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Intranasal phenylephrine in the operating room - how many drops?

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Abstract

Background: Topical vasoconstrictors are commonly used to minimize bleeding during Ear, Nose and Throat surgeries. Phenylephrine is one of the most commonly used and could be an underrecognized source of intra-operative events. Total dose of administered drugs is often unmeasured.

Cases of acute pulmonary edema, cardiac arrest and death after topical vasoconstrictors (TV) have been reported. Maintenance of cardiac output is of paramount importance to avoid cardiovascular collapse. β1 receptor blockage reduces cardiac output, it is contraindicated in these situations.

Case: This case describes an intra-operative critical event after an unknown amount of phenylephrine administration.

Conclusion: Lack of awareness about phenylephrine maximum recommended dose may induce complications leading to haemodynamic instability. The authors aim to raise awareness about the use of TV, including dose, administration site and also clinical signs and symptoms that may appear after its use. Special attention must be addressed to the drug choice for the support treatment of eventual cardiovascular symptoms.

List of abbreviations

Topical Vasoconstrictors (TV)

Oxygen Saturation (SpO2)

Heart Rate (HR)

Blood Pressure (BP)

Background

Topical vasoconstrictors (TV) are commonly used by nose and throat surgeons to minimize bleeding and to enhance surgical field view. Nasal packing with TV is also applied at the end of certain surgical procedures to prevent postoperative complications such as bleeding, septal hematoma, and adhesion formation. These drugs are used in a variety of concentrations and the exact total dose administered is, in most cases, unmeasured nor documented[1,2].

Phenylephrine is one of the most commonly used TV. It is a non-catecholamine, selective alpha-1 adrenoceptor agonist, with very little beta effect, that has powerful vasoconstrictive properties[3]. However, its absorption in the surgical field is unknown and it could be a source of intra-operative complications.

Hypertensive crisis is the most common complication caused by phenylephrine topical use, but there are reports describing pulmonary edema, cardiovascular collapse and even death after its use during surgery[4].

We report a case of a patient where lack of awareness about phenylephrine maximum recommended dose may have induced TV possible complications leading to an unnecessary overnight stay after a programmed ambulatory turbinectomy.

Case presentation

A 13 years-old-boy (weight 40 Kg, height 147 cm) underwent an elective ambulatory bilateral turbinectomy.

No history of systemic diseases was present nor any report of being on daily medication; an American Society of Anesthesiologists classification (ASA) I was performed. His preoperative physical examination and vital signs were unremarkable.

ASA standard monitoring was used intraoperatively. Bispectral index monitoring and quantitative kinemyographic neuromuscular monitoring (NMT-Mechanosensor) on the right forearm, were also applied.

Anesthesia was induced with fentanyl (100mcg i.v.), lidocaine (40mg i.v.) and propofol (100mg i.v.). After calibrating the neuromuscular monitor, rocuronium (25mg i.v.) was given. Uneventful tracheal intubation was performed with a normal 6.0 tube. Anesthesia was maintained with sevoflurane at 1.0 MAC and bispectral index values between 40 and 60. Ventilation was controlled to maintain end-tidal normocapnia (30–35 mmHg). Surgery lasted for 30 minutes without complications.

At the end of the procedure, both nostrils were packed with cotton balls soaked in an unmeasured amount of phenylephrine 2,5% and normal saline. Surgical fields covers were removed and neuromuscular block was reversed with sugammadex (80mg i.v.).

When preparing for anesthetic emergence, about five minutes after cotton balls placing in the nostrils, heart rate decreased from 85 to 44 beats/min with an arterial blood pressure (BP) of 119/68 mmHg (mean 88 mmHg) Oxygen saturation (SpO2), end-tidal carbon dioxide and temperature were recorded within the normal range. Atropine (0.5mg i.v.) was given and HR increased to 138 bpm and BP to 220/150 mmHg. At this time, anesthetic depth was increased with sevoflurane and vagal maneuvers were started. Labetalol (2.5 mg) was administered in order to control the hypertension peak but the patient remained hypertensive and the supraventricular tachycardia previously observed was still present.

At this point, poor peripheral perfusion was noticed and the anesthesiologist removed nasal packing considering that it could be the leading cause for this clinical situation.

Almost immediately after nasal packing removal, patient HR and BP returned to his initial values. Bronchospasm and crackles were not heard in the lung auscultation and no other signs of pulmonary edema were noticed. After acknowledging safe anesthetic emergence conditions, extubation was possible without any remark. The patient remained in a post-anaesthesia care unit under surveillance for 24 hours and was discharged without reported complications.

Discussion and Conclusions

Nasal package with phenylephrine caused extreme hypertension and bradycardia in our patient, which only reverted when removed. Actually, phenylephrine's main action is systemic and pulmonary arterial vasoconstriction, leading to increased systemic vascular resistances and systemic arterial pressure (systolic, diastolic, and mean). Ultimately this may cause activation of baroreceptor reflex and result in bradycardia[4].

Initially, identification of phenylephrine as a potential cause of haemodynamic changes were not made, so atropine and labetalol were given. Actually, there are some reports where hypertensive crises from topical phenylephrine were managed with beta-blockers, probably because of lack of awareness for the hypertension cause, as in this case[1].

Phenylephrine's effects are responsible for a blood shift to the pulmonary circulation, where those effects are less felt. The increase in the afterload also causes an increased impedance of left ventricular ejection and end-diastolic volumes and pressures[4]. There are some compensatory mechanisms to preserve cardiac output including the ability to increase heart rate and contractility. So, in this situation the use of b-blockers is not recommended as they impair these mechanisms by decreasing heart rate and further compromising left-sided output (b1-block receptors)[1]. The use of b-blockers to treat hypertension caused by topical phenylephrine use may cause pulmonary edema and even death, as reported by a commission formed by anesthesiologists, intensivists, otorhinolaryngologists and pharmacologists that established recommendations for phenylephrine's use in the operating room (New York Guidelines on the Topical Use of Phenylephrine in the Operation Room)[2].

The incidence of this problem is not negligible. Kalyanaraman et al. described twelve cases of cardiopulmonary compromise after TV, most of them topical phenylephrine. In seven of them b-blockers were administered. Of these seven patients, three suffer cardiac arrest[1].

Reviewing literature there is a recurring pattern in all cases: significant hypertension with or without baroreceptor-induced bradycardia, after TV application in the surgical field. Because it avoids the first pass effect from either the gut wall or liver, the intranasal route offers higher concentrations than oral formulations[3]. The systemic effects are noted within minutes and can lead

to more dangerous complications: cardiac dysrhythmias, cardiac arrest, and subarachnoid hemorrhage[1].

There are no official guidelines defining the maximum dose of TV, but the NYS Department of Health recommended the initial dose of phenylephrine should not exceed 0.5 mg in adults (4 drops of a 0.25% solution) or 20.0 μg/kg in children (up to 25 kg). Despite these doses of TV, if hypertension develops, appropriate treatment includes direct vasodilators or α-antagonists[2].

Higgins et al. published in 2011 a systematic review overviewing the available literature of local vasoconstrictor used in the nose, nasopharynx, or sinuses. The author concluded it is difficult to recommend a safe amount of topical vasoconstrictors to use because the absorption rate is difficult to measure. However, the author proposes that, based on the reviewed literature, topical phenylephrine should be avoid as much as possible, especially in patients with history of cardiovascular disease; moreover, beta-blockers should not be used for intraoperative hypertension after topical vasoconstrictor use[5].

In this case report, the patient had been submitted to a bilateral turbinectomy, exposing blood vessels and the eroded endothelium. This may have had an important role, increasing the phenylephrine absorption. The initial choice of drugs was due to a support treatment approach, being the identification of the true cause and the removal of cotton soaked in phenylephrine, essential to reverse the clinical situation.

With this report, the authors aim to raise awareness about the use of TV, including dose, administration site and also clinical signs and symptoms that may appear after its use. In addition, special attention must be addressed to the drug choice for the support treatment of eventual cardiovascular symptoms.

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