

KLINIKINIS ATVEJIS

Pulmonary edema and hemorrhage as complications of acute airway obstruction following anesthesia

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Key words: airway obstruction; pulmonary edema; pulmonary hemorrhage; ketamine anesthesia.

Summary. Airway obstruction is a quite common complication while its conditioned pulmonary edema – rare. Causes associated with anesthesia are various. Forced inspiratory efforts against an obstructed upper airway generate peak negative intrathoracic pressure. This may cause pulmonary edema and in some cases pulmonary hemorrhage. Last-mentioned is extremely rare.

Pulmonary edema may arise soon after airway obstruction as well as later, after some hours. Damage of bronchi is found seldom during bronchoscopy in case of pulmonary hemorrhage, while more often alveolar damage is observed due to alveolar membrane damage. Hemorrhage is conditioned by hydrostatic pressure level, level of hypoxia, damage to bronchi or alveoli (disruption of alveolar membrane).

Early diagnosis of negative-pressure pulmonary edema or pulmonary hemorrhage is very important, because this affects postoperative morbidity and mortality of the patients.

Two cases of pulmonary edema and hemorrhage after upper airway obstruction as well as literature overview are presented in this article. Pulmonary hemorrhage developed during anesthesia with ketamine, conditioned by increment of hydrostatic pressure, hypoxia, and effects of ketamine on hemodynamics.

Background

Negative-pressure pulmonary edema (NPPE) as a complication of upper airway obstruction is well known and it has been described in the literature on anesthesia since 1977 (1). The incidence of NPPE among complications of general anesthesia reaches 0.1% according to different authors (2, 3). In pediatric patients, this complication is observed even more often (9.4–9.6%) (4, 5).

As this condition might be life threatening, NPPE is an important cause of perioperative morbidity and mortality (6–9).

Its mechanism is upper airway obstruction and subsequent increase in negative intrathoracic pressure (10). This results in increased preload to right parts of the heart, causing increment of hydrostatic pressure in pulmonary capillaries, which subsequently leads to fluid transudation to alveoli (11). Typical clinical findings such as frothy sputum and easily recognizable radiological changes are observed. Pulmonary hemorrhage following upper airway obstruction is an extre-

mely rare phenomenon. Vasoconstriction due to hypoxia and hyperadrenergic state is among possible factors for the development of such edema and hemorrhage.

Further, we report two cases: severe postobstructive pulmonary edema following laryngospasm in a pediatric patient and negative-pressure pulmonary hemorrhage during ketamine anesthesia in an adult patient.

Case 1

A 14-year-old boy (weight, 53 kg) was hospitalized to the Clinic of Pediatric Surgery for an urgent appendectomy. His medical history and preoperative physical examination did not reveal any signs of pathology. The diagnosis of appendicitis was based on echoscopic findings. The decision to perform appendectomy was made within one hour after hospitalization. Anesthesia was induced with 450 mg of sodium thiopental, 100 µg of fentanyl citrate, and 25 mg of atracurium besylate. Tracheal intubation, anesthesia,

mechanical ventilation, and surgery were uneventful. Anesthesia was maintained with halothane (minimum alveolar concentration (MAC), 0.7–0.8) and 200 µg of fentanyl citrate. During the procedure, the patient had stable blood pressure and oxygen saturation (SpO₂ 98–99%); ETCO₂ ranged from 32 to 36 mm Hg. The duration of surgery was 50 min. A volume of 1000 mL of Ringer lactate solution was administered (the patient did not receive any infusion therapy the day before surgery).

When the operation ended and the patient woke up, endotracheal tube was removed. After the extubation, intensive cough has started. The patient became agitated, and marked inspiratory stridor developed. Status was assessed as laryngospasm. The anesthesiologist attempted to ventilate the patient through the mask; despite this, oxygen saturation has decreased to 80%.

