



Cardiogenic Shock following Dacryocystorhinostomy- an Alarming Experience

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Abstract: This is a case report of 40 year old male patient, posted for dacryocystorhinostomy (DCR), who suffered ventricular tachycardia and pulmonary oedema, after sub mucosal intranasal application of epinephrine soaked pellets under general anaesthesia, managed successfully.

Keywords: Dacryocystorhinostomy

Dacryocystorhinostomy (DCR) is a surgical procedure done under local or general anaesthesia for relief of nasolacrimal duct obstruction¹. It is indicated when there is symptomatic obstruction of the nasolacrimal duct that is not relieved by simple probing and syringing. The infiltration of epinephrine along with a local anaesthetic has been used for many years to provide hemostasis necessary for the surgical field in ear, nose and throat surgeries².

Epinephrine has been used to reduce bleeding and prolong the duration of local anaesthetics². Though rare, there are case reports in the literature where fatal complications due to systemic absorption of locally placed or injected epinephrine has resulted in significant mortality^{3,4}. These cases need warrant of guidelines for the use of local administration of epinephrine in such surgeries for the safety of patients and to reduce mortality and morbidity associated. Till date, such guidelines are lacking.

Case Report: Forty year old male patient presented to ENT OPD with chief complaints of continuous watering from left eye since one month. There was no other clinically significant past history. On examination, his heart rate was 84/minute and regular, blood pressure was 110/80 mm Hg. His systemic examination revealed no abnormality. His routine biochemical investigations were within normal limits. His ECG and X-Ray chest were within normal limits.

He was given general anaesthesia for Dacryocystorhinostomy. Pulse oximeter, ETCO₂ and ECG monitor with defibrillator were attached. Premedication of injection glycopyrrolate 6 µg/kg



intramuscular was administered half an hour prior to surgery. In addition intravenous midazolam 0.5 mg, intravenous pentazocine 0.3 mg/kg, and intravenous ondansetron 0.8 mg/kg were administered. Intravenous induction of anaesthesia was done with injection pentothal sodium 5 mg/kg and injection succinylcholine 2 mg/kg. Oral intubation was done with 9.5 numbered portex cuffed endotracheal tube under vision. Air entry was checked and was equal bilaterally. Tube was fixed. Throat packing was done. Maintenance of anaesthesia was done with oxygen 50% and nitrous oxide 50% with vecuronium 0.08 mg/kg and propofol 4 mg/kg/hour.

After induction of anaesthesia, ENT surgeon packed both nostrils with cotton patties soaked in solution of epinephrine 1:1000 with lignocaine 2%. The dilution was 1:20000. Immediately after nasal packing, patient's heart rate increased to 180/minute. ECG showed ventricular tachycardia. Patient developed hypotension with blood pressure of 70/50 mm Hg. His oxygen saturation dropped to 85%. Pink frothy sputum was seen through endotracheal tube. In view of these findings, the procedure was stopped and epinephrine soaked patties were removed. Ventricular tachycardia was treated with intravenous 2% lignocaine in the dose of 1 mg/kg. The ventricular arrhythmia reverted back to sinus rhythm. Simultaneously dopamine 5 µg/kg/min, Ringer's lactate and 100% oxygen were started. He was started on mechanical ventilation. He was subsequently shifted to surgical ICU and electively ventilated with assist control mode with FiO₂ 100%. Dopamine infusion was continued at rate of 8 µg/kg/min. Central line was inserted and central venous pressure was maintained between 10-15 cm of water. His chest X-Ray showed ground glass appearance of pulmonary oedema. His ECG showed T inversion from V3 to V6. Echocardiography showed global hypokinesia and ejection fraction 30%.

He was electively ventilated for next 48 hours. His condition gradually stabilized. He was successfully weaned off ventilator and inotropes on third postoperative day. Chest X-ray also showed improvement in pulmonary oedema. ECG changed reverted to normal and echocardiographic changes improved with ejection fraction 55 %. His urinary VMA levels were within normal limits. USG abdomen was normal. He was discharged on fifth postoperative day.

Discussion: Majority of ENT surgeries can be done under local anaesthesia with lignocaine and epinephrine¹. Lignocaine with epinephrine is used as nasal mucosal decongestion for achieving better field and operating conditions². Severe cardiovascular complications and death after elective ENT surgery is extremely rare and unexpected³. The cardiovascular effects of epinephrine are well known which include vasoconstriction, increased heart rate, arrhythmias and pulmonary oedema^{4,5}. Stimulation of sensitive β₁ adrenergic receptors results in tachycardia, cardiac arrhythmias, increased cardiac output and myocardial oxygen consumption³.

