CASE REPORT

Acute massive pulmonary edema and myocardial infarction after intranasal infiltration of adrenaline during septorhinoplasty

Indira Kumari, MD*, Udita Naithani, MD**, Sandeep Arora, MBBS***, Pradeep Gupta, MBBS***

*Professor, **Associate professor, ***PG Student
Dept of Anaesthesiology, R.N.T. Medical College, Udaipur (Raj). India

Correspondence: Prof, Indira Kumari, Dept of Anaesthesiology. R.N.T. Medical College, Udaipur (Raj). India; E-mail:

ABSTRACT

We present a case report of 13 years old male child undergoing septorhinoplasty under general anesthesia, who developed acute massive pulmonary edema following intranasal infiltration of 330 micrograms of inj. adrenaline by the ENT surgeon. Echocardiography showed local wall hypokinesia with ejection fraction (EF) reduced to 20% and raised troponin-T levels (10 times of normal) suggesting it was adrenaline induced acute myocardial infarction and subsequent cardiogenic pulmonary edema. The surgery was postponed and the patient was successfully treated in ICU with positive pressure ventilation, frusemide and inotropice support. His EF returned to 50% at 5hr and to 70% at 10 hr; and he was extubated after 14 hours and discharged after 5 days.

Key Words: Cardiogenic pulmonary edema; Acute myocardial infarction; Adrenaline; APE (adrenaline induced Pulmonary edema).


INTRODUCTION

During nasal surgery vasoconstrictors have been used as hemostatic agents. Currently adrenaline (epinephrine), noradrenaline (norepinephrine), phenylephrine, vasopressin or oxymetazoline are being used by ENT surgeons for vasoconstriction1 of the nasal mucosa. Most commonly adrenaline is used with lignocaine to pro vide bloodless surgical field and to prolong the action of local anesthetics, but its use may produce complications due to its agonist action on both alpha and beta receptors. Adverse reactions of adrenaline can range from agitation, restlessness, headache, tachycardia & hypertension leading to life threatening myocardial ischemia, ventricular arrhythmia, cerebral haemorrhage and pulmonary edema.2 Adrenaline induced acute cardiovascular crisis in the form of myocardial infarction,3 ventricular tachycardia4,5 and pulmonary edema5 have been reported in adults and children6. Data are
unavailable for the total dose of use of this drug by ENT surgeons.

We present the case of a 13 years old male, ASA-I, child undergoing elective septorhinoplasty under general anaesthesia, who developed acute massive pulmonary edema and myocardial infarction after intranasal infiltration of lignocaine with adrenaline by the ENT surgeon.