After administration of 5 mg diazepam and 100 mg succinyl methonium, the patient was re-intubated and ventilated at a fraction of inspired oxygen (FiO₂) of 1.0. SpO₂ increased to 96–98%. After 30 min, the patient was woken up with SpO₂ of 98–99% at FiO₂ of 0.6. Cardiovascular status was stable. Respiratory sounds were normal. The patient was extubated because of intolerance to endotracheal tube.

After the extubation, severe inspiratory stridor (laryngospasm) reoccurred. Inspiratory efforts were insufficient despite active work of respiratory muscles. He continued to make inspiratory “crowing” sounds. Oxygen therapy through mask was ineffective during 15 min; hypoxia (SpO₂ 96–78%), hypercapnia (ETCO₂ 39–50%), and tachycardia (up to 120 beats/min) have progressed. Sodium thiopental at a dose of 100 mg and succinyl methonium at a dose of 50 mg were administered, and the patient was re-intubated again. Auscultation revealed moist rales. During suction, foamy secret was evacuated from the endotra-

cheal tube.

This status was defined as postobstructive pulmonary edema. The diagnosis was confirmed by x-ray examination (signs of stasis and interstitial changes in both lungs). Mechanical ventilation with application of positive-end expiratory pressure (up to 6 cm of water) was continued. After the stabilization of status (FiO₂ 0.7, SpO₂ 96%, ETCO₂ 45 mm Hg), the patient was transferred to pediatric intensive care unit continuing mechanical ventilation with a portable mechanical ventilator.

The patient was sedated by intravenous administration of diazepam and ventilated for 9 h applying positive end expiratory pressure (PEEP) regimen up to 5 cm of water, later – continuous positive airway pressure (CPAP) regimen. During all this period, foamy secret was evacuated from the endotracheal tube. The need of oxygen therapy was decreased progressively during the following day. The patient was extubated only after 24 h. Chest x-ray examination performed after 9-hour mechanical ventilation revealed a progressive resolution of pulmonary edema – interstitial changes in the right upper lobe were still observed despite reduction of edema signs in other parts of the lungs. Regressing rales were auscultated for 36 hours. The patient was discharged to a regular ward, the Department of Pediatric Surgery, after 34 h from the admission to the pediatric intensive care unit (PICU). Further postoperative period was uneventful. The patient was successfully discharged for outpatient follow-up on the 7th day of hospitalization. Changes in blood gases are presented in Table.

Case 2

A 40-year-old man was admitted for urgent surgery due to acute paraproctitis. Previously subjectively healthy patient had no complaints except tenderness

Table. Changes in blood gases during the follow-up of the patient

Parameter	Before last intubation	2 hours after admission to PICU	4 hours after admission to PICU	12 hours after admission to PICU	17 hours after admission to PICU
pH	7.17	7.41	7.427	7.524	7.435
pCO ₂ , mm Hg	71.5	36.9	25.5	24.6	30.1
pO ₂ , mm Hg	71.8	381.4	165.8	165	277.3
BE	-9.1	-0.6	-5.8	-0.7	-2.7
SpO ₂ , %	89.9	99.9	99.3	99.4	99.8
FiO ₂	0.7	0.6	0.4	0.3	0.3
Condition	MV	MV	MV	CPAP	CPAP

MV – mechanical ventilation, CPAP – continuous positive airway pressure; PICU – pediatric intensive care unit; BE – base excess.

and pain in perianal region. According to medical records, the patient suffered from arterial hypertension and was treated daily with angiotensin-converting enzyme inhibitor, enalapril. The patient was a heavy smoker (more than 20 cigarettes per day). Initial examination revealed blood pressure of 148/94 mm Hg, heart rate—78 beats/min; respiration rate was 14 breaths/min. The weight of patient was 110 kg, height—184 cm, body mass index—32.8 kg/m². The patient was evaluated as II class according to the American Society of Anesthesiologists physical status classification (regarding to hypertension, smoking, and obesity).

