CASE REPORT

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Sudden pulmonary edema induced by phenylephrine misuse: a case report

Iznenadni edem pluća indukovan nepravilnom primenom fenilefrina

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Abstract

Introduction. Phenylephrine, a widely used vasoactive drug in clinical practice, may lead to severe cardiovascular complications when misused. Among these complications, sudden pulmonary edema, though rare, warrants the attention of clinicians. The given case report presents a sudden pulmonary edema caused by the misuse of phenylephrine. **Case report.** A female patient aged 68-years undergoing radical mastectomy for left breast cancer developed severe hypertension and hypoxemia 40 min into the procedure. The patient's medical history included meningioma but no other significant comorbidities. A diagnosis of sudden (acute) pulmonary edema was made. The patient received prompt treatment, including strict perioperative blood pressure control, lung protective ventilation, glucocorticoids, diuretics, coronary-dilation and cardiotonic drugs, postoperative oxygen therapy, and continuous vital sign monitoring. Investigation revealed that phenylephrine had been mistakenly administered intravenously instead of dexamethasone. She recovered and was discharged one week postoperatively. **Conclusion.** This case highlights the risks associated with the inadvertent administration of a high dose of phenylephrine, leading to sudden pulmonary edema. It underscores the importance of vigilance among anesthesiologists, prompt management of complications, and strategies to prevent errors, including enhanced education for resident anesthesiologists, measures to address practitioner fatigue, and improved drug packaging to minimize look-alike errors.

Keywords:
anesthesia, intravenous; anesthesiologists; intensive care units; medical errors; phenylephrine; pulmonary edema.

Apstrakt

Uvod. Fenilefrin, vazoaktivni lek koji se često koristi u kliničkoj praksi, može dovesti do teških kardiovaskularnih komplikacija u slučaju nepravilne primene. Među ovim komplikacijama, iznenadni edem pluća, iako redak, zaslužuje pažnju kliničara. Prikazan je slučaj iznenadnog edema pluća izazvanog pogrešnom upotrebom fenilefrina. **Prikaz bolesnika.** Bolesnica starosti 68 godina, koja je bila podvrgnuta radikalnoj mastektomiji zbog karcinoma leve dojke, razvila je tešku hipertenziju i hipoksemiju 40 minuta nakon početka operacije. U anamnezi je naveden meningeom ali ne i drugi značajni komorbiditeti. Postavljena je dijagnoza iznenadnog (akutnog) edema pluća. Bolesnici je odmah ordinirana terapija, uključujući strogu kontrolu krvnog pritiska perioperativno, protektivnu plućnu ventilaciju, glukokortikoide, diuretike, koronarne dilatatore i kardiotonike, terapiju kiseonikom postoperativno, uz kontinuirano praćenje vitalnih znakova. Istragom je utvrđeno da je umesto deksametazona greškom primenjen fenilefrin intravenski. Bolesnica se oporavila i otpuštena je nedelju dana posle operacije. **Zaključak.** Prikazani slučaj ističe rizike povezane sa nenamernom primenom visoke doze fenilefrina, što dovodi do iznenadnog edema pluća. Time je naglašen značaj postojanja opreznosti među anesteziolozima, brzog rešavanja komplikacija i strategija za sprečavanje grešaka, uključujući bolju edukaciju specijalizanata iz anesteziologije, mera za rešavanje zamora lekara praktičara i poboljšanja pakovanja lekova, u cilju što manjih grešaka zbog njihove sličnosti.

Ključne reči:
anestezija, intravenska; anesteziolozi; intenzivna nega, odeljenja; medicinske greške; fenilefrin; pluća, edem.

Introduction

Phenylephrine is a commonly used vasoactive drug in clinical practice, primarily for hypotension management during the perioperative period and in the intensive care unit. Due to its vasoconstrictive effect, phenylephrine is administered intravenously (i.v.), either as an infusion or in small incremental boluses, to increase systemic vascular

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resistance and maintain blood pressure (BP). It is also commonly used in delicate surgeries with demanding surgical field visualization [1] (e.g., otolaryngological endoscopic procedures, ophthalmic mydriasis) as well as for the treatment of priapism [2]. Improper administration of phenylephrine can result in serious complications, including hypertension, arrhythmias, myocardial infarction, left ventricular failure, and cardiac arrest [3, 4]. Among these, pulmonary edema (PE) is relatively rare.

Previous reports in the literature have primarily described cases of sudden PE caused by improper local administration of phenylephrine, typically with rapid recovery [4, 5]. This report presents a case of severe, sudden PE induced by inadvertent i.v. administration of a high dose of phenylephrine.

The patient data were handled according to the Declaration of Helsinki. Written informed consent was obtained from the patient.

Case report

The patient was a 68-year-old woman (height: 156 cm, weight: 64 kg) scheduled for a radical mastectomy of the left breast due to axillary lymphadenopathy that had been present for 2 weeks. The patient's medical history included meningioma, for which regular observation had been recommended by her neurosurgeon, and a prior cholecystectomy.

Preoperative physical examination and laboratory investigations revealed no abnormalities. Cardiac ultrasound indicated left ventricular diastolic dysfunction, mild regurgitation of the aortic, mitral, and tricuspid valves, and an ejection fraction of 64%. Liver ultrasound revealed fatty liver and liver cysts. Bilateral lower extremity vascular ultrasound showed no thrombus or other abnormalities. Pulmonary computed tomography revealed scattered small nodules and pulmonary fibrosis in both lungs. The electrocardiogram showed sinus rhythm with premature atrial contractions.

Upon entering the operating room, the electrocardiogram, oxygen saturation (SpO_2), and non-invasive BP of the lower extremities were continuously monitored. The monitoring device used was Datex-Ohmeda S/5 CAM. General anesthesia was induced with i.v. injections of etomidate (12 mg), fentanyl (0.3 mg), and rocuronium bromide (40 mg). After the endotracheal intubation, anesthesia was maintained with propofol (4 mg/kg/hr), sevoflurane (0.6 minimum alveolar concentration), and remifentanil (0.2 μg/kg/min). The fraction of inspired oxygen (FiO_2) was maintained at 60%. The tidal volume was set to 400 mL, the respiratory rate was set to 12 breaths *per* minute, and positive end-expiratory pressure was not applied.

Thirty minutes after the start of the surgery, dexamethasone (5 mg) was routinely administered *via* i.v. injection to prevent postoperative nausea and vomiting. Ten minutes after the injection, the patient's BP suddenly increased to 257/134 mmHg, with a heart rate (HR) of 85 beats *per* min (bpm) and SpO_2 of 99%. It was confirmed that BP cuff on the lower limb and i.v. access were functioning normally. Repeated measurements recorded BP of 245/136 mmHg, HR of 84 bpm, and SpO_2 of 98%. When we were about to administer urapidil for BP reduction, the patient's BP (165/82 mmHg) had already started to decrease spontaneously. Given that the cause of BP abnormality remained unclear, we suspended the antihypertensive treatment.

Meanwhile, SpO_2 rapidly dropped to 90%, airway pressure rose to 24 cm H_2O, and crackles were audible bilaterally on lung auscultation. FiO_2 was immediately increased to 100%, with simultaneous manual ventilation. SpO_2 improved to 98% after a few minutes. At the same time, the surgeon observed a strong contraction of the arrector pili muscle in the patient's breast, raising suspicions of a drug-related reaction. Upon inspection of the empty medication bottles, it was discovered that phenylephrine (10 mg, 1 mL) had been mistakenly administered i.v. instead of dexamethasone (5 mg, 1 mL).

To further assess the patient's condition, puncture and catheterization of the right radial artery were performed for invasive arterial pressure monitoring and blood gas analysis. The first recorded invasive arterial BP at that time was 105/72 mmHg. The blood gas results revealed a partial pressure of oxygen (PaO_2) of 82 mmHg, partial pressure of carbon dioxide (PCO_2) of 38.4 mmHg, and PaO_2/FiO_2 < 100. Two minutes later, BP began to drop and was stabilized with epinephrine (3 μg/min). Approximately 50 min later, the patient's hemodynamics stabilized. During this period, urinary catheterization was performed, and methylprednisolone (40 mg), furosemide (10 mg), and inhaled albuterol (100 mg) were administered to improve pulmonary oxygenation.

At the conclusion of the operation, the FiO_2 was reduced to 60%, and the SpO_2 was 100%. Repeat blood gas analysis revealed PaO_2 of 171 mmHg and a PCO_2 of 43.9 mmHg. In the preparation for extubation, we gradually discontinued the anesthetic agents and simultaneously tapered the dose of epinephrine until complete cessation. Simultaneously, bloody secretions were observed in the endotracheal tube, raising suspicion of acute PE. We aborted the extubation procedure. Morphine (5 mg) was administered, and the patient was transferred to the post-anesthesia care unit (PACU) for further observation. Mechanical ventilation was continued in the PACU under sedation, with dobutamine administered to maintain stable circulatory dynamics. The inhaled oxygen concentration was adjusted to 40%. Blood gas analysis conducted 2 hrs later revealed a PaO_2 of 113 mmHg, a PCO_2 of 39.3 mmHg, and no significant change in PaO_2/FiO_2. Bedside echocardiography showed preserved systolic function, with an ejection fraction value of 60%. No further bloody secretions were observed during airway suction, and sedation was discontinued.

Vasoactive drugs were stopped, and the endotracheal tube was removed after the patient regained consciousness and hemodynamic stability. The patient was positioned with the head elevated and observed for 30 min, during which vital signs remained stable. The patient was then transferred to the ward with tabplemental oxygen at a flow rate of 3 L/min.

On the first postoperative day, the patient underwent a series of diagnostic evaluations. High-resolution pulmonary computed tomography revealed exudative lesions in both lungs and bilateral pleural thickening. Laboratory results showed aspartate aminotransferase of 46 U/L [reference range (RR): < 35 U/L], high-sensitivity cardiac troponin I of 0.853 ng/mL (RR: < 0.016 ng/mL), and N-terminal pro B-type natriuretic peptide (NT-ProBNP) of 2,127 pg/mL. Postoperatively, the patient complained of chest tightness and discomfort and was treated with oxygen inhalation at 2 L/min. On the third day postoperatively, re-examination of the laboratory tests showed normal results. One week after the operation, chest tightness was relieved, and the patient was safely discharged from the hospital.