**CASE REPORT**

A 13-year-old, 30 kg boy was scheduled for elective septorhinoplasty under general anaesthesia. Pre anaesthetic examination was unremarkable. After taking written informed consent from parents; peripheral line was secured and Ringer lactate infusion started. His noninvasive blood pressure (NIBP) was 120/80 mmHg, heart rate 110/min and SpO₂ to be 99% with normal rhythm on cardiac monitor. Patient was premedicated with glycopyrrolate 0.1 mg, ondansetron 2 mg and tramadol 60 mg. Anesthesia was induced with thiopentone 150 mg and succinylcholine 50 mg, the trachea was intubated with a Portex™ cuffed endotracheal (ET) tube, size 6 mm I.D. and oropharyngeal packing was done. Anesthesia was maintained with 50:50 N₂O and O₂, atracurium (15 mg), propofol infusion (50-100 mcg/kg/min) and intermittent positive pressure ventilation (IPPV) through a Bain's circuit. The ENT surgeon infiltrated nasal mucosa with adrenaline injection in the nose in order to achieve a bloodless surgical field. For this purpose, one ampoule of adrenaline (1 ml of 1:1000 = 1 mg) was added to 30 ml normal saline in bowl (33.33 mcg/ml). A total of 10 ml of this solution (330 mcg) was used. Immediately after injection heart rate (HR) raised to 170/min. After a few minutes increased resistance to ventilation was noticed, SpO₂ decreased to 78% and pink frothy secretions started filling the ETT. BP decreased to 80/50, HR rose to 180/min and SpO₂ fell to 90%. Chest was full of bilateral crepts and massive pink frothy pulmonary secretions filled the Bain's circuit inspite of repeated suctioning through the endotracheal tube. N₂O was switched off and the patient ventilated with 100% O₂. Patient was propped up; IV infusion was stopped, urinary bladder was catheterized, frusemide 20 mg, dexamethasone 8 mg, hydrocortisone 100 mg were given. Cardiologist was called and ecocardiography was done in OR, which revealed local wall hypokinesia and an ejection fraction (EF) of 20%; however, all heart valves were normal, and no associated congenital cardiac anomaly was seen. Diagnosis of cardiogenic pulmonary edema was made. Digoxin 0.5 mg and dopamine infusion 7 mcg/kg/min was started to maintain systolic blood pressure (S.B.P) > 90 mmHg. The surgery was postponed, the patient was shifted to cardiac ICU and electively ventilated with 100% of O₂ in assist control mode, with tidal volume (TV) 300 ml, respiratory rate (RR) 15/min, and positive end expiratory pressure (PEEP) 5 cm H₂O on Puritan Benett® ventilator. Inj. vecuronium and midazolam were administered during ventilation. Blood sample was sent for haemogram, complete blood count (CBC), liver enzymes, urea, creatinine, sugar, electrolytes and troponin-T. Arterial blood gas (ABG) analysis could not be done as ABG machine of hospital was not in working condition at that time. After 1 hour, SBP was maintained to 100 mmHg, dopamine infusion was switched over to dobutamine 5 mcg/kg/min, SpO₂ was 98% and heart rate (HR) 130/min. FIO₂ was reduced to 60%. After 5 hours, a repeat echocardiography showed EF of 50%, which rose to 70% at 10 hours. After 12 hours ventilation, midazolam and vecuronium were stopped, and the patient was weaned off ventilator over 2 hours of synchronized intermittent mandatory ventilation (SIMV) and bilevel positive airway pressure (BiPAP) ventilation. Neostigmine 2 mg and glycopyrrolate 0.4 mg were given for reversing the residual neuromuscular blockade. Patient was extubated at 14 hours. Blood investigation reports came normal except troponin-T, which was 0.1 ng/ml (10 times of normal value; normal value=0.01 ng/ml, >0.03 ng/ml is diagnostic of myocardial infarction). He was observed for 48 hours and discharged uneventfully after 5 days.
DISCUSSION

Nasal surgery is always associated with bleeding and to establish a bloodless surgical field, vasoconstrictors like adrenaline are instilled into the nasal cavity in the form of nasal drops, pledgets or submucosal infiltration. Lidocaine 2% solution containing adrenaline in a concentration of 1:200,000 or 1:00,000 (0.001%) is usually used for infiltration to provide local anesthesia and vasoconstriction. In surgeries conducted under general anesthesia, surgeons sometimes use adrenaline diluted in saline for infiltration to provide vasoconstriction as there is no need of local analgesia.

Adrenaline acts on both alpha and beta adrenergic receptors of tissues innervated by sympathetic nerves and may cause adverse reactions, e.g. fear, anxiety, restlessness, irritability, headache, nausea, vomiting, urinary retention, hypertension, palpitation, tachycardia, angina and cardiac arrhythmias. Severe hypertension leading to cerebral hemorrhage, pulmonary edema, myocardial infarction and ventricular fibrillation may cause death. Usual recommendation for safe dosage of adrenaline suggests that doses of adrenaline should not exceed 3 mcg/kg for healthy patients. In the present case, 330 mcg adrenaline was infiltrated intranasally that far exceeded the recommended dose limit and caused toxic side effects. The effect of adrenaline causes tachycardia; excess effect causes coronary spasm, marked vasoconstriction leading to pulmonary hypertension and disruption of alveolar capillary membrane and massive pulmonary edema. In our patient, pulmonary edema and coronary spasm also contributed to decreased O2 supply, thus supply-demand ratio of myocardium got disturbed resulting in myocardial ischemic insult as evidenced by raised troponin-T, local wall hypokinesia and a reduced ejection fraction. There are case reports in the literature where fatal complication due to systemic absorption of locally administered or injected adrenaline given to reduce bleeding has resulted in significant mortality. Submucosal intranasal application of epinephrine soaked pledgets under general anesthesia produced ventricular tachycardia, pulmonary edema, cardiogenic shock, that was successfully treated by supportive therapy as in our case.