Spinal anesthesia was recommended for the patient, but the patient insisted on general anesthesia. According to initial plan of surgery—it was planned to perform incision and drainage of subcutaneous abscess—it was decided to perform intravenous anesthesia with ketamine and fentanyl citrate. Premedication of 10 mg of diazepam was administered.

An intravenous dose of 2 mg/kg of ketamine and 1 µg/kg of fentanyl citrate fractionally were given in the operating room. Oxygen therapy through facial mask at a rate of 4 L/min and infusion therapy of 500 mL of sodium chloride were applied. Initial 15 min of anesthesia and surgery were uneventful. During the surgery, it was discovered that abscess penetrated to deeper pararectal regions reaching prostate; therefore, the decision was taken to increase the extent of the surgery—to perform drainage of whole abscess cavity.

Anesthesia level was deepened by administration of 200 mg of ketamine intravenously. Despite the patient had sufficient and even vigorous respiratory muscle contractions, upper airway obstruction occurred, manifesting as inefficient respiratory efforts, following by cyanosis; hypertension (190/110 mm Hg), tachycardia (up to 120 beats/min), and following bradycardia were observed. Ventilation through facial mask was inefficacious. Within 10 min, it was tried to ventilate the patient through a facial mask by applying methods for airway management (oropharyngeal tube was inserted), and oxygen delivery was increased to 8 L/min. Oxygen saturation was decreased from 99% to 86% despite appropriate measures applied. Surgery was successfully finished at this time.

Tracheal intubation and general endotracheal anesthesia were performed giving 100 mg of succinyl methonium, 0.2 mg of fentanyl citrate, and 500 mg of sodium thiopental. The patient was successfully intubated. Laryngoscopy has not reveal any pathological signs. Lung ventilation was initiated by applying FiO₂ 1.0. ET-CO₂ was slightly decreased from 60 to 43 mm

Hg after intubation. Soon after intubation, bloody sputum was observed in the endotracheal tube.

Urgent bronchoscopy was performed in the operating room to exclude possible aspiration, as saturation after intubation remained 86–87% despite the ventilation by applying 100% oxygen. Bronchoscopy did not reveal any signs of aspiration or bronchial damage; only bloody sputum was found in trachea and principal bronchi. Saturation was gradually increased from 86 to 97%, despite the application of PEEP up to 8 cm H₂O. Blood gases showed hypoxemia: pH, 7.28; pO₂, 40.2 mm Hg; pCO₂, 63.2 mm Hg; base excess (BE), 7.1; SpO₂, 89.2%; while FiO₂, 0.8.

It was decided to transfer patient to the intensive care unit (ICU) for further mechanical ventilation and postoperative care.

Chest x-ray examination was performed in the ICU, which revealed the signs of stasis and interstitial edema in both lungs progressing to alveolar edema in the right lung.

After 2 h, the patient was extubated, but signs of respiratory insufficiency exacerbated due to this patient was re-intubated and mechanical ventilation was extended applying PEEP (up to 6 cm H₂O). The patient was treated with diuretics, furosemide at a dose of 80 mg intravenously. Other medications included 10 mg of morphine hydrochloride and 10 mg of diazepam for sedation of the patient, 500 mg of metronidazole.

Bloody sputum was observed in endotracheal tube during the first 3 h of the treatment in the ICU. Clinical signs of pulmonary edema disappeared after 4 h from admission to ICU. Subsequent x-ray examination was performed showing reduction of interstitial and stasis changes in both lungs. After 5 h, the patient was extubated. Next morning, the patient was discharged to surgical department and after two days for outpatient follow-up.

Discussion

We have presented a case of 15-year-old pediatric patient who underwent routine surgery performed under conditions of general anesthesia and who experienced hypoxemic respiratory failure following extubation due to prolonged laryngospasm. Vigorous inspiratory efforts in the case of obstructed airway led to the development of NPPE. Resolution of this edema was slow; duration of treatment in the intensive care unit was relatively prolonged.

