PATIENT-VENTILATION ASYNCHRONY CAUSING NEGATIVE PRESSURE PULMONARY EDEMA IN AN INTUBATED OBESE PATIENT

Sahar M. Siddik-Sayyid*, Waseem AlFahel**
and Mohamad F. El-Khatib*

Negative pressure pulmonary edema is a potentially life-threatening condition that may occur when a large negative intrathoracic pressure is generated against a ‘physically’ obstructed upper airway during emergence from anesthesia. We report a 35 year old male patient who is morbidly obese and undergoing laparoscopic gastric bypass who developed negative pressure pulmonary edema without any evidence of a ‘physical’ upper airway obstruction. In our patient, the negative pressure pulmonary edema occurred after complete reversal of neuromuscular blockade and during manual positive pressure ventilation with the endotracheal tube still in place and in the presence of an oral airway. Since the patient was still intubated and had an airway in place with no possibility for physical obstruction, we speculate that the occurrence of the negative pressure pulmonary edema was mainly due to a ‘functional’ obstruction secondary to the severe patient-ventilation asynchrony that ensued upon reversal of the neuromuscular blockade.

Keywords: Negative pressure pulmonary edema; endotracheal tube; Asynchronized ventilation

Negative pressure pulmonary edema (NPPE) is a serious complication that may develop in the event of upper airway obstruction (UAO) during the emergence from anesthesia following extubation1. Normally the airway obstruction is physical and is manifested as a laryngospasm or other causes of physical upper airway obstructions2. Patients with difficult airways due to anatomic variations (i.e. short neck, nasopharyngeal soft tissue disorders), history of obstructive sleep apnea (OSA), obesity, and acromegaly are considered to be at higher risk for developing UAO and NPPE while emerging from anesthesia3. We report an unusual case of NPPE in an obese patient upon emergence from general anesthesia for bariatric surgery, while the patient was still intubated and having an oral airway in situ with no chance for biting on the tube and no possibility of an obstruction. We postulate that NPPE in our patient was secondary to a ‘functional’ rather than ‘physical’ upper airway obstruction. Written informed consent was obtained from the patient.

Case Presentation

A 35 year old male patient presented for an elective laparoscopic gastric bypass under general anesthesia. The patient had a past medical history of hypertension (controlled by amlodipine) and OSA. His body mass index (BMI) was 42 kg/m². The preoperative assessment revealed Mallampati class II, adequate mouth opening, thyromental distance of 5 cm, and a large neck circumference. His laboratory investigations were all within normal limits. Echocardiography was normal with an

* Professor, Department of Anesthesiology, American University of Beirut-Medical Center, Beirut-Lebanon.
** Medical Resident, Department of Anesthesiology, American University of Beirut-Medical Center, Beirut-Lebanon.
Address correspondence to: Dr. Mohamad El-Khatib, Professor Department of Anesthesiology, American University of Beirut, P.O. Box 11-0236 Beirut, Lebanon, Fax#: 961-1-745249. E-mail: mk05@aub.edu.lb
Conflict of Interest: None of the authors declare any personal, professional or business conflict of interest.
In the operating room, general anesthesia was induced with midazolam, xylocaine, fentanyl, and propofol. The patient was given succinylcholine and was smoothly and successfully intubated by direct laryngoscopy with the aid of a bougie. Anesthesia was maintained with sevoflurane and remifentanil. Intermittent boluses of rocuronium were supplemented to maintain adequate level of neuromuscular blockade (NMB). The patient received a total of 2 liters of crystalloids given that the blood loss was minimal, and his hemodynamics variables were all well maintained. At the end of the surgery which lasted for 2 hours, sevoflurane and remifentanil were stopped, and morphine 5 mg IV was given to control postoperative pain. Furthermore, sugammadex (400 mg IV) was administered to ensure complete reversal of NMB. The patient began spontaneous breathing and started regaining his consciousness while receiving gentle manual ventilation with an oxygen saturation of 98-99%. However, prior to extubation and removal of the oral airway and upon performing gentle suctioning of the oropharynx, the patient became severely agitated. Only forceful manual ventilation could be applied to overcome the patient’s agitation and ensure adequate ventilation. This was very difficult to impossible as the patient was extremely agitated and fighting against the manual breaths; then oxygen saturation started to decrease and reached 70-75%. Propofol (50 mg IV) was given immediately to sedate the patient and enable synchronized and efficient ventilation. Endotracheal tube (ETT) suctioning showed large amount of pink frothy secretions. Chest auscultation revealed bilateral inspiratory crepitus. The patient was kept intubated and manually ventilated. Chest x-ray (CXR) showed bilateral infiltrates with congestion consistent with the diagnosis of pulmonary edema. Manual positive pressure ventilation was continued and resulted in improvement in oxygen saturation to the range of 85% to 90%, and lasix (20 mg IV) was given. Thirty minutes later, the patient started to wake up and became fully conscious, more cooperative, and able to maintain his oxygenation (85%-90%) by spontaneous breathing without manual ventilation. Arterial blood gases revealed pH=7.31, PaO₂=59mmHg, PaCO₂=47mmHg, HCO₃⁻=23mEq/L, SaO₂=88%, BD=−2.7mEq/L. The patient was extubated to noninvasive ventilatory support in the form of bilevel positive airway pressure (BiPAP) and was transferred to the recovery room where he received an additional dose of lasix (40 mg IV), and was kept on BiPAP. His oxygen saturation improved to 95-96%. The next day, arterial blood gas showed significant improvement in arterial blood gases (pH=7.41, PaO₂=102mmHg, PaCO₂=43mmHg, HCO₃⁻=27mEq/L, SpO₂=98%, BD=2.7mEq/L). Consequently, the patient was deescalated to oxygen facemask, and was transferred to a regular floor. He was discharged home two days later after showing normal clinical and radiological findings.

**Discussion**

The pathophysiology of the NPPE is multifactorial, and is thought to be a result of a gradient between the negative intrathoracic pressure generated by vigorous inspiratory efforts against an obstructed airway and the positive hydrostatic pressure of the pulmonary capillaries (created mainly by the increased venous return to the right heart accompanied by the hypoxic-induced systemic and pulmonary vasoconstriction). This gradient favors a transudation of fluid from capillaries into alveoli, and the resulting edema is referred to as type I NPPE.

In our case, the clinical findings (rapid desaturation, pink frothy secretion and inspiratory crepitus on chest auscultation) as well as the radiological findings (bilateral infiltrates with congestion) were strongly suggestive of pulmonary edema. The negative cardiac history of the patient speaks against a cardiac source for the observed lung edema. The presence of an ETT although decreases but does not eliminate the possibility for an obstruction and subsequently the development of NPPE. Sow Nam et al. reported a case of significant negative pressure pulmonary edema in an intubated patient who was biting on the ETT and causing airway obstruction. However in our intubated patient, an oral airway was inserted throughout the whole procedure which excludes the possibility of external airway obstruction by biting on the endotracheal tube.

At the time our patient developed pulmonary edema, he was still intubated but had already regained full muscle power following administration of
sugammadex. Forceful breathing against a physical obstruction could not be the underlying cause of NPPE in our patient since he was still intubated and could not bite on the ET tube secondary to the presence of the mouth piece. We speculate that the main cause for the NPPE in our patient is the severe and repetitive asynchronization between the patient’s vigorous breathing efforts and the aggressive prolonged and deep manual ventilation that was provided by the anesthesia team while the patient was agitated and exhibiting oxygen desaturation in an attempt to provide ventilation to the patient’s lungs and reverse the oxygen desaturation. Bhaskar and Fraser described a type II NPPE that is related to forceful exhalation against an obstruction which creates intrinsic positive end expiratory pressure in the alveoli. This mimics the valsalva maneuver which increases the alveolar pressure and decreases the venous return to the pulmonary vasculature, thus decreasing the pulmonary capillary pressure. Sudden relief of the obstruction causes abrupt decrease in alveolar pressure as well as increase in the venous return to the pulmonary capillaries, which in turn increases the hydrostatic pulmonary capillary pressure. Consequently, the pressure gradient will increase leading to pulmonary edema. Due to the severe agitation, it is highly probable that our patient was forcefully exhaling while positive pressure was being applied through aggressive manual ventilation. This could have created an obstruction against the patient exhaled breath. Releasing the bag and pausing manual ventilation would result in abrupt resolution of the obstruction which may contribute to the development of pulmonary edema type II by the above-mentioned mechanism.

Our unusual case illustrates the possibility of severe patient-manual ventilation asynchrony in the development of NPPE. This highlights the importance of smooth resumption of spontaneous breathing prior to extubation especially in such scenarios where the patient is obese, young, with a history of OSA and with full recovery of neuromuscular power. In such scenarios, we recommend the use of pressure support ventilation that can provide adequate ventilatory assistance while maintaining superior synchrony with the patients’ spontaneous breaths.
References

1. LORCH DG, SAHN SA: Post-extubation pulmonary edema following anesthesia induced by upper airway obstruction. Are certain patients at increased risk? *Chest*; 1986, 90:802-805.
BRIDION—for optimal neuromuscular blockade management and improved recovery

Predictable and complete reversal
- 98% of BRIDION patients recovered to a TOF* ratio of 0.9 from reappearance of T₂ within 5 minutes²
- 97% of BRIDION patients recovered to a TOF* ratio of 0.9 from 1 to 2 PTCs* within 5 minutes³

Rapid reversal
- BRIDION rapidly reversed patients from reappearance of T₂ in 1.4 minutes¹
- BRIDION rapidly reversed patients from 1 to 2 PTCs† in 2.7 minutes³

BRIDION is indicated for the reversal of neuromuscular blockade induced by rocuronium or vecuronium. In children and adolescents (aged 2-17 years), BRIDION is only recommended for routine reversal of moderate rocuronium-induced neuromuscular blockade.