Discussion

This case highlights the occurrence of sudden PE induced by phenylephrine misuse. Phenylephrine is a selective α1 receptor agonist commonly used to increase vascular tone and elevate BP. However, at very high doses, phenylephrine can also elicit β-adrenergic receptor activation, potentially leading to tachycardia [4]. Based on clinical experience, misuse of phenylephrine tends to induce significant bradycardia in patients and may even lead to cardiac arrest in severe cases. However, HR of the patient in this case did not decrease but remained at 85 bpm, and this special clinical manifestation also verified the validity of the conclusions from the aforementioned literature.

Previous reports have documented hypertensive crisis or acute PE following topical instillation [5] or local injection [6] of phenylephrine during pediatric ophthalmic surgeries, as well as in *puerperae* with accidental 2 mg administration [7], all of whom achieved rapid recovery. In contrast, the 10 mg overdose in our case caused more severe myocardial injury and PE, with persistent chest tightness requiring daily oxygen therapy for one week postoperatively, highlighting critical implications for clinical medication safety.

Misuse of phenylephrine caused a sudden increase in peripheral vascular resistance, which induced left ventricular end-diastolic dysfunction and a sharp elevation of left atrial pressure, ultimately leading to acute PE [8]. The study confirmed that rapid BP reduction can alleviate capillary injury, which also explains the rapid resolution of bloody tracheal secretions, indicating that timely identification and standardized management are crucial for avoiding adverse events [9]. The relatively severe condition of the patient in this case was considered to be associated with pre-existing left ventricular diastolic dysfunction, which lowered the threshold for PE [10].

Additionally, the patient might have developed transient left heart failure. This speculation was supported by perioperative hypotension and postoperative elevations of myocardial enzymes, troponin, and NT-proBNP. According to the literature [11], in patients aged 50–75 years, an NT-proBNP level > 900 pg/mL yields a sensitivity > 90% and specificity > 84% for the diagnosis of acute heart failure, which further verifies this inference.

In response to the phenylephrine misuse incident in this case, we promptly implemented corrective measures. Rooted in the similar packaging of the two drugs, the error occurred when the pharmacy misplaced phenylephrine vials into dexamethasone storage boxes. We immediately reported this adverse event to the hospital quality control center to raise awareness among relevant departments and facilitate rectification. Meanwhile, we procured specialized storage boxes for high-alert medications, which are designated for storing vasoactive agents, potassium chloride, and other high-risk drugs, to achieve segregation from regular medications; additionally, we suspended the storage of dexamethasone in standardized boxes, with clinical demand to be met by pharmacy dispensing upon request. Given that the involved staff member was a junior resident physician, not only did we intensify the intensity of pre-service training, but we also added reflective teaching courses based on clinical adverse cases [12]. As confirmed by a previous study [13], reflective training can improve diagnostic accuracy in uncertain and complex scenarios, thereby reducing medical errors. Investigation into the incident revealed that the resident physician had been experiencing fatigue due to prolonged working hours, which contributed to the medication preparation error. In China, the shortage of anesthesiologists and excessively long working hours are the major causes of professional burnout among this cohort [14]. Similarly, an overseas study has also indicated that anesthesiologists worldwide are confronted with unprecedented work pressure and staffing shortages, with professional burnout being an extremely prevalent issue [15]. Therefore, improving the working conditions of anesthesiologists is imperative and urgently requires attention at the national level.

Following the incident, the relevant personnel reported it to the hospital's adverse event management system without delay. Subsequently, the department convened a special meeting and decided to temporarily remove the dexamethasone storage box from the standard anesthesia drug kits. If dexamethasone is indeed required during surgery, medical staff should retrieve it from the operating room pharmacy as needed.

In the meantime, the department customized a batch of dedicated storage boxes for high-alert medications, which are used to store vasoactive agents, potassium chloride, and other high-risk preparations, thus achieving the classified storage and management of general medications and high-alert medications.

In addition, the department has specially added a dedicated training module on such medication error incidents to the standardized training curriculum for resident physicians, aiming to reduce the risk of recurrence of similar events.

Conclusion

The accidental intravenous administration of a high dose of phenylephrine (10 mg) poses a significant risk of sudden pulmonary edema, requiring vigilant attention from an anesthesiologist. While the patient recovered and was discharged, this case underscores critical lessons for clinical

practice and highlights areas for improvement. We hope that this report will serve as a cautionary example for clinicians, emphasizing the importance of vigilance and precision in drug administration. Preventing such incidents necessitates a multifaceted approach, including enhanced post-graduation education for anesthesiologists, proactive measures to address anesthesiologist burnout, and improvements in drug packaging to minimize the likelihood of medication errors.

Conflict of interest

The authors declare no conflict of interest.

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