Pulmonary oedema is a well described complication of systemic epinephrine^{3,4,5}. It is caused by:

1. Increased pulmonary circulation pressure.
2. Tachycardia and decreased diastolic filling time.



3. Increased left atrial pressure with pulmonary vasoconstriction, causing a rise in pulmonary arterial pressure and disturbances in the oncotic and hydrostatic pressure relationships in the pulmonary capillary bed
4. Decrease in the left ventricular compliance. The subsequent rise in left ventricular end diastolic pressure causes increased pulmonary capillary pressure manifesting as acute pulmonary oedema.

Pulmonary oedema has three phases; acute, hypotensive and recovery phase^{4,5}. Our patient suffered ventricular tachycardia after systemic absorption of topical epinephrine followed by pulmonary oedema. The strength of epinephrine soaked patties was 1:20000 in our case. There is a case report in the literature⁴ where ENT surgeon has used strength of 1:1000 epinephrine soaked cotton patties. This patient suffered cardiogenic shock.

In our case, the rhythm returned back to sinus rhythm after administration of 1 mg/kg of 2% lignocaine. Cardiac arrest did not occur, but later on, there was copious amount of pink frothy sputum through endotracheal tube with hypotension. The SPO₂ dropped to 85% with 100% oxygen. Diagnosis of pulmonary oedema was made and treatment was started with injection furosemide 40 mg, inotropic support with dopamine 5 µg/kg/min and intermittent positive pressure ventilation. In the Canadian report³, post-extubation hypotension and ventricular bigemini occurred followed by cardiac arrest requiring resuscitation.

The patient was transferred to surgical intensive care unit and later with his relative's wish he was transferred to a nearby private hospital with well equipped coronary care unit (CCU). His ECG, CVP and echocardiographic findings were suggestive of cardiogenic shock. He was electively ventilated for two days with inotropic support of dopamine and norepinephrine. He responded to this treatment and was weaned off ventilator on third day and later was shifted to ward on fourth day with improved ECG, echocardiographic and X-ray findings. Schwalm et al³ reported a case with profound hemodynamic compromise requiring percutaneous cardiopulmonary support (CPS) where the patient was weaned off CPS after five days and ventilator was discontinued after five weeks.

There are two case reports⁴ where a patient posted for modified radical mastoidectomy developed ventricular tachycardia and fibrillation with hypotension and cardio-respiratory arrest, managed successfully as in our case. The second case that underwent modified radical mastoidectomy also developed ventricular tachycardia and pulmonary oedema. In both these cases the concentration of epinephrine infiltrated was 1:100000. There is a case in Woldorf and Pastore's original report bearing striking resemblance to our case^{4,5}.

Though epinephrine has been used in ENT surgery since many years, there are no recommended standard guidelines in ENT or anaesthesia literature for these surgeries. Halothane has been documented to produce arrhythmias^{6,7} more than other inhalational agents with local infiltration of epinephrine. In all the cases mentioned in literature^{3,4,5}, isoflurane has been used for maintenance of anaesthesia. In our case, the arrhythmia occurred immediately after packing the nostrils with epinephrine soaked pellets where a propofol infusion was started for maintenance of anaesthesia.



Epinephrine hypersensitivity induced cardiovascular crisis has been reported in the literature⁵. In this case, proposition was that exogenously administered epinephrine might have triggered release of endogenous catecholamines which was confirmed by Carter et al⁵ who measured the total excreted epinephrine and metabolites in a patient who received 5 mg of epinephrine subcutaneously and found out that the patient excreted more than twice as much as would have been expected. Rare patients may demonstrate an extreme sensitivity to subcutaneously injected epinephrine in doses within the recommended guidelines for administration⁵. This sensitivity seems to be mediated by massive release of endogenous catecholamines and produces hypertension, arrhythmias and pulmonary oedema normally seen only with overdoses of epinephrine administered intravenously.

We measured urinary VMA levels of this patient which were normal, ruling out hypersensitivity as one of the causes of this complication. Another cause of cardiogenic shock is secondary to acute ischemia which is unlikely as the patient had no significant ECG changes suggestive of ischemia. He had no cardiac risk factors. The third possibility is Addisonian crisis⁸ with overt cardiogenic shock and left ventricular dysfunction. However our patient had no persistent electrolyte abnormalities and he recovered completely without need for stress dose steroids. Adrenaline secreting pheochromocytoma⁹ was unlikely in our case as his USG abdomen was normal and urinary catecholamine levels were normal following discontinuation of all inotropes. Viral myocarditis¹⁰ was ruled out as patient had no viral prodrome and other associated precipitating factors. Unusual cause of acute pulmonary oedema is cocaine use¹¹. Our patient had no history of substance abuse.

