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Case Report

Acute Pulmonary Edema Due To Inadvertent Overdosage of Adrenaline in a Pediatric Patient Undergoing Tympanoplasty

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ABSTRACT

Adrenaline is the preferred vasoconstrictor for infiltration in ear, nose and throat surgeries, with or without local anaesthetic agent, for its useful properties as a hemostatic agent, constricting capillaries and providing better visualisation for surgery. But not giving attention to dose and dilution of adrenaline, particularly in pediatric patients can lead to undesirable side effects, as happened in this case.

Key words: Adrenaline, Pulmonary edema, Pediatric patient.

INTRODUCTION

Local infiltration of adrenaline before surgery is a common practice during Ear, Nose and Throat (ENT) procedures. Not paying close attention to the dosage of adrenaline by weight of patient can lead to undesirable side effects, one of them being pulmonary edema, as happened with our case.

CASE REPORT

A 9 year old girl, weighing 16 kgs, belonging to American Society of Anaesthesiologists (ASA) physical status 2, was posted for tympanoplasty under general anaesthesia. Patient's history and clinical findings were non significant. Pre operative blood pressure (BP) was 100/70 mm of hg and pulse rate of 102 bpm. Laboratory investigations were within normal limits. Inj. ranitidine 20 mg and inj. ondansetron 2 mg were given intravenously in the preinduction room.

Inside the operation theatre, routine monitors like electrocardiogram, non-invasive blood pressure cuff and oxygen saturation probe were connected. Inj. Glycopyrrolate 0.1mg, inj. Midazolam 1 mg and inj. Fentanyl 50 mcg were given intravenously (i.v). After preoxygenation with 100% oxygen for three minutes, patient was induced with inj. propofol 50 mg iv and intubated under inj. Succinylcholine 30mg i.v. Anesthesia was maintained with nitrous oxide : oxygen in ratio 70:30 and non depolarising muscle relaxant inj. Vecuronium 2mg. Post intubation BP was 107/62, heart rate was 104 bpm and oxygen saturation was 99%. Once the child was positioned for surgery, surgeon infiltrated the field with 0.5 ml of 1:1000 of adrenaline diluted to 5ml (1:5000) and we were not

aware of the dose and dilution of adrenaline he was using. Assisting nurse also used swabs soaked in adrenaline diluted in normal saline (1:200000). Within few minutes of starting surgery, patient developed tachycardia of 160 bpm and blood pressure was 180/110 mm of hg. Our initial diagnosis was 'inadequate depth 'of anaesthesia, so we gave a bolus of 10mg of propofol and started propofol infusion at the rate of 2.5mg/min and inj vecuronium 0.5 mg i.v was repeated. But tachycardia and hypertension persisted and surgeon was asked to stop the surgery. After few minutes saturation started dropping, pink frothy secretions were seen in the endotracheal tube and chest auscaltation revealed bilateral coarse crepitations. Immediately nitrous oxide was cut off and 100% oxygen was given. Inj frusemide 20 mg, Inj morphine 3mg i.v, inj hydrocortisone 40 mg iv was given. Patient was propped up and IPPV (Intermittent Positive Pressure Ventilation) continued. At the interval of 5 minutes, 10 mg of frusemide was repeated twice. Gradually oxygen saturation improved from 84-85% to 91-92% with 100% oxygen. Surgeon was asked to continue surgery and procedure was completed in 30 minutes. At the end of surgery, BP was 160/100 mm of hg and heart rate 180-200 bpm. Child was reversed with inj neostigmine 1mg and glycopyrollate 0.1 mg. As child was sedated, respiratory efforts were inadequate and crepitations , even though decreased than earlier, persisted, she was shifted to Intensive Care Unit and connected to ventilator on SIMV (Synchronised Intermittent Mandatory Ventilation) with fiO2 of 1 and PEEP of 5. As bed-side echocardiography is not available in our institute, echocardiogram could not be done immediately. After about seven hours after surgery, patient was extubated when patient was conscious, breathing spontaneously and maintaining saturation of 97-98% with fiO2 of 0.4. She was put on simple face mask with fiO2 of 0.4. Echocardiogram done after extubation showed normal findings thus ruling out presence of any congenital cardiac disease and all features developed so far pointing towards inadvertent overdosage of adrenaline infiltration. Next day her vital signs were stable and maintaining oxygen saturation of 99% on room air. She was allowed to take orally and shifted to ENT ward and discharged from hospital after three days.

DISCUSSION

For most ENT surgeries, pre-surgical local infiltration with adrenaline is a time tested and widely used practice. Adrenaline, with or without local anesthetic, is infiltrated for its useful properties as a hemostatic agent, constricting capillaries and providing a better visualisation of surgical field. [1] When used with local anaesthetic agent, adrenaline delays its absorption and hence toxicity of agent.

Adrenaline acts on both alpha and beta adrenergic receptors of tissues innervated by sympathetic nerves and may cause adverse reactions like hypertension, tachycardia and cardiac arrythmias in patients under general anaesthesia when safer doses are exceeded. [2] It can also lead to fatal complications like pulmonary edema, cerebral haemorrhage and myocardial infarction. [1] Although main purpose of vasopressor infiltration is to provide hemostasis and reduce complications, systemic absorption of the vasoconstrictor itself can lead to undesirable toxic effects as happened in our case.

In this patient, surgeon infiltrated 0.5 ml of 1:1000 adrenaline (500mcg) which amounts to 10 times the recommended dose of adrenaline (3mcg/kg i.e 50 mcg). The normal recommended dilution for infiltration is 5mcg/ml of 1:200000 dilution. [2,3]

Our initial diagnosis of severe hypertension and tachycardia was that of inadequate depth, which after adequate measures did not settle the problem. So we asked the nurse to stop using adrenaline swabs as tachycardia and hypertension were persisting. Subsequent development of pulmonary edema led us to think of probable undiagnosed congenital heart disease, but history and preoperative examination findings were not supportive.

Pulmonary edema is known to be caused by excess adrenaline. Increased systemic vascular resistance and tachycardia cause excess load on left ventricle which leads to pulmonary congestion. [4] There is a mismatch in increased pulmonary artery pressure compared with pulmonary alveolar pressure, causing hydrostatic flux of fluid. It is found in studies that excess adrenaline causes changes in the endothelial and both types of clara cells leading to toxic lung injury causing pulmonary edema. [4]

There are case reports in literature which describe development of arrythmias, acute massive pulmonary edema. [5] myocardial infarction with cardiogenic shock. [6,7] and even cardiac arrest. [8] due to either adrenaline overdosage or inadvertent intravascular injection. While most patients have recovered early due to aggressive treatment, some patients with severe cardiogenic shock have taken days to weeks for total recovery. [6] Treatment of systemic side effects with beta adrenergic blocker may be followed by irreversible cardiovascular collapse, due to decreased ability of stressed myocardium to increase contractility and heart rate.

CONCLUSION

In conclusion, role of anesthesiologists is very important and surgeons have to discuss with anaesthesiologists regarding the dosing and dilution of adrenaline, particularly in pediatric patients as overdosage can lead to undesirable side effects and morbidity.

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