**Adrenaline Induced Pulmonary Oedema**

Sir,

Many ENT surgeries are done under local anaesthesia, where lignocaine is combined with adrenaline and infiltrated. Adrenaline is the preferred vasoconstrictor in combination with local anaesthetic agents acting as a haemostatic agent, constricting capillaries and providing a clear field. Also by delaying systemic absorption it increases the amount of local anaesthetic agents which can be used safely. We want to report a case of Adrenaline toxicity which caused ventricular tachycardia and pulmonary oedema.

A 28 year old patient was being operated for unsafe ear under local analgesia (Lignocaine plus Adrenaline) by ENT department. A call to Anaesthetist went for acute rise in blood pressure. On reaching the following signs were found: BP: 210/130 mm Hg, Pulse 38-40/minute and patient complaining of headache.

A provisional diagnosis of Adrenaline toxicity was made and multiparameter monitor attached. Ventricular tachycardia was noted and loxicard (Preservative free Lignocaine) 1.5 mg/kg i/v slow was given and esmolol 20 mg i/v slow was given for acute treatment. Patient had also received injection pentazocine i/v 30 mg before LA. Patient was ventilated with 100% O2 and ECG returned normal. Fluids were rushed in anticipation of withdrawal hypotension. Patient recovered well but with hypotension (85/56mmHg) and hypoxia ($SpO_2$ 85%). So fluids were given and patient given 100% $O_2$. Soon patient started having persistent hypotension and hypoxia with arrival of basal crepts. Also patient started having pinkish frothy secretions. So considering adrenaline induced pulmonary oedema following were given:

Inj. Lasix 20 mg I/v slow under cover of Dopamine titrated drip, then 4hourly 10 mgi/v; Inj Morphine 6mg i/v slow, then 3 mgi/v 4hourly; 100% oxygen through NIV mask with pressure support of 15 mmHg; and Inj. Hydrocortisone 200 mg i/v. Patient was shifted to ICU and kept on this treatment for 12 hours. Patient recovered well and was shifted to ward after 18 hours and then discharged.

Vasoconstrictors, since cocaine[1] have been traditionally used along with local anaesthetic agents to provide a clear field. Many agents are used like adrenaline, nor-adrenaline, oxymetazoline, vasopressin analogues, etc, but adrenaline is most commonly used.

Adrenaline toxicity was kept as the first possibility due to signs and symptoms (volume and concentration of adrenaline injected was not revealed). The $a_1$ action causes vasoconstriction, increasing systemic and pulmonary resistance, whereas $ß_1$ causes tachycardia and increased myocardial contractility. Increased blood pressure and headache (perfusion headache) were due to either intravascular or excess adrenaline injection. A dose of 3mcg/kg up to a maximum of 200 mcg has been considered as a safe dose in healthy patients.[2] By minimizing the concentration of epinephrine, we may be able to attenuate the effects of accidental intravascular injection or rapid systemic absorption of vasoconstrictors. It has been recommended in head and neck surgery that epinephrine in concentration of 1: 2,00,000 or 1:4,00,000 be used for optimal hemostasis.[3] Most clinical evidence suggests that increase the epinephrine concentration beyond 5mcg.$ml^{-1}$ (1:2,00,000) does not result in a stronger vasoconstriction effect, but does increase toxic circulatory side effects.[4] Tachycardia leads to catecholaminergic polymorphic

ventricular arrhythmia which can also cause dizziness, collapse, anoxia and death.

Pulmonary oedema is known to be caused by excess adrenaline.[5] Tachycardia and increased systemic resistance causes excess load on left ventricle, causing pulmonary congestion. This is accompanied with mismatch in increased pulmonary artery pressure compared with pulmonary alveolar pressure, causing hydrostatic flux of fluid. Further it is found in studies that excess adrenaline causes changes in endothelial and both types of Clara cells leading to toxic lung injury causing pulmonary oedema.[6]

Treatment is for the condition as it develops, for hypertension and tachycardia. ß- blockers like esmolol can be used. Once ventricular tachycardia develops then lignocaine (1.5-2.5 mg/kg) is very effective. For pulmonary oedema standard treatment of lasix, 100% oxygen with positive pressure ventilation, propped up position and morphine is used. Steroids in moderate doses have also been recommended.

Once effects of adrenaline ceases (in 15-30 minutes) and pulmonary oedema ensues hypotension becomes predominant, which may require inotropic support like dopamine.

To conclude adrenaline is a very effective vasoconstrictor to be used along with local anaesthetic agents but precautions must be taken with regard to intravascular or excess injection. Other vasocaonstrictors like oxymetazoline have been considered by some to be safer, hence can be used specially in conditions where adrenaline is more risky. Intuitive and quick treatment leads to negligible mortality rates due to adrenaline toxicity.

## REFERENCES

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**Kartik Syal, Anil Ohri, J. R. Thakur**

*Indira Gandhi Medical College & Hospital, Shimla, H.P.*
*Correspondence: Dr. Kartik Syal*
*Email: kartik.syal@gmail.com*