Many authors have recommendations for safe dosage of epinephrine. Milam and Giovannitti suggested that doses of epinephrine should not exceed 3 µg/kg for healthy patients³. Johnson et al⁷ reported an injection of 1 µg/kg with halothane and 3.4 µg/kg with isoflurane. These doses could be increased by 50% when concomitant 0.5 % lignocaine is administered⁷.

As there are no standard international guidelines, we believe that there are important clinical implications of this case which we witnessed. Hence we recommend following measures to be taken to prevent further occurrence of similar cases.

1. As nose is a highly vascular structure, epinephrine soaked patties may be avoided and instead only 4% lignocaine alone can be used as topical anaesthesia.
2. Minimum dose of epinephrine for topical pellets for any nose case should be 1: 50000.
3. Patties should be squeezed off the local anaesthetic and epinephrine solution and exposed to air to have drying and evaporation¹² with special instruction to paramedical staff preparing the trolley for such cases.
4. The dose of topical epinephrine should be administered in a calibrated syringe and should be verified by anaesthesiologist.
5. ENT surgeons should be asked to always aspirate before injection.
6. Epinephrine concentration for nasal septum infiltration should be 1:100000 onwards.
7. Small bore needles should be used and speed of injection should be slow.



8. Beta blockers or calcium channel blockers should be avoided as a treatment of hypertension as it may worsen the failing heart¹³.
9. Needle size and drop rate when epinephrine is used from the standard epinephrine ampoule should be following for the concentration for pannies 1:80,000 when diluted with 10 ml saline
 - a. 20 G needle 10 ½ drops
 - b. 21 G needle 12 ½ drops
 - c. 22 G needle 14 drops
 - d. 23 G needle 13 drops
 - e. 24 G needle 12 drops
10. Each hospital should form an advisory committee¹³ for epinephrine and local vasoconstrictor dose use that can monitor careful dosing of these drugs, the resulting rhythm and blood pressure disturbances
11. The dose for paediatric population should be adjusted as per weight and age.
12. The importance of these precautions should be discussed with operating ENT surgeon and paramedical staff.

We hope that these recommendations will result in more cautious use of epinephrine in ENT surgeries and alert the anaesthesia community to the potentially lethal complications of topical epinephrine.

References:

1. Neil Fergie, Nicholas s. Jones Dacryocystorhinostomy Scott and Brown's Otorhinolaryngology Seventh edition (3), 133: 1689-1697
2. Riegler EV, Gunter JB, Lusk RP Comparison of vasoconstrictors for functional endoscopic sinus surgery in children Laryngoscope 1992;102: 820-823
3. Schwalm Jon David, Joel Hamstra, Mulji Amin. Cardiogenic shock following nasal septoplasty: a case report and review of the literature. Canadian Journal of anaesthesia 55;(6) 2008:376-379
4. Wanamaker H.H., Arandia H.Y. Epinephrine hypersensitivity induced cardiovascular crisis in otologic surgery Otolaryngology – Head and neck surgery , 1994:841-845
5. Woldorf NM, Pastere PN. Extreme epinephrine sensitivity with general anaesthesia Arch Otolaryngology (96); 1972: 272-277
6. Stoelting Robert K., Hillier Simon C Inhaled anaesthetics Pharmacology and physiology In anaesthetic practice 4th edition: 54 -56
7. Johnston, Leger E, Wilson C A comparison interaction of epinephrine with enflurane, isoflurane and halothane in man Anesth Analg 1976;55: 709-712
8. Wolf B, Machill K. Acute reversible cardiomyopathy with cardiogenic shock in a patient with Addisonian crisis, a case report Internet Journal of Cardiology 2007; 116:71-73
9. Slen SW, Deal LE, Piesman M Epinephrine secreting pheochromocytoma presenting with cardiogenic shock and profound hypocalcemia Ann Intern Med 2004;140:849-851
10. McCarthy Re 3rd, Boehmer JP. Long term outcome of fulminant myocarditis as compared with acute (non fulminant) myocarditis N Eng J Med 2000;342: 690-695



11. Ashi M, Wiedemann H.P.,Karen B.James. Cardiac complication from use of cocaine and phenylephrine in nasal septoplasty Arch Otolaryngol Head neck surg (121); 1995: 681-684
12. Stamberger Heinz Preoperative preparation, sinus problems and endoscopic solutions Functional endoscopic sinus surgery Merklinger technique fourth edition; 328-330,336
13. Groudine SB, Hollinger I,Jones J. New York state guidelines on the topical use of phenylephrine in the operating room. Anaesthesiology (92),(3); 2000:100-103