OLGU SUNUMU

EARLY POSTOPERATIVE COMPLICATION CALLED NEGATIVE PRESSURE PULMONARY EDEMA SEEN IN A YOUNG ATHLETIC MALE

Esra MERCANOĞLU (EM), Cenk İLHAM (Cİ), Derya YURTLU (DY)
Ahmet BAŞEL (AB), Erol SAĞATLI (ES)

(EM, Cİ, DY, AB) Zonguldak Atatürk Devlet Hastanesi, Anesteziyoloji ve Reanimasyon Kliniği
(ES) Zonguldak Atatürk Devlet Hastanesi, Kardiyoloji Kliniği

ABSTRACT

EARLY POSTOPERATIVE COMPLICATION CALLED NEGATIVE PRESSURE PULMONARY EDEMA SEEN IN A YOUNG ATHLETIC MALE: A CASE REPORT

Postobstructive pulmonary edema is a well-recognized complication of upper airway obstruction. Negative pressure pulmonary edema (NPPE) is a potentially life-threatening complication especially during emergence in patients undergoing general anesthesia. Laryngospasm-mediated upper airway obstruction leads to forced inspiration generating excessive negative intrathoracic pressure and causes negative pressure pulmonary edema. In this paper we report a young athletic man who developed NPPE immediately after operation. This paper aims to not only to report the diagnose and treatment of NPPE but also to remind the importance of prevention NPPE.

KEYWORDS: *Pulmonary Edema; Postoperative Complication.*

ÖZET

GENÇ ATLETİK ERKEKTE GELİŞEN NEGATİF BASINÇ PULMONER ÖDEM: OLGU SUNUMU

Postobstrüktif pulmoner ödem, üst havayolu obstrüksiyonun iyi bilinen bir komplikasyonudur. Negatif basınç pulmoner ödem (NBPÖ) genel anestezi alan hastalarda özellikle uyanma sırasında görülen hayatı tehdit eden bir komplikasyondur. Laringospazm kaynaklı üst hava yolu obstrüksiyonu aşırı negatif intratorasik basınç oluşturan zorlu inspirasyona yol açar ve negatif basınç pulmoner ödeme neden olur. Bu olguda operasyondan hemen sonra NBPÖ gelişen genç atletik bir erkek hastayı sunmayı amaçladık. Bu olgu NBPÖ tanı ve tedavisinin yanında ayrıca NBPÖ'den korunmanın önemini de hatırlatmaktadır.

ANAHTAR KELİMELER: *Pulmoner Ödem, Postopertatif Komplikasyon.*

INTRODUCTION

Negative pressure pulmonary edema (NPPE) is a rare but well known complication which is seen secondary to upper airway obstruction (1). Negative pressure pulmonary edema is one of the form of noncardiogenic pulmonary edema that can occur following any general anesthesia especially after extubation. This complication can be severe, even fatal if not diagnosed and treated promptly (2).

In this article we report a complication called NPPE which was seen soon after general anesthesia. It also emphasizes the importance, prevention, diagnosis and treatment of NPPE.

CASE

A 21 years old, ASA I, 80 kg man with a history of smoking for 2 years was scheduled for a tibia fracture operation under general anesthesia. Indication of general anesthesia was the refusal of the regional anesthesia by the patient. His prior history was unremarkable and physical examination was normal. No premedication was given. Before induction of anesthesia blood pressure was 110/70 mmHg, heart rate was 76 min^{-1}, oxygen saturation was 98%. Anesthesia was induced with 100 µg fentanyl, 5 mg kg^{-1} sodium thiopenthal and 0.5 mg kg^{-1} rocuronium without any complication. Entubation was achieved without difficulty. Anesthesia was maintained with 6-7 MAC desflurane and 1:1 mixture of O_2 and N_2O. During the 150 minutes surgical procedure the patient had a measured blood loss 300 mL and received 1800 mL intravenous fluids. All hemodynamic measurements were stable during operation. The patient was extubated after reversal of neuromuscular block. Immediately, after extubation the patient developed clinical manifestations marked upper airway obstruction and respiratory distress like dyspnea, tachypnea and coughing and the oxygen saturation determined by means of pulse

oximetry had fallen from 99% to 85%. Bronchospasm signs were audible. Eighty mg prednisolone and 120 mg aminophylline were administered slowly intravenously and 120 mg aminophylline was infused slowly. For sedation 3 mg midazolam was administered and although he was ventilated with positive pressure with 100% oxygen, oxygen saturation had fallen more down to 40%. Then the patient reintubated with 150 mg propofol and 30 mg rocuronium. A pink frothy edema fluid was aspirated from endotracheal tube, and diffuse rales were audible. Oxygen saturation was increased up to 85%, blood pressure was 135/75 mmHg and then 20 mg furosemide were administered. Urine output was 800 mL. Oxygen saturation was increased up to 90% in 10 minutes. After patient was taken to ICU (approximately 30 minutes after extubation) a bedside chest x-ray showed us pulmonary edema signs; above hilus like bilateral centralized interstitial pulmonary edema, wide vascular pedicle and normal heart size (Figure 1). After the patient was ventilated mechanically for two hours (with SIMV, volume controlled mode, preset tidal volume 550 mL, frequency 12 min^{-1}, PEEP 5 cm H20 and PSV 10 cm H_20), he was able to tolerate weaning from the ventilator and underwent extubation. Very soon after extubation stridor was audible and he was breathing with difficulty. 1.5 mg kg^{-1} lidocaine was administered for relieving laryngospasm and in a few minutes after lidocaine, his respiratory pattern turned to normal without any pathologic sound. Clinical examinations and chest radiograph were normal in a few hours (Figure 2). On the first day after operation he was discharged from the ICU to surgery ward. The remainder of the hospital course was uneventful.

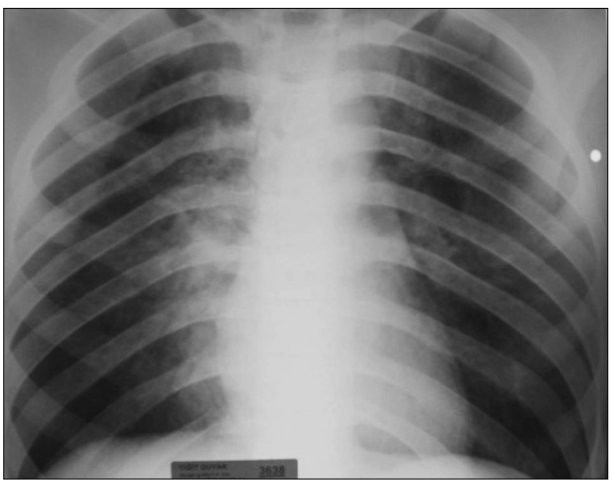

Figure 1: Chest x-ray taken approximately 30 minutes after extubation

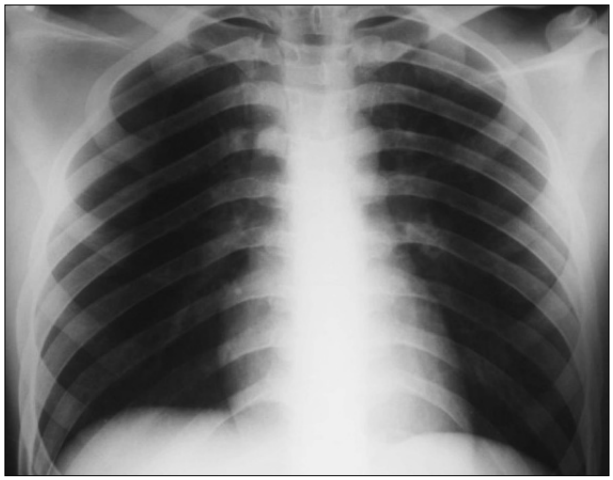

Figure 2: Chest x-ray taken in a few hours after extubation

DISCUSSION

Postobstructive pulmonary edema is an uncommon, but well-described, complication of upper airway obstruction especially during emergence from anesthesia (3,4,5). In our case NPPE was seen immediately after extubation, as it is known generally.

The incidence of NPPE has been reported to be 0.05 to 0.1% of all anesthetic practices however it is estimated that it occurs more commonly than is generally documented (6). In the adults, risk factors include obesity with obstructive sleep apnea, anatomically difficult intubations, the presence of airway lesions, and patients undergoing nasal, oral or pharyngeal surgery. Young male athletes are at risk because of their ability to generate significant negative intrapleural pressures. Pediatric patients also are at risk because of their extremely compliant chest walls that can generate large negative intrapleural pressures (7). In our case there wasn't any risk factor other than our patient was a young and athletic male. Forceful inspiration against a closed glottis generates very high negative intrathoracic pressures. This results in a clinical picture of pulmonary edema and rarely hemorrhage associated with edema (8). The most common cause of postobstructive pulmonary edema is laryngospasm during extubation or after anesthesia in the postoperative period. Laryngospasm has been reported to be the cause in > 50% of cases of postobstructive pulmonary edema (9). In our case, secretion and laryngospasm caused the obstruction and his increased muscle mass created very high intrathoracic pressures that made him prone to NPPE.

The symptoms of NPPE usually develops immediately after extubation although sometimes the onset may

be considerably delayed up to a few hours in the postoperative period. A possible explanation for this delayed manifestation is a positive pressure, created by forceful expiration against a closed glottis, opposing fluid transudation (5,10). As airway obstruction relieves, increased venous return causes blood shift from peripheral to central circulation and hydrostatic transudation. Thus close postoperative observation must be continued for an extended time in patients experiencing respiratory difficulty (11).

Signs and symptoms of respiratory distress are often present, but frothy, pink sputum is the hallmark sign of NPPE. Auscultation reveals rales and, occasionally, wheezes from fluid-compressed airways. The chest radiograph typically shows diffuse interstitial and alveolar infiltrates appearing as "whited out" areas. Tachycardia, hypertension, and diaphoresis reflect sympathetic system stimulation (12). When clinical signs and symptoms present, the anesthesia provider must form a differential diagnosis. Differential diagnoses for consideration include acute respiratory distress syndrome, intravascular volume excess, cardiac abnormalities, and pulmonary embolus (7). After the diagnosis of NPPE has been made, treatment is directed toward reversing hypoxia and decreasing the fluid volume in the lungs. Maintaining the airway and providing supplemental oxygen is usually all that is required for a positive outcome. If oxygenation does not improve in the intubated patient, positive end-expiratory pressure (PEEP) should be administered to promote alveolar expansion. If oxygenation does not improve in the nonintubated patient, then immediate intubation with positive pressure ventilation and positive end-expiratory pressure is necessary (11). All these treatment approaches were the same in our case. The use of diuretic therapy (eg, furosemide, 1 mg kg^{-1}) to remove excess intrapulmonary fluid is controversial. It is possible for the patient to be hypovolemic, and consequently diuretic therapy would only worsen the clinical condition. In our case the administrated diuretic therapy did not worsen the clinical condition. NPPE is a rapidly reversible condition with relatively simple management.

Specifically the use of tracheal anesthesia, intravenous lidocaine prior to extubation, the use of lidocaine in the endotracheal cuff are current strategies employed to prevent laryngospasm after extubation (11). Unfortunately in our case these precautions were missed. In management of NPPE relieving the obstruction and correcting hypoxemia are the first aims and some of these cases are reintubated like our case (1). Besides subsequent removal of the obstructive event, providing a patent airway and adequate oxygen saturation were the first steps in the initial management of our case. Although symptoms usually resolve with restoration and maintenance of a patent airway and supplemental O_2, they may sometimes progress to adult respiratory distress syndrome and result in death (13). Early recognition and appropriate positive pressure breathing respiratory therapies like in our case may prevent these potentially deletorious iatrogenic complications.

Patients in whom postobstructive pulmonary edema develops generally have an uncomplicated hospital course followed by the rapid resolution of the pulmonary edema and short hospital stays (2,10,14). The patients in our study had similarly uncomplicated courses with an average ICU length of stay of 1 day. As it is seen from the case he was extubated on the day of pulmonary edema developed and was subsequently discharged from ICU without any prolonged pulmonary sequelae. His unremarkable hospital course makes a diagnose of a significant acute lung injury unlikely. After this case, we reached out two main results. First; it is very important to prevent from NPPE. For this purpose, lidocaine could be administered 3-5 minutes before extubation for preventing laryngospasm to prevent from obstruction and atropine could be administered to reduce secretions. Second; if NPPE is seen despite of all these precautions it has to be diagnosed and treated without wasting time. Unfortunately atropine as a premedication and 1.5 mg kg-1 lidocaine before extubation were not administered in our case but NPPE was managed without wasting time. We were really successful with our treatment. In conclusion NPPE, if anticipated and recognized early, will be a self-limited condition with excellent prognosis and simple management very quickly otherwise it may have serious complications. However both surgeons and clinicians must be provided with an understanding that any patient who is otherwise well has the potential for NPPE, which is of anesthesiologic relevance, even late in the postoperative period. As a result, good prevention and rapid diagnosis and treatment is necessary to achieve early resolution from NPPE and avoid significant patient morbidity and mortality.

Yazışma Adresi: Dr. Esra MERCANOĞLU
Zonguldak Atatürk Devlet Hastanesi
Anesteziyoloji ve Reanimasyon Kliniği
Meşrutiyet Mahallesi Huzur Sokak No:35
Zonguldak
Tel: 0 372 252 19 00
e-posta: esramercan76@hotmail.com

REFERENCES

1. Fistikci H, Cakar N, Kirisoglu C, Kara O, Polat S. Non-invasive Possitive Pressure Ventilation in Treatment of Negative Pressure Pulmonary Edema and Alveolar Hemorrhage. Turk Anest Rean Der 2008; 36: 178-81.
2. Tarrac SE. Negative pressure pulmonary edema-a postanesthesia emergency. J Perianesth Nurs 2003; 18: 317-23.
3. Koh MS, Hsu AA, Eng P. Negative pressure pulmonary oedema in the medical intensive care unit. Intensive Care Med 2003; 29: 1601-4.
4. Mehta VM, Har-El G, Goldstein NA. Postobstuctive pulmonary edema after laryngospasm in the otolaryngology patient. Laryngoscope 2006; 116: 1693-6.
5. Van Kooy MA, Gargiulo RF. Postobstructive pulmonary edema. Am Fam Physician 2000; 62: 401-4.
6. McConkey PP. Postobstructive pulmonary oedema-a case series and review. Anaesth Intensive Care 2000; 28: 72-6.
7. Sulek C. Negative-pressure pulmonary edema. In: Gravenstein N, Kirby RR. Complications in Anesthesiology. 2nd ed. Philadelphia, Pa: JB Lippincott; 1996: 191-7.
8. Sow Nam Y, Garewal D. Pulmonary hemorrhage in association with negative pressure edema in an intubated patient. Acta Anaesthesiol Scand 2001; 45: 911-3.
9. Louis PJ, Fernandes R. Negative pressure pulmonary edema. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002; 93: 4-6.
10. Westreich R, Sampson I, Shaari CM, Lawson W. Negative-pressure pulmonary edema after routine septorhinoplasty: discussion of pathophysiology, treatment, and prevention. Arch Facial Plast Surg 2006; 8: 8-15.
11. Kesimci E, Aslan B, Gümüs T, Kanbak O. Management of unpredicted postoperative negative pressure pulmonary edema: a report of two cases. The Internet Journal of Anesthesiology ISSN: 1092-406X 07 Sep 09
12. Thomas CL, Palmer TJ, Shipley P. Negative pressure pulmonary edema after a tonsillectomy and adenoidectomy in a pediatric patient: Case report and review. AANA J 1998; 67: 425-30.
13. Ackland GL, Mythen MG. Negative pressure pulmonary edema as an unsuspected imitator of acute lung injury/ARDS. Chest 2004; 125:1530-5.
14. Devys JM, Balleau C, Jayr C, et al. Biting the laryngeal mask: an unusual cause of negative pressure pulmonary edema. Can J Anaesth 2000; 47: 176-8.

DÜZELTME

Anestezi Dergisi 2009; 17 (3): 165-167 sayısında yayınlanmış olan **"GÖZ CERRAHİSİ SONRASI BEKLENMEYEN POSTOPERATİF DELİRYUM"** başlıklı olgu sunumunun, bu sayının içindekiler bölümünde yer alan yazar isimleri için yapılan düzeltme aşağıdaki gibidir.

Göz Cerrahisi Sonrası Beklenmeyen Postoperatif Deliryum (Olgu Sunumu)
Unexpecded Postoperative Delirium Following Eye Surgery (A Case Report)
Banu BARAN, Hale BORAZAN, Alper YOSUNKAYA

Düzeltilmiş

Göz Cerrahisi Sonrası Beklenmeyen Postoperatif Deliryum (Olgu Sunumu)
Unexpecded Postoperative Delirium Following Eye Surgery (A Case Report)
Şennur UZUN, Didem DAL, Aysun ANKAY YILBAŞ