Case Report

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Negative pressure plmonary oedema after adenotonsillectom: a case report and litrature review

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Background: Negative pressure pulmonary is a non-cardiogenic pulmonary oedema that can occur after reliving of upper airway obstruction. It is life threatening clinical scenario developed due to increase intrathoracic pressure during marked inspiratory effort against a closed glottis.

Case presentation: A successful adenotonsillectomy was done for a healthy 12-year-old, 33 kg male patient and transferred to post-anaesthesia care unit. In the unit the patient developed signs and symptoms of negative pressure pulmonary oedema so he was treated with diuretics and oxygen while restricting fluid. However, the dyspnoea was persisted so he was transferred to ICU.

Clinical discussion: This case report shows the development of negative pressure pulmonary oedema after adenotonsillectomy. In this report the clinical presentation, diagnosis, treatment, prevention and prognosis of negative pressure pulmonary oedema after adenotonsillectomy was discussed in detail with citing updated evidences.

Conclusions: Post-extubation pulmonary oedema can occur after reliving of chronic air obstruction. Usually it occur within 5 min after reliving the obstruction but it can occur at any time. All healthcare professionals must be knowledgeable about clinical presentation and managements of negative pressure pulmonary oedema.

Keywords: adenotonsillectomy, negative pressure, obstruction, pulmonary oedema

Introduction

Negative pressure pulmonary oedema (NPPE) is also known as post-obstructive pulmonary oedema caused by a rapid increase in negative intrathoracic pressure. The pathological relationship between upper airway obstruction and pulmonary oedema was first described in 1927 by Moore and Binger[1] and first case published in 1927 C by RL Moore in spontaneously breathing dogs exposed to resistive load[2]. This oedema is a non-cardiogenic pulmonary oedema and majority of patients are have been healthy, without underlining pulmonary or cardiac disease. Therefore significant portion of patients with NPPE recovers with observation and oxygen supplementation.

Based on the cause NPPE classified into types 2, each with similar clinical pictures[2,3]. NPPE varies in severity of presentation, and there are no specific indicators of decompensating or prognosis. The natural process of healing is variable, ranging from spontaneous recovers with observation and oxygen supplementation to ICU escalation and prolonged mechanical ventilation. A significant portion of patients with NPPE recovers with observation and oxygen supplementation. However, some patients require advanced interventions, including support with mechanical ventilation[4].

Risk factors that are associated with NPPE include yang male patients, airway infections or tumours in children and laryngospasm during anaesthesia or following extubation in adults patients and overzealous intraoperative fluid administration[5]. It is the result of marked decrease in intrathoracic pressure caused by marked inspiratory effort against a closed glottis and result in disruption of the normal intravascular starling mechanism ultimately leads to transudation of intravascular protein and fluid

HIGHLIGHTS

- Negative pressure pulmonary oedema (NPPE) develops due to increase intrathoracic pressure secondary to reliving of upper airway obstruction or laryngospasm.
- The incidence varies 9.6–12% for type I and 40–45% for Type II.
- In adults, 50% of the NPPE cases are a result of post-operative laryngospasm.
- Usually NPPE diagnosed within 5 min after the onset of laryngospasm or reliving obstruction.
- Usually NPPE resolves within 24 h without significant complication and management will depend on the severity of pulmonary oedema.

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into the interstitium. The presentation can be immediate or delayed that could confused with other causes of postoperative respiratory distress[3].

We present the case of a healthy patient who was intubated for elective adenotonsillectomy. He was admitted to the paediatric ICU and recovered with oxygen supplementation and medical management. This is an exemplary case to alert health professionals the occurrence of NPPE after adenotonsillectomy. Physician awareness of complications that may arise after intubation is critical for preventing serious outcomes.

Case presentation

A healthy 12-year-old, 33 kg male patient was undergo adenotonsillectomy surgery for obstructive sleep apnoea secondary to hypertrophied adenoid-tonsil. He had no history of previous surgery or any medical disorders. Preoperative investigations were also within normal limits and no extra finding in physical examination.

Preoperatively oral paracetamol 500 mg was given at midnight and morning before surgery. His blood pressure was 110/70 mmHg, heart rate 92 bpm, SpO_2 94% on room air. After he arrived to the induction room, consent was taken and adequate starvation was confirmed. A 22-gauge peripheral intravenous (IV) catheter was inserted in the left hand and we premeditate with IV dexamethasone (4 mg) and metoclopramide (5 mg) and took into ENT operation room. In the OR Standard monitors applied and 50 mg pethidine was given. After pre-oxygenation for 5 min anaesthesia induction was started with Propofol (80 mg) and succinylcholine (60 mg) followed by single attempt intubation with 5.5 mm ID cuffed endotracheal tube by a Mackintosh 3 blade. Placement of the ETT was confirmed by the presence of foggy through the ETT, bilateral breath sounds and bilateral chest movement. After fixing the tube and taping the eye, role placed under shoulder for neck extension and oropharyngeal pack placed by the surgeon. Tube position was re checked after positioning and surgeons proceed the tonsillectomy.

Intraoperative maintenance was achieved with isoflurane (1–2 vol %) and vecuronium (2.5 mg). Fluid administration rate was adjusted and continued until postoperative period. Large adenoid and tonsillar tissues were resected, and blood loss was less than 30 ml. About 10 minutes prior to end of surgery, dexamethasone 4 mg and propofol 20 mg was given. At the end of surgery inhalational agent is turned off, 100% oxygen delivered and neuromuscular blockage reverses with atropine (0.5 mg) and neostigmine (1 mg). The surgical procedure lasted 55 min and the hypertrophied tensile was resected (Fig. 1) and or pharyngeal pack was removed. Throughout the surgery HR, BP and SpO2 were maintained as 85–100/min, 100/60–120/70 mmhg and 98–100%, respectively, without hemodynamic or surgical complications. Then he extubated in a deep level of anaesthesia in left lateral position with suctioning the oropharynx. Following extubation, the patient continued to spontaneously breathe with no signs of obstruction, waited until fully awakening and transferred to the post-anaesthesia care area (PACU) in left lateral position.

Upon arrival in the PACU, the SpO2 was 98% and we were handover with the responsible PACU nurse. During this time the patient became tachypnoeic, restless and developed wet cough with pink frothy sputum with fluctuating SpO2 between 85 and 92%. Progressively he become Tachycardia (HR persist > 120 beat/min) and blood pressure become high normal (>135/90). Chest auscultation revealed bilateral crepitation and wheezing. The patient does not complain about chest pain, dyspnoea or any other cardiac related symptoms. One hundred percent oxygen (SpO2 98%) with face masks at 6 l/min and diuretics (5 mg furosemide) were administered while fluid was restricted. The SpO2 continued to stay below 92% for about 15–20 minutes and began to slowly trend up to 94%. We follow him for an hour in PACU and transferred to ICU for follow-up. On the way to ICU a chest X-ray done and the result showed peri-bronchial and perihilar interstitial thickening suggestive of pulmonary oedema (Fig. 2). In ICU we keep him on nasal oxygen and administer 50 mg tramadol for pain management. Crepitation and wheezing resolve within 8 h of addition and fluid was given slowly. After following his vital signs for 24 discharged to surgical ward with BP 110/70, HR 95 and SPO2 98% with room air.

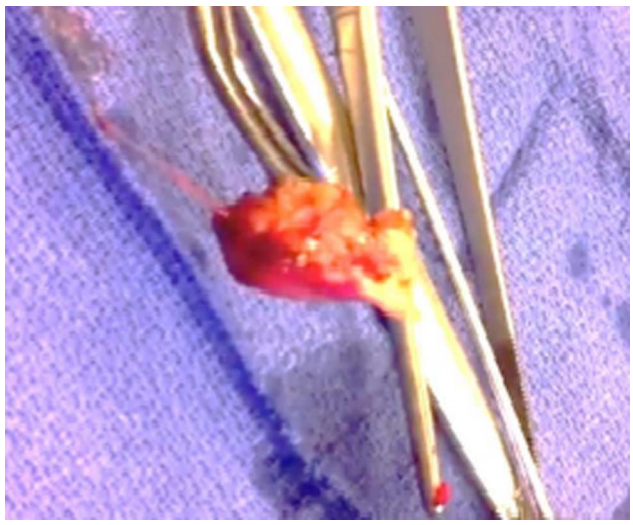

Figure 1. The hypertrophied specimen after of adenotonsille after procedure.

History of present illness

Recurrent on/off throat pain, dysphagia, snoring.

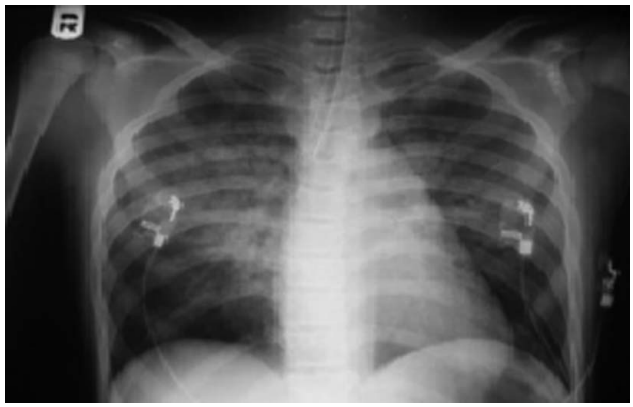

Figure 2. Chest radiograph after the development of post-obstructive pulmonary oedema (bilateral interstitial infiltrates are present).

General condition

Awake, Alert and Oriented, wt. 33, Ht. 115 cm.

Medications

No significant past medication history except for taking antibiotics and analgesia.

Allergies

Not known drug allergy.

Past medical history

Obstructive sleep apnoea + tonsillar and adenoid hypertrophy obstructive + chronic upper respiratory infections.

Physical examination

VS: within normal limit for age; BMI = 24.

HEENT: pink conjunctive, tonsillar enlargement, otherwise normal.

Airway examination: TMJ: free/mobile Mallampati class II + tonsillar enlargement.

Pulmonary: Clear to auscultation and percussion, bilateral air entry.

CVS: s1 and s2 well heard + no murmur or gallop.

Investigation

Hgb./Hct.: 12.3/39 BG: O^{+ve} PT = 12 sec[5–14] INR: 1.2,

Platelets: 283 000, electrocardiogram (ECG) normal.

Assessment

ASA II + OSA + adenotonsillar hypertrophy + fit for anaesthesia.

Methodology

After facing this case scenario we searched for supportive evidences using online databases including PubMed Central, Cochrane Library and Google Scholar using negative pressure pulmonary oedema, NPPE, post obstruction pulmonary oedema, POPE, preventive methods of post-upper airway obstruction pulmonary oedema, treatment or management of negative pressure pulmonary oedema as keywords for the articles published since 2010 to date. This case report has been reported in line with the SCARE criteria[6].

Discussion

This case report shows an example of post obstruction pulmonary oedema following adenotonsillectomy in a paediatric patient with a history of OSA. The oedema was self-limiting within 24 h with supportive treatment.

Post-obstructive pulmonary oedema also called NPPE. It is a life threatening clinical scenario in which immediate onset pulmonary oedema develops after upper airway obstruction. Patients will develop acute respiratory distress, significant hypoxaemia, and bilateral infiltrates[5].

There are two types of NPPE. Type 1 develops after sudden upper airway obstruction or after manipulation of the airway surgically and some authors call it laryngeal spasm-induced pulmonary oedema. Other factors that increase the risk of Type I NPPE are hanging, strangulation, foreign bodies, epiglottitis, croup, chocking, near drowning, endotracheal tube (ETT) obstruction, goitre, and mononucleosis[4]. Type II NPPE develops after surgical release of chronic airway obstruction caused by big tonsils, hypertrophic adenoids, or a redundant uvula such as resection. In our case the oedema is developed after reliving of adenotonsilar obstruction.

Incidence

Acute post-obstructive pulmonary oedema is a rare complication after adenotonsillectomy. Dr. P.P. McConkey review 6 cases in 2000 and reported with an incidence of 0.05–0.1%[7]. John F. Fraser also review literatures and showed that the incidence of type I NPPE is 9.6–12% while Type II NPPE was 44%[3]. In adults, 50% of the NPPE cases are a result of postoperative laryngospasm[3]. From the statistics of the Australian Incident Monitoring Study, the reported incidence rate of NPPE in patients with laryngospasm was 3%[8]. Thus, the incidence of NPPE can be ranged between 0.1 and 12%[9]. The incidence is higher in men (80%) and ASA physical states I and II (73%). This is possibly due to the fact that the healthy ones are capable of generating more negative intrathoracic pressures[4].

Pathophysiology of NPPE

In normal physiologic state intrapulmonary pressure is equal to atmospheric state. This balance is absent during obstruction and there are four main disturbance: increased hydrostatic pressure in the pulmonary capillary bed, decreased osmotic pressure of plasma, increased permeability of the membrane, and decreased lymphatic drainage[3]. Forced inspiration inducing a negative gradient of intra pleural and trans pulmonary pressure[1]. The high pressure gradient causes fluid shift from the pulmonary vessels into the interstitial spaces and alveoli of the lungs, resulting in pulmonary oedema[10]. During obstruction and forceful inspiration there will be severe mechanical stress that could damage the epithelial lining of pulmonary vasculature. This damage increase permeability of pulmonary capillary results protein-rich pulmonary oedema[3].

Clinical presentation of NPPE

Usually the sign and symptoms of NPPE can be seen within 5 min but it can occur at any time after reliving of obstruction. In our case the symptom was seen with in 5 min as most cases but Koid. M and other researchers showed that there will be a probability of developing pulmonary oedema 1–6 h after upper airway obstruction[9,11].

The patient may show symptoms of upper airway obstruction include stridor, respiratory distress, paradoxical chest movements and using the accessory muscles in breathing. The clinical presentation of acute pulmonary oedema include dyspnoea, tachypnea, cyanosis, asthma and the production of a profuse pink foamy sputum[1]. Almost all symptoms were seen on this case.

The common presentation in NPPE is the occurrence of an episode of airway obstruction at emergence from general anaesthesia, followed by the rapid onset of respiratory distress, wheezing, dyspnoea, paradoxical chest movements and bilateral radiological changes with a profuse pink foamy sputum. Of these symptoms, frothy pink sputum is the hallmark sign of NPPE[12]. But in some

cases it may take longer time to resolve, which is related to the degree of pulmonary micro vascular damage Therefore, a patient at risk must be observed in the ICU for an extended period of time[13]. In our cases the symptom resolved within 24 hour which may indicate lesser degree damage. According to Bhaskar, with on time diagnosis and proper treatment, NPPE resolves generally within 24 h without complication. However, when recognition is delayed, patients with NPPE have mortality rates ranging from 11 to 40%[4]. Some patients with NPPE may suffer from associated long-term complications, such as myocardial infarction, transient ischaemic attack, non-ST-elevation myocardial infarction, hypoxic brain injury and pulmonary haemorrhage. A small number of patients may die from causes such as septic shock and cardiac arrest[5].

Differentiating NPPE

Clinically, it is difficult to diagnose NPPE in patients with mild symptoms such as mild respiratory distress, a mild decrease in oxygen saturation, a mild cough or cough with frothy sputum or mild chest discomfort. The clinical presentation may mimics aspiration pneumonia during anaesthesia (Mendelson's syndrome), cute respiratory distress syndrome (ARDS), neurogenic hypertension, and anaphylactic reaction and other causes of pulmonary oedema, including cardiogenic pulmonary oedema and iatrogenic volume overload and there for need high level of suspicion.

The radiographic findings may be useful in differentiating NPPE from cardiogenic pulmonary oedema. NPPE often demonstrates marked bilateral perihilar alveolar infiltrates, while in cardiogenic pulmonary oedema the infiltrates follow a more interstitial pattern and marked diversion of blood flow to the lung apices is usually seen[2].NPPE itself can promote cardiac depression in consequence to hypoxia and the subsequent acidotic state, which may make the diagnostic workup misleading[10].

This case was totally health patient except the adenotonsilar hyperplasia. There was no perioperative aspiration incident or preexisting cardiac and pulmonary problems. We also used chest X -ray as differentials and the case was typically NPPE. NPPE should be differentiated from the above diseases, as their treatment differs from that of NPPE.

Management of NPPE

Management of NPPE will depend on the severity of pulmonary oedema and associated hypoxia[4]. Most cases resolve spontaneously within a day and require only supportive therapy such as oxygen by facemask. This case was also recovered spontaneously within a day with noninvasive ventilation support, facemask. Generally the basis of the NPPE treatment is to maintain upper airway patency and avoid further damage to the lung.

Unless the case is critical, invasive ventilator treatments may not require. Close observation with oxygen support in ICU is recommended until the condition is stable. If the case is sever endotracheal intubation and putting on mechanical ventilation is required. A primitive case series report done by Lang *et al*.[14] showed that 85% of adult patients and children required intubation, 50% required mechanical ventilation and 50% required continuous positive end-expiratory pressure (PEEP) ventilation. Most patients treated with mechanical ventilation were able to resolve pulmonary oedema and extubation within 24 h.

Setting PEEP of 5–10 cmH2O may be required this could improve oxygenation and reduce the required oxygen concentration. Positive-pressure ventilation (PPV) exerts a beneficial effect in oedema resolution, as positive pressure helps to normalize the pulmonary hydrostatic pressures, contributes to the reabsorption of interstitial fluid. The oropharynx should be sucked if secretion is there to avoid aspiration[13]. Restriction of intravenous crystalloids is required to avoid further shifting and exacerbating the oedema[15]. In this case fluid was restricted starting from the diagnosis until oedema resolved. There was no any hemodynamic disturbance associated with the discontinuing of the maintenance fluid.

From a pharmacological standpoint, muscle relaxation with low doses of succinylcholine (0.1–0.2 $mg.kg^{-1}$) is able to relieve laryngospasm and/or tracheal tube biting, in addition to facilitate airway manoeuvre, such as PPV via facemask[4]. The use of diuretics is controversial[1,3,4]. Against evidence reasoned as "it can cause hypovolemic and hypotension in postsurgical patients and mainly oedema during NPPE is not due to fluid overload and using diuretics in hemodynamic ally unstable patients is risky". The supporters reasoned by there is an increase in adrenergic activity during obstruction that may cause hypertension and hypervolemia. some clinician use it despite inconclusive literature[16] and the last edition of Barash also indicates to use it in some cases[17]. For this case 5 mg furosemide was administer because standard anaesthesia books such as Barash recommended to use it and the teams were the supportive of the principles. Corticosteroids showed no benefits in NPPE, whereas beta 2-agonists may be useful, as they aid in the transport of Trans membrane ions, facilitating the clearance of the pulmonary interstitial fluid[4].

Observation and monitoring of patients at risk for NPPE (post-laryngospasm, post-upper airway obstruction) is recommended for 2–12 h at PACU or ICU. NPPE resolution occurs from 24 to 48 h in most cases, usually without the need for additional therapies or prolonged hospital stay[3].

Prognosis

Observation and monitoring of patients at risk for NPPE (post-laryngospasm, post-upper airway obstruction) is recommended for 2–12 h at PACU or ICU. NPPE resolution occurs from 24 to 48 h in most cases, usually without the need for additional therapies or prolonged hospital stay[3]. According to Bhaskar, "NPPE resolves generally within 24 h. However, when recognition is delayed, patients with NPPE have mortality rates ranging from 11 to 40%[4].

The mortality rate of NPPE has previously been described as 11–40%, with a more recent literature review showing a mortality rate of only 2% (15–25). A recent systematic review of NPPE in adult ear, nose and throat (ENT) surgery reported a mortality rate of 5% and identified age and ICU admission as the main risks for increased mortality[3]. Due to the high incidence and misdiagnosis rate of NPPE, further improvements in the level of diagnosis, treatment and management of NPPE are expected in clinical practice. The present review aims to summarize the latest advances in the epidemiology, pathophysiology, clinical manifestations and treatment of NPPE.

Prevention

Currently there is no reliable method for predicting which children will experience this clinical syndrome after their airway obstruction has been resolved[17]. However, to decrease the risk, the anaesthesia professional should take measures to avoid laryngospasm by assuring adequate anaesthesia during mask ventilation or deep extubation, or ensure a patient is fully awake with adequate airway function prior to extubation and allowing positive leak around the ETT at less than 15–20 cm of water. This patient was extubated in deep anaesthesia with no changes in heart rate or cough with oropharynx suctioning. His airway was clear after extubation and he did not exhibit laryngospasm. Administering 1–2 mg/kg of lidocaine 5 min before tracheal extubation or 0.5 mg/kg propofol 60 sec before extubation may decrease the incidence of spasm so prevent NPPE. After multiple intubation attempts giving 5 mg of dexamethasone before extubation may be helpful to reduce laryngeal oedema caused by multiple[18,19]. In addition, the cuff leak test could help prevent the risk of post-extubation oedema. The cuff leak test is based on the principle that air leaks around the tracheal tube where the cuff is deflated will be inversely proportional to the degree of laryngeal obstruction resulting from laryngeal oedema. Extubation may be successful if air leaks can be heard when the patient coughs during PEEP[20].

Conclusion

Post-extubation pulmonary oedema continues to occur after laryngospasm or reliving of chronic air obstruction. It usually develops in healthy young patients who may not have any risk factors for its development. Although NPPE occur rapidly after obstruction, it may delay hours and may cause unrecognized death. Prolonged observation may needed for risky patients.

Recommendation

NPPE can occur at any time in any patient particularly patients with obstructive upper airway disease. Therefore any healthcare professionals must be knowledgeable about clinical presentation and managements of NPPE.

Ethical approval

After informing the benefits and risk of the procedure and anaesthesia, the patient was volunteered to take anaesthesia and signed on the hospital consent form to be operated. Since this is a case report which is we decided to report after the procedure done, we have no formal ethics approval and consent to participate that should be reviewed by ethical committees.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal.

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There was no any source of funding.

Author contribution

A.B.W.: draft the concept or design of the study, collect, analysis or interpretation the data , writing the prepare and write the case report. S.T.N.: collect data, review and edit and the manuscript. F.M.: collect data, review and edit and the manuscript.

Conflicts of interest disclosure

There was no any conflict of interest.

Research registration unique identifying number (UIN)

Registration site: Clinical Trial.gov.
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Guarantor

Aynalem Befkadu Woldemichael, the PI and corresponding author.

Availability of supporting data

“Not applicable” because there is no supporting data left since all data are shared.

Provenance and peer review

Not invited.

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References

[1] Fremont RD, Kallet RH, Matthay MA, *et al*. Postobstructive pulmonary edema: a case for hydrostatic mechanisms. Chest 2007;131:1742–6.
[2] Oswalt CE, Gates GA, Holmstrom FM. Pulmonary edema as a complication of acute airway obstruction. JAMA 1977;238:1833–5.
[3] Bhaskar B, Fraser JF. Negative pressure pulmonary edema revisited: Pathophysiology and review of management. Saudi J Anaesth 2011;5:308.
[4] Bhattacharya M, Kallet RH, Ware LB, *et al*. Negative-pressure pulmonary edema. Chest 2016;150:927–33.
[5] Din-Lovinescu C, Trivedi U, Zhang K, *et al*. Systematic review of negative pressure pulmonary edema in otolaryngology procedures. Ann Otol Rhinol Laryngol 2021;130:245–53.
[6] Sohrabi C, Mathew G, Maria N, *et al*. The SCARE 2023 guideline: updating consensus Surgical CAse REport (SCARE) guidelines. Int J Surg 2023;109:1136–40.
[7] McConkey P. Postobstructive pulmonary oedema—a case series and review. Anaesth Intensive Care 2000;28:72–6.
[8] Kluger M, Visvanathan T, Myburgh J, *et al*. Crisis management during anaesthesia: regurgitation, vomiting, and aspiration. Quality Safety Health Care 2005;14:e4.
[9] Ma J, Liu T, Wang Q, *et al*. Negative pressure pulmonary edema. Exp Ther Med 2023;26:1–7.
[10] Ezri T, Roth Y. Don’t overlook the complications of tonsillectomy. J Fam Pract 2010;59:E4–E9.
[11] Rosero-Britton B, Uribe A, Stoicea N, *et al*. Negative pressure pulmonary edema postextubation following medial nerve repair with sural graft surgery in a young patient: a case report. Medicine 2018;97:e13743.

[12] Schwartz AJ, Davidson S, Guinn C, *et al*. Diagnosis and treatment of negative pressure pulmonary edema in a pediatric patient/response. AANA J 2005;73:17.
[13] LAe al. Negative Pressure Pulmonary Edema, Robotic-Assisted Cystoprostatectomy, General Anesthesia in the Parturient, Acute Intermittent Porphyria, Accidental Dural Puncture, Supraorbital Hematoma Cri du Chat Syndrome, Mitral Valve Prolapse, Myasthenia Gravis, ACE inhibitor, TIVA for MH. Int Student J Nurse Anesth Summer 2015;14:1–5.
[14] Lang SA, Duncan PG, Shephard DA, *et al*. Pulmonary oedema associated with airway obstruction. Can J Anaesth 1990;37:210–8.
[15] Mehta Vishvesh M, MG H-E, Goldstein Nira A. Postobstructive Pulmonary Edema After Laryngospasm in the Otolaryngology Patient The American Laryngological. Rhinological and Otological Society, Inc; 2006.
[16] More Preeti G, Durve Shubhada R. Acute pulmonary oedema, a post oprerative complications due to neostigmin and post obstractive pulmonary oedema in case of tonsillectomy jornals of clinical and diagnostic research: JCDR 2015;9:UD05–6.
[17] Paul G, Barash BFC, Robert K, *et al*. Anesthesia for otolaryngologic surgery. Clin Anaesth (Barash), Eighth edition. 2017.
[18] Lee C-H, Peng M-J, Wu C-L. Dexamethasone to prevent postextubation airway obstruction in adults: a prospective, randomized, double-blind, placebo-controlled study. Crit Care 2007;11:1–8.
[19] Kuriyama A, Umakoshi N, Sun R. Prophylactic corticosteroids for prevention of postextubation stridor and reintubation in adults: a systematic review and meta-analysis. Chest 2017;151:10020.
[20] Huang Y-C, Lin H-Y, Huang T-T, *et al*. Negative pressure pulmonary edema after vocal augmentation. J Formosan Med Assoc Taiwan yi zhi 2019;118:1166–7.