VF as first documented rhythm as was in our case report.

In conclusion, this case shows that cardiovascular crisis, such as arrhythmia and cardiac arrest (PEA), took place after a small amount, clinical volume of epinephrine was injected in a healthy patient without any heart disease. Adrenaline is a very effective vasoconstrictor to be used alongwith local anaesthetics but precautions must be taken with regards to intravascular injections , excess dosages, desired concentrations . Proper monitoring in all cases irrespective of young/old, is mandatory . Facilities for providing early recognition and treatment by means of ventilatory support & ACLS is inescapable and will lead to negligible morbidity and mortality .

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