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Local turned into Lethal anesthetics- A Surgeons horror

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Abstract

A 26 yr old male patient developed cardiogenic shock following instillation of local anesthesia (lignocaine+ adrenaline) given for tooth extraction. Intubated, resuscitated and was referred to higher center. Echocardiography was done showing global hypokinesia with EF-30%. Patient was managed conservatively and echo was repeated before discharge which showed persistent findings; indicating that the patient was having previous underlying cardiac abnormality.

Key words : Lignocaine, epinephrine.

Introduction :

Local anesthesia is a very frequent procedure in daily practice[1]. Lignocaine with epinephrine combination is most widely used by surgeons.

Toxic plasma concentration of local anesthetic (resulting from systemic absorbtion) is associated with adverse cardiovascular effects (such as bradycardia, hypotension and cardiovascular collapse, cardiac arrest)[2]. It is necessary to carefully monitor cardiovascular and respiratory vital signs after each dose of local anesthetics. In most studies it was not possible to distinguish the real cause of the observed disorder.

We present a young patient who developed cardiovascular collapse after submucosal lignocaine epinephrine infiltration in scheduled tooth extraction.

Case Report :

A 26 year old male presented to casualty with history of sudden loss of consciousness following submucosal injection of Local anesthetic (lignocaine+adrenaline) given for tooth extraction. Patient was apparently normal before the procedure. Baseline cardiac evaluation was not done before giving L/A. As soon L/A was given patient collapsed, patient was given CPR and intubated and was referred to our hospital for further management. On examination pulse was 110/min, BP-90mmhg systolic and GCS 3/15.

Chest was clear and CVS normal . ECG showed ST depression in lead V2-V5. ABG was normal. 2Decho was done which showed global hypokinesia with ejection fraction 30%.

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Patient was managed conservatively on mechanical ventilation. By 3rd day patient was extubated and showed dramatic improvement. 2DEcho was repeated just before discharge which showed no improvement in ejection fraction, indicating that patient had underlying cardiac problem since before.

Discussion :

Although historically a safe and effective means of anesthesia[3]. Cardiovascular toxicity from locoregional anesthesia has been known for over three decades[4]. Most commonly used local anesthesia is lignocaine and epinephrine. The main purpose of vasoconstrictor (epinephrine) is to reduce systemic toxicity of a local anesthetic agent[5].

There are chances of rapid absorption of local anesthetic following inadverent intravascular injections[6]. Lignocaine is a tertiary amine that is an amide derivative of diethylaminoacetic acid. The onset of anesthesia , the duration of anesthesia and degree of muscular relaxation are proportional to the volume and concentration of LA used. The half life of lignocaine is 1.6 hours[9]. Therapeutic levels of lignocaine are 1–5 μg/mL. Toxicity may sometimes occur at therapeutic drug levels. Serious poisoning may occur at lignocaine levels above 5μg/mL. Lidocaine toxicity affects many systems[10,11].

In cardiovascular system, it depresses cardiac conduction and contractility, causing dysrhythmias (sinus bradycardia, atrioventricular junctional or ventricular bradycardia, second or third-degree heart block, asystole, and rarely ventricular tachycardia or fibrillation resulting from re-entrant mechanisms)[12,13].

The half life of epinephrine used along with LA is about 1-3 min[8]. Further epinephrine is largely eliminated from the blood within 10min due to its metabolism by catecola O methyl transferase in the blood, liver, lungs and other tissues[7].

In our patient, the initial event was a cardiac arrest presenting as pulseless ventricular tachycardia, apnea, 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 20 minute period after submucosal infiltration of 6 ml lignocaine 2% (120 mg) and 1:200,000 epinephrine (30 μg). Which likely correlates to be of lignocaine induced cardiogenic shock.

Cardiac arrest must be managed similarly as is managed in cardiac arrest due to different causes, 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 standby, 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. In two case series, precordial thump has been found to be effective at reverting VF or pulseless VT to sinus rhythm[14]. 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.

It is also important to know any cardiovascular disease if the patient is suffering from as they are more prone to develop cardiovascular complication, as in our case the patient was having underlying cardiac abnormality. So it is important to take minimum of ECG to rule out cardiac abnormality.

Conclusion : To conclude, this case highlights the need for vigilance for symptoms of systemic toxicity while using lignocaine. 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. It is also important to evaluate underlying cardiac abnormality if any.

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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