Adrenaline induced pulmonary edema was first reported in 1971, where two healthy male patients undergoing elective surgery developed pulmonary edema following the inadvertent injection of large doses of adrenaline. Both patients showed an immediate and dramatic response to treatment with chlorpromazine. Similarly a 27-year-old male; who was drug addict, developed pulmonary edema and severe metabolic acidosis following accidental IV injection of eye drops containing 20 mg adrenaline, responding well to symptomatic treatment. In another case report, an emergency doctor injected 1 mg of undiluted adrenaline in place of morphine by mistake, the patient immediately collapsed and pulmonary edema developed. He was treated successfully with frusemide, nitroglycerine and morphine.

Acute cardiovascular crisis with hypertension, tachycardia, pulmonary edema and cardiac arrest occurred with clinical volume of subcutaneous infiltration of 1:200,000 adrenaline in 2% lidocaine. Similarly during adenotonsillectomy local adrenaline infiltration caused pulmonary edema and intracranial hemorrhage with bulbar paralysis. Intranasal adrenaline induced ventricular tachycardia in endoscopic sinus surgery and adrenaline induced myocardial infarction in patients having normal coronary arteries has also been reported.

Experimental studies on dogs have proved that IV adrenaline produces acute pulmonary edema. A series of experiments in rats showed that adrenaline, noradrenaline and methoxamine induce acute pulmonary edema (APE) but not isoproterenol; so they exclusively connected APE production to alpha adrenergic action of these agents; phenoxybenzamine was found to have protective effect. In contrast another experimental study in mice demonstrated that blockade did not show significant
effect on APE, whereas, beta blockade prevented APE in mice so they concluded that beta adrenergic receptors seem to have a role in production of APE in mice.  

Because of the rapid onset and short duration of adrenaline action, severe toxic reaction due to the accidental administration of an overdose or to hypersensitivity should be treated with an immediate IV injection of a quick-acting alpha-adrenergic blocking agents, such as 5-10 mg of phentolamine or phenoxybenzamine 20 mg; followed by a beta blocking agent such as 2.5-5 mg propranolol. Amyl nitrite, glyceryl trinitrate, chlorpromazine, tolazoline and trimetaphan have also been used as alter natives in an emergency.  

In present case, we did not give alpha or beta blockers since patient developed hypotension immediately after pulmonary edema due to myocardial ischemic insult, that needed dopamine support switched over to dobutamine later on. Dobutamine has 1 and 2 action to provide inotropic and vasodilator effect respectively.  

We suggest that diluted lidocaine 2% solution containing adrenaline should be used for vasoconstriction. Since lidocaine provides a safeguard against adrenaline induced ventricular arrhythmias and also limits the dose of adrenaline. By minimizing the concentration of adrenaline, effects of accidental intravascular injection or rapid systemic absorption can be attenuated. In head and neck surgery adrenaline in concentration of 1:200,000 or 1:400,000 is recommended for optimal hemostasis. Clinical evidence suggests that increasing the concentration beyond 5 mcg/ml(1:200,000) does not result in a stronger vasoconstrictor effect but may increase toxic circulatory effects. Alter natively nasal decongestants such as oxymetazoline, a selective ±1 agonist, can be used. It has been suggested that each hospital should form an advisory committee for adrenaline and local vasoconstrictor use to monitor dosing of these drugs, the resulting rhythm and blood pressure disturbances.

We conclude that adrenaline can produce acute pulmonary edema and myocardial infarction if given in overdose. Early diagnosis and prompt supportive management can save the patient’s life.

REFERENCES


A HIGH-CONCENTRATION CAPSAICIN PATCH, FOR THE TREATMENT OF POSTHERPETIC NEURALGIA (PHN)

PHN occurs in about 8-19% of patients with herpes zoster, although the likelihood of developing PHN after shingles increases with age. Although low-concentration capsaicin creams (0.025 and 0.075%) have demonstrated favorable results in clinical trials, these agents must be applied multiple times daily, cause burning pain each time they are applied during the early phase of treatment, and provide only modest pain relief. To counteract these limitations, while retaining the benefits of capsaicin therapy, NGX-4010 has been developed. NGX-4010 is a high concentration capsaicin dermal patch (capsaicin, 8%) that delivers a therapeutic dose of capsaicin during a single 60-minute application. Full version of the article is available at: