Kastamonu Sağlık Akademisi

Olgu Sunumu / Case Report

Negative Pressure Pulmonary Edema Due To Endotracheal Tube Bite In A Patient Who Could Not Be Placed Guedel Orophaeryngeal Airway Before Extubation/ *Ekstübasyon Öncesinde Orofarengeal Guedel Airway Yerleştirilememesine Bağlı Endotrakeal Tüp Isırılması Sonucu Gelişen Negatif Basınç Pulmoner Ödemi*

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Gönderim Tarihi | Received: 26.08.2019, Kabul Tarihi | Accepted: 07.09.2019, Yayım Tarihi | Date of Issue: 01.08.2021, DOİ: 10.25279/sak.610933

Atıf | Reference: "ÇARDAKÖZÜ, T; SEREZ, BY. (2021). Negative Pressure Pulmonary Edema Due To Endotracheal Tube Bite In A Patient Who Could Not Be Placed Guedel Orophaeryngeal Airway Before Extubation. *Sağlık Akademisi Kastamonu (SAK),* 6 (2), s.138-144."

Öz

Amaç: Akut negatif basınç pulmoner ödemi genel anestezi alan hastalarda ekstübasyondan kısa bir süre sonra ortaya çıkan bir komplikasyondur. Ayrıca ekstübasyondan önce endotrakeal tüpün ısırılmasına bağlı olarak da oluşabilir. Olgu sunumu: 52 yaşında erkek hastaya ventrikülоperitoneal şant operasyonu planlandı. Genel anestezi uygulandı. Solunum ve hemodinamik değişkenler ameliyat süresince stabil seyretti. Ameliyat sonunda anestezik ilaçlar kesildi, endotrakeal tüpün lümeni ve orofarenks aspire edildi. Hastaya orofarengeal airway yerleştirileceği esnada hasta endotrakeal tüpünü ısırıp tıkadı ve güçlü bir şekilde solunum eforu yapmaya başladı. Hızlıca desatüre oldu ve endotrakeal tüp içinden pembe köpüklü sekresyon geldi. Bilateral difüz krepitan raller mevcuttu. Akciğer grafisinde bilateral pulmoner ödem saptandı. Hasta yoğun bakım ünitesine transfer edildi, sedasyon uygulandı ve volum kontrollü pozitif basınçlı mekanik ventilasyon başlandı. Hasta ameliyat sonrası 12. saatte ekstübe edildi ve üçüncü gün servise gönderildi. Tartışma: Ekstübasyon planı iyi yapılmalıdır. Aspirasyon ve ekstübasyon, derin anestezi altında veya hasta tamamen uyanık olduğunda uygulanmalıdır. Orofarengeal airway ekstübasyondan önce, yeterli anestezi derinliği altında yerleştirilmelidir. Orofarengeal airway endotrakeal tüpün ısırılmasını ve buna bağlı gelişebilecek negatif basınç pulmoner ödemi riskini azaltsa da tamamen önleyemeyebilir. Negatif basınç pulmoner ödemi tedavisinde, obstrüksiyonun ciddiyetine ve hipoksinin derecesine bağlı olarak invaziv veya non-invaziv mekanik ventilasyon tercih edilebilir. Negatif basınç pulmoner ödemi'nin erken teşhisi ve tedavisi hayat kurtarıcıdır.

Anahtar Sözcükler: Negatif Basınç Pulmoner Ödemi, Endotrakeal Tüpün Isırılması, Isırma Bloğu

Abstract

Background: Acute negative pressure pulmonary edema is a complication that usually occurs shortly after extubation in patients receiving general anesthesia. It may also occur due to the bite of the endotracheal tube prior extubation. Case presentation: A 52-year-old male patient was scheduled for ventriculoperitoneal shunt operation. General anesthesia was applied. Respiratory and hemodynamic variables were stable during surgery. At the end of the surgery, anesthetic drugs were discontinued, the lumen of the endotracheal tube and oropharynx were aspirated. When oropharyngeal airway was placed the patient bit and occluded his endotracheal tube, and began exerting breathing effort. Rapid desaturation was observed and pink foamy secretion came through the endotracheal tube. Bilateral diffuse crackles were present. A chest X-ray revealed bilateral pulmonary edema. The patient was transferred to the intensive care unit, sedation was applied and volume controlled positive pressure mechanical ventilation was started. The patient was extubated at the 12th postoperative hour and sent to the ward on the third postoperative day. Discussion: The extubation plan should be done well. Aspiration and extubation should be performed either under deep anesthesia or when the patient is fully awake. Oropharyngeal airway should be placed under adequate depth of anesthesia before extubation. Although oropharyngeal airway reduces the risk of biting of the endotracheal tube and subsequent development of negative pressure pulmonary edema, it may not prevent it completely. In negative pressure pulmonary edema treatment, invasive or non-invasive mechanical ventilation may be preferred depending on the severity of obstruction and degree of hypoxia. Early diagnosis and treatment of negative pressure pulmonary edema is life-saving.

Keywords: Negative Pressure Pulmonary Edema, Biting The Endotracheal Tube, Bite Block

Introduction

Negative pressure pulmonary edema (NPPE) is a condition that may develop after acute obstruction of the upper respiratory tract or secondary to the removal of chronic obstruction (Oswalt, Gates & Holstrom, 1977, p. 1833-1835). Although rare, it is a serious complication. If not diagnosed, mortality of NPPE can reach up to 40% (Saraswat, Madhu & Kumar, 2007, p. 42). In this case report, endotracheal tube (ETT) occlusion due to delay in Guedel oropharyngeal airway (OPA) placement and consequent development of NPPE in a patient who underwent ventriculo-peritoneal (VP) shunt operation under general anesthesia will be presented.

Case Report

A 52-year-old male patient with the American Society of Anesthesiologists (ASA) II (Behçet's Disease) was scheduled for VP shunt operation due to normal pressure hydrocephalus. After anesthesia induction with fentanyl 1 μg/kg, thiopental 5 mg/kg and rocuronium bromide 0.8 mg/kg, he was orally intubated with 8.0 cuff ETT. Mechanical ventilation was initiated with a tidal volume (TV) of 600 ml, respiratory rate (RR) of 10/min and a 1:2 I:E ratio.

Anesthesia was maintained with fresh gas flow of 3 L/min containing 1 MAC sevoflurane, with oxygen and air mixture. During the operation RR, was adjustedto achieve a $ETCO_2$ between 30-35mmHg. The operation lasted 60 minutes. During surgery 450 mL of 0.9% NaCl was infused and theurine output was 150 ml. Oxygen saturation, airway pressures, $ETCO_2$ values, heart rate (HR) and blood pressure (BP) of the patient were within normal limits in the intraoperative period. At the end of the surgery, anesthetic agents were discontinued, and mouth cavity and ETT lumen were aspirated. Neuromuscular block was reversed. At the time of size 4 of Guedel OPA placement, the patient bit the ETT and he was exhibiting a strong respiratory effort. Simultaneusly, the patient began to desaturate. By auscultation, bilateral diffuse crackles were present. A systolic BP between 110-130 mmHg, diastolic BP between 60-90 mmHg and HR between 95-110beat/min was present. 200 mg propofol was administered to the patient and Guedel OPA was placed. With manual ventilation, peripheric oxygen saturation (SPO_2) was increased to 75% with 100% oxygen, while $ETCO_2$ was 33-38 mmHg. In the arterial blood gas (ABG) analysis, the values were as follows:pH:7.34, PaO_2:57.5 mmHg, $PaCO_2$:36.6 mmHg, HCO_3:20.9 mEq, BE:-4.2, and SaO_2:80 percent. When the ETT was aspirated again, pink foamy secretion came through. Chest X-ray was taken in the operation room and images of bilateral pulmonary edema were obtained (Figure 1). Urinary catheter was inserted and intravenous furosemide (20 mg) was administered. The patient was transferred to the intensive care unit, sedation was applied and volume controlled positive pressure mechanical ventilation was started in synchronised intermittent mandatory ventilation (SIMV) mode with FiO_2: 100%, TV:600 ml, RR:10/min, I:E ratio:1:2 and PEEP:10cmH_2O. FiO_2 was gradually reduced with ABG monitoring. Furosemide and steroid treatment was initiated. With the improvement of oxygenation, sedation was discontinued at the 8^{th} postoperative hour, continuous positive airway pressure (CPAP) mode was started, and the patient was extubated at the 12^{th} postoperative hour. With 5L/min O_2 through mask, ABG values were as follows:pH:45, PaO_2:67.5 mmHg, $PaCO_2$:30.6 mmHg, HCO_3:22.9 mEq, BE: -1.2, and SpO_2: 94 percent. Chest X-ray returned to normal (Figure 2). The patient was sent to the ward on the third postoperative day. The patient was discharged on the fifth postoperative day without any respiratory distress.

Discussion

In this case report, attention was drawn to NPPE which developed as a result of biting the ETT by the patient in whom Guedel OPA could not be inserted prior to extubation. Pink foamy sputum, rapid onset and rapid recovery after airway obstruction are characteristics of NPPE (Krodel, Bittner, Abdulnour, Brown & Eikermann, 2010, p. 200-207). In this patient who developed acute hypoxia, other causes of acute pulmonary edema that should be considered apart from NPPE are [cardiogenic-noncardiogenic (excessive fluid overload, anaphylaxy-related, neurogenic lung edema)] and iatrogenic pneumothorax or tension pneumothorax that may occur rarely in VP shunt operations (Gümüş et.al., 2012, p. 59-62 ; Kono, Tomura, Okada & Terada, 2014, p. 123-126).

Pneumothorax or tension pneumothorax were excluded due to bilateral equal lung sounds, unchanged airway pressure, lack of sudden drop or increase in $ETCO_2$, and absence of accompanying hemodynamic symptoms. Cardiogenic pulmonary edema was also excluded because there was no previous cardiac problem and patient was hemodynamically stable. Volume overload and anaphylactic pulmonary edema were not considered in the differential diagnosis, because they were not clinically compatible. One of the non-cardiogenic causes to be considered in this patient was neurogenic pulmonary edema (NPE). NPE is usually related with subarachnoid hemorrhage, epileptic seizures and head trauma, its occurrence during VP shunt operation has also been reported (Gümüş et.al., 2012, p. 59-62; Davidyuk, Soriano, Goumnerova & Mizrahi-Arnaud, 2010, p. 594-595). Our patient did not have cranial parenchymal damage or increased intracranial pressure, and there was no evidence suggesting hemodynamic or clinical sympathetic activation (i.e. fever, tachycardia, hypertension), and therefore, the diagnosis of NPE was excluded.

In NPPE treatment, invasive or non-invasive mechanical ventilation may be preferred depending on the severