CASE REPORT

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Acute Massive Pulmonary Oedema and Myocardial Ischaemia after Adrenaline Intranasal Pack Application

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Abstract

A 35-year-old female patient presented to hospital for left endonasal dacryocystorhinostomy (endo DCR) under sedation. The patient developed acute massive pulmonary oedema and cardiac abnormality following intranasal application of 3 mg adrenaline (1:2,00,000) in 30 ml lignocaine as nasal packing. A minute after nasal packing she developed tachycardia with heart rate of 135 - 145 per minute and blood pressure of 170/120 mmHg, noted transiently, along with occasional ventricular extrasystoles recorded on monitor. Patient was started on drip of injection dexmedetomidine 16 microgram per hour and the dose was stepped up to 24 microgram per hour. Echocardiography done subsequently revealed lateral wall hypokinesia with ejection fraction (52%), troponin -T was positive. She was put on frusemide and ionotropic support besides assisted positive pressure ventilation and was discharged after 1 week with normal vitals.

Key words: Adrenaline nasal pack, dexmedetomidine, myocardial ischaemia, pulmonary oedema.

Introduction

Haemostatic agents such as vasoconstrictors are often used during nasal surgeries by otorhinolaryngologists. Currently used drugs are adrenaline, noradrenaline, oxymetazoline, vasopressin and phenylephrine for achieving nasal mucosal vasoconstriction[1]. However, the most commonly used local vasoconstrictor is adrenaline with lignocaine for prolonging the local effects. Adrenaline has both alpha and beta receptor agonist actions, hence may result in tachycardia and hypertension besides restlessness and headaches: It may be even lead to life-threatening myocardial ischaemia, ectopics and pulmonary oedema[2].

Dexmedetomidine offers the unique ability of providing sedation and analgesia without respiratory depression, which this patient received. The drug has highly selective alpha-2 adrenoreceptor agonist activity. We present the case of a 35-year-old female who was taken to operation theatre for elective dacryocystorhinostomy who developed pulmonary oedema soon after receiving adrenaline nasal pack and injection dexmedetomidine drip for sedation.

Case report

A 35-year-old, 44 kg non-diabetic and non-hypertensive female was hospitalised for elective dacryocystorhinostomy under sedation. The medical and pre-anesthetic examination was unremarkable. Her vitals were normal with SpO_2 of 98% with normal sinus cardiac rhythm. Pre-medication drugs used were glycopyrrolate 0.2 mg, midazolam 1 mg intravenous, injection pentazocine 20 mg and injection dexmedetomidine at the rate of 16 mcg/hr: the dose stepped to 20 mcg/hr and later to 24 mcg/hr. Nasal packing was done with 3 ampules of adrenaline (1:2,00,000) added with 30 ml of lignocaine in bowl (resultant concentration: 100 mcg/ml). Soon after adrenaline soaked nasal packing, the heart rate rose to 135 - 145/min and blood pressure transiently to 170/120 mmHg, SpO_2 recorded was 100%. Injection dexmedetomidine was enhanced to 24 mcg/hr within 5 minutes of nasal packing and after enhancing the drip of injection dexmedetomidine, the SpO_2 declined to 78%. Pink frothy secretions were noticed. BP decreased to 80/50 mmHg with circulatory failure and multiple ventricular ectopics were observed on monitor transiently when the patient received the above drugs. Patient was then propped up, intravenous infusions were stopped, bladder was catheterised, 80 mg furosemide and hydrocortisone 100 mg was once given. Urgent X-ray taken showed evidence of massive pulmonary oedema (Fig. 1). Noradrenaline was started by resident and soon it was changed to dobutamine to maintain systolic blood pressure. Patient was shifted to Intensive care unit from surgical theatre and kept on assisted ventilation. Patient's haemogram, liver enzymes, urea, creatinine, sugar were normal, Troponin-T was positive. Urine output varied from 75 - 100 ml /hr from initial anuric status that lasted for 6 hours. The blood gases revealed metabolic acidosis with marked hypoxia. 2D echocardiography revealed an ejection fraction of 52%, multiple ventricular extrasystoles

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with lateral wall hypokinesia. Repeat chest X-ray done after 48 hours showed recedence of pulmonary oedema (Fig. 2). Patient was under observation for one week with normal vital parameters and a normal repeat 2D echocardiogram.