NPPE due to upper airway obstruction is a well-recognized problem.

The most common reason of NPPE is laryngo-

spasm (12–17), and less common reasons are as follows: endotracheal tube (18) or laryngeal mask (19, 20) clench during wake up after general anesthesia, intensive hiccup during anesthesia (21), aspiration (8), or bilateral palsy of vocal chords (22).

An extremely rare case of NPPE was described, induced by direct suction of endotracheal tube adapter (23). During postanesthesia, NPPE is observed more often among obese patients, patients with short neck or with sleep apnea (24), and patients who underwent ear, nose, and throat surgery (6, 25).

Recently, original papers have been published describing data of Australian Incident Monitoring Study (AIMS) concerning 4000 incidences during anesthesia (8, 12, 26). According to some authors, NPPE is rather common and is considered to occur in up to 4% of all incidence reports of laryngospasm, and only in two cases (among 4000), pulmonary edema developed due to other etiology of acute airway obstruction.

What influences the occurrence of NPPE?

Intrathoracic pressure during obstruction seems to be the most significant in NPPE pathogenesis. This leads to subsequent increment of venous blood return and intrathoracic blood volume, decreases pulmonary venous outflow resulting in decreased cardiac output. In turn, increment of hydrostatic pressure gradient in pulmonary capillaries induces fluid transudation to alveoli and development of edema (7, 27, 28).

Maximal negative inspiratory pressure exceeds -50 – 100 cm H₂O during acute airway obstruction in adults, especially after vigorous inspiratory efforts (29), while in pediatric patients it reaches -24 – 50 cm H₂O (28). Normal inspiratory pressure ranges from -2.5 to -10 cm H₂O.

As we experienced in our cases, respiratory efforts are more prominent among young, athletic men with good thoracic musculature (30), as well as children with greater chest wall compliance are able to generate more negative pressure (28).

Hypoxemia is among other reasons, possibly causing the development of pulmonary edema (31, 32). Vasoconstriction, conditioned by hypoxemia and increment of catecholamines due to stress and subsequent increment of left ventricle afterload, plays a great role in the development of such type edema. On the other hand, hypoxemia and stress activate sympathetic nervous system and subsequently increase venous blood return, induce vasoconstriction of pulmonary capillaries, thus favoring a hydrostatic fluid shift into the interstitium (33).

Hydrostatic mechanism of pulmonary edema development without alveolo-capillary membrane lesion

was proved by studies measuring the ratio of total protein concentration between pulmonary edema fluid and plasma of adults and pediatric patients (34, 35). A ratio of less than 0.65 is characteristic of hydrostatic pulmonary edema, whereas a ratio between 0.75–1.0 is characteristic of acute lung injury.

NPPE diagnosis after acute airway obstruction is confirmed by clinical and x-ray findings. On the other hand, chest x-ray examination is not always performed after airway obstruction (36). According to this, it would be fair enough to acknowledge that NPPE ratio following anesthesia is greater than is diagnosed. One study reported that chest x-ray examination revealed up to 29% of pulmonary edema cases following airway obstruction with no clinical evidence of pulmonary edema (37). In case of high hydrostatic pressure, erythrocytes might diffuse through alveolo-capillary membrane, resulting in pink frothy sputum, while blood (hemoptysis) indicates the damage to alveolo-capillary membrane.

Negative-pressure pulmonary hemorrhage conditioned by acute airway obstruction is an extremely rare condition. Diffuse alveolar hemorrhage for the first time was described by Schwartz et al. in 1999 (30) and later by other authors (2, 9, 36–39). As no signs of tracheal and bronchial mucous damage were found during bronchoscopy, and fresh blood was aspirated during broncho-alveolar lavage (9, 30, 39), this pathology was qualified as negative-pressure alveolar hemorrhage. Analysis of hemorrhagic fluids aspirated during bronchoscopy showed rather high levels of hemoglobin (40). Only Koch et al. found hemorrhage lesion lining the trachea and main airways (41).

The mechanism of negative-pressure pulmonary hemorrhage remains unclear. It is discussed that mechanical rupture of alveolo-capillary membrane due to increased negative intrathoracic pressure conditions diffuse damage of pulmonary capillaries. Rare cases of hemoptysis are described due to clench of the endotracheal tube after general anesthesia, which lasted in one case only 20 s (9), while in other case – 1–2 min (38).

Recently, Pandey et al. discussed that some agents used in anesthesia, such as ketamine, could be responsible for the development of pulmonary edema (42). They report a case of pulmonary edema after intramuscular injection of ketamine in a previously healthy girl who underwent necrectomy because of burns. There is at least one report of pulmonary hypertension and pulmonary edema following ketamine administration in a patient who had a history of crack

cocaine abuse (43).

Ketamine is a potent sympathomimetic. Even in healthy patients, ketamine produces an increased pulmonary artery pressure and increases pulmonary vascular resistance (44–46). The cardiovascular effects produced by ketamine resemble sympathetic nervous stimulation. The mechanism for the ketamine-induced cardiovascular effects is complex. Direct stimulation of the central nervous system leading to increased sympathetic nervous system outflow seems to be the most important mechanism (47).

Pulmonary hemorrhage as it was presented in our second case (the only case in our clinic) was associated

with increment of intrathoracic pressure due to vigorous inspiratory efforts produced by the patient. Most probably negative pressure was the leading cause of it, while other additional factors such as hypoxia and impact of ketamine may be important in the development of pulmonary hemorrhage. Although the precise etiology of the bleeding in pulmonary hemorrhage is uncertain, the disruption of pulmonary capillaries could play a role. The application of term “negative-pressure pulmonary hemorrhage” is recently under discussion and seems to be acceptable to describe the development of diffuse alveolar hemorrhage following exposure to negative intrathoracic pressure.

Neigiamo slėgio plaučių edema ir plaučių hemoragija, sąlygota po anestezijos atsiradusios kvėpavimo takų obstrukcijos

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Raktažodžiai: kvėpavimo takų obstrukcija, plaučių edema, plaučių hemoragija, ketamino anestezija.

Santrauka. Kvėpavimo takų obstrukcija gana dažna komplikacija, atsirandanti po anestezijos. O jos sąlygota plaučių edema yra reta. Intensyvios įkvėpimo pastangos sąlygoja neigiamo intratorakalinio slėgio atsiradimą. Tai gali sukelti plaučių edemą, o kai kuriais atvejais ir plaučių hemoragiją. Pastaroji ypač reta.

Plaučių edema gali išsivystyti tuoj pat po kvėpavimo takų obstrukcijos, tačiau atskirais atvejais gali atsirasti praėjus net kelioms valandoms po buvusios kvėpavimo takų obstrukcijos. Plaučių hemoragijos atveju bronchų pažeidimas bronchoskopijos metu pastebimas retai, o alveolių pažeidimas yra sąlygotas alveolių membranos pažeidimo. Hemoragija yra sąlygota padidėjusio hidrostatinio slėgio, hipoksijos, bronchų ir alveolių pažeidimo (alveolinės membranos įplyšimas).

Ankstyva neigiamo slėgio plaučių edemos ir plaučių hemoragijos diagnostika yra labai svarbi, nes tai turi įtakos pooperaciniam pacientų sergamumui ir mirštamumui.

Straipsnyje pateikiami du plaučių edemos ir hemoragijos atvejai, sąlygoti po anestezijos išsivysčiusios kvėpavimo takų obstrukcijos. Plaučių hemoragija išsivystė anesteziją atliekant ketaminu. Būklė buvo sąlygota padidėjusio hidrostatinio slėgio, hipoksijos ir ketamino įtakos hemodinamikai.

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