Important safety information
BRIDION is not recommended in patients with severe renal impairment. Studies in patients with hepatic impairment have not been conducted and therefore, patients with severe hepatic impairment should be treated with great caution. Caution should be exercised when administering BRIDION to pregnant women as no clinical data on exposed pregnancies are available.

BRIDION has not been investigated in patients receiving succinylcholine or succinylcholine in the intensive care unit (ICU) setting.

If neuromuscular blockade is required within 24 hours of BRIDION administration, a non-depolarizing neuromuscular blocking agent should be used instead of succinylcholine or succinylcholine. The most commonly reported adverse reactions were dizziness (mild or bilateral tinnitus) and anesthetic complications (movement, coughing, grinning, or talking on the endotracheal tube). In patients treated with BRIDION, a few cases of awareness were reported. The relation to BRIDION was uncertain. In a few individuals, allergic-like reactions (i.e.: flushing, erythematous rash) following BRIDION were reported. Children should be prepared for the possibility of allergic reactions and take the necessary precautions. Bronchospasm was reported in 2 patients and a causal relationship could not be fully excluded.

Withdrawal studies have demonstrated a state (17%) to 20%) prolongation of the pentobarbital concentration (in PTCs confirmed with BRIDION; however, clinical studies have demonstrated no clinically relevant effect on postoperative bleeding complications with BRIDION alone or in combination with anticoagulants. As BRIDION has demonstrated in vitro pharmacodynamic interactions with anticoagulants, caution should be exercised in patients on anticoagulants for a proper or corrected condition. This pharmacodynamic interaction is not clinically relevant for patients receiving routine postoperative prophylactic anticoagulant.

Although funnel-shaped studies have not been conducted, no drug interactions were observed in clinical trials. Preclinical data suggest that clinically significant drug interactions are unlikely with the possible exceptions of tobramycin, lidocaine, and hemorhoidal ophthalmic.

¹ Time of four
² PostSURGICAL CARE
³ Second twitch


Please see summary of product characteristics for full prescribing information.

MSD Be Well

Copyright © 2010 Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc., Whitehouse Station, NJ, USA. All rights reserved. 05-2013 BRID-2011 LEVANT-1196 J
**PAJUNK® Pioneering Medical Technology**

**TAP Block And InfiltraLong**
For Effective Treatment Of Long And Deep Incisions

**Sono Cannulas**
For Single Shot Ultrasound Guided Nerve Blocks

**SonoSystem And SonoLong Curl**
For Ultrasound Guided Nerve Blocks

**Sprotte® 2.G**
The New Generation Dura Puncture In Minimum Time

**SonoEye Ophthalmic Block**
For Peribulbar And Retrobulbar Blocks Under Ultrasonic Monitoring

www.mediline-lb.com Tel:+961 1 697500
Question.
Your patient requires urgent pain medication. How can you administer this less invasively?

Answer.

LMA MAD Nasal™
Needle-free intranasal drug delivery device

Atomization spray
The spray atomizes drugs into a fine mist of particles (50-100 microns in size).¹

Malleable stylet
The malleable stylet allows 180° positioning of the nasal plug.

Accurate dosing
The syringe enables the accurate measurement of drugs to be delivered.

Pressure
High applied pressure ensures that drugs are atomized into a fine mist of particles through the tip of the plug.

Soft conical plug
The plug forms a seal with the nasal passages, preventing expulsion of fluid.

Spray geometry
Spray cone with a wide 62.7° average spray angle and a 36.8 mm average plume width.²

References:
PRINCIPLES OF PEDIATRIC ANESTHESIA AND CRITICAL CARE
The Fairmont Copley Plaza Plaza Boston
May 6-8, 2016

GUEST SPEAKERS
DEAN ANDRIPoulos, MD, MHCM
CHARLES COTE, MD
KELSEY TAINSH
BRAND TUMOR SURVIVOR AND MOTIVATIONAL SPEAKER

WORKSHOPS
Pediatric Airway (Included in tuition)
TEE
Regional Anesthesia
MOCA® SIMULATION COURSE

COURSE DIRECTORS:
Kirsten C. Odegard, MD
Mary Ellen McConn, MD, MPH
Janet Valicenti, CRNA
Bistra Vlassakova, MD

SAVE $75
Promo Code: PediatricAnesthesia75
Expires 1/31/16

Conference focused on pediatric anesthesia research, hot topics, challenges, risk management techniques, and interactive workshops
www.PediatricAnesthesiaConference.com