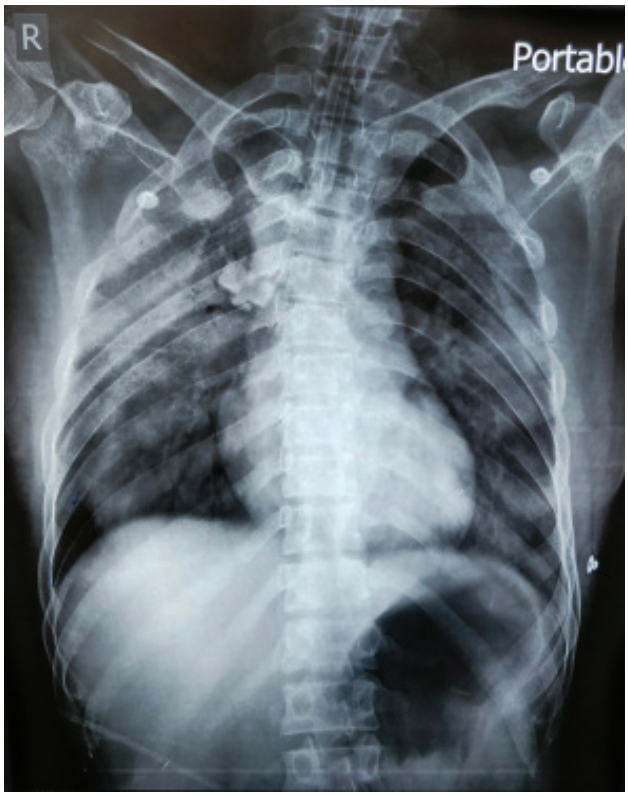

Fig. 1: *X-ray showing evidence of acute pulmonary oedema.*

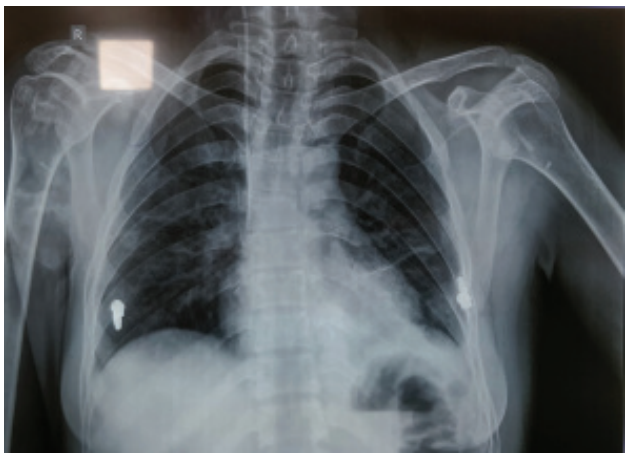

Fig. 2: *X-ray showing near complete recedence of pulmonary oedema.*

Discussion

Nasal surgeries are often associated with bleeding. For countercheck and ensuring a bloodless surgical field vasoconstrictors like adrenaline are commonly in use along with lidocaine as 2% or 4% solution. Surgeons use adrenaline 1 in 2 lakh or 1 in 4 lakh diluted in saline along with lidocaine, to achieve vasoconstriction. Adrenaline acts through alpha-1, alpha-2, beta-1 and beta-2 receptors and has a host of effects. Alpha-1 receptors mediate arteriolar vasoconstriction and have positive inotropic and chronotropic effects. The drug may also induce coronary vasoconstriction, while alpha-2 receptors mediate venoconstriction increasing systemic and pulmonary resistance while beta-1 and beta-2 receptor action causes tachycardia and increase in myocardial contractility and also vasodilates the coronary[2]. The adverse reactions include restlessness, irritability, fear, anxiety, headaches, urinary retention, hypertension, tachycardia, angina and cardiac arrhythmias. Hypertensive crisis may lead to brain haemorrhage, pulmonary oedema, myocardial infarction and ventricular fibrillations[3,4].

Use of lidocaine 2% or 4%, along with adrenaline, provides protection against induced arrhythmias and limits the dose of adrenaline absorbed. The usual recommended safe dosage of adrenaline is should not exceed 3 microgram per kg in a patient[5]. In the present case, three ampules of adrenaline, with 30 ml of 4% lidocaine was used for dilution; of this 15 - 18 ml were used for nasal package. The patient received a dose beyond 5 mcg/ml, resulting in adverse circulatory effects.

The case under discussion also received injection IV dexmedetomidine, an alpha-2 adrenergic receptor agonist. The drug was used as an adjuvant for sedation along with adrenaline (as local instillation). Dexmedetomidine offers the unique property of sedation and analgesia without respiratory depression but could possibly trigger, directly or indirectly, adverse reactions of hypertension or hypotension and acute pulmonary oedema. Dexmedetomidine may also induce coronary vasoconstriction through alpha-2 receptor action[6].

This patient developed cardiac and pulmonary complications on receiving adrenaline pack while the drip of injection dexmedetomidine was ongoing and its dose was stepped up from 16 mcg/hr to 24 mcg/hr. One needs to be careful regarding the rate of drug administration especially along with the use of local vasoconstrictors like adrenaline. Whether this combination of the drugs has any synergistic effects, needs review. This patient developed a rise in blood pressure to 180/110 mmHg and soon had fall of blood pressure to 90 mmHg systolic along with features of pulmonary oedema when nasal adrenaline pack was applied while the dexmedetomidine drip was ongoing. Troponin-T was positive in the case. This positivity may be attributed to excess alpha-1 activity of adrenaline that could have resulted in transient myocardial ischaemia, tachycardia

and subsequent pulmonary oedema[7]. A similar observation was made by Jayamali (2017) wherein the author reports a young adult who received a therapeutic dose of adrenaline for anaphylaxis developed myocardial ischaemia transiently for around half an hour who was reported to have had healthy coronaries[8]. Our patient, had initial normal electrocardiogram and acute myocardial ischaemia, based on troponin-T positivity and lateral wall hypokinesia on echocardiogram. In a large multi-hospital observational study of over 7,33,191 patients, 4.4% demonstrated normal presenting ECG. This observational study has demonstrated that the normal ECG myocardial infarction presents with secondary adverse outcomes in the hospital like ventricular tachycardia, pulmonary oedema, cardiogenic shock, and hypotension that required interventions[9]. Our patient having normal ECG, troponin-T positivity, echocardiographic features of lateral wall hypokinesia, pulmonary oedema and hypotension needed inotropic interventions. Patient's condition improved in the next 48 hours. All vitals were well maintained. She was shifted to non-ICU wing and discharged the next day. Follow-up chest X-ray, ECG and echocardiography were normal.

Conclusion

A case of massive pulmonary oedema and myocardial ischaemia in a 35-year-old female patient is described who developed this complication soon after intranasal adrenaline-lignocaine nasal packing while on a drip of dexmedetomidine. Possible synergistic effects on coronaries with adrenaline through alpha-1 receptor and dexmedetomidine through alpha-2 receptor has not been reported in literature; however, it needs focus in situations of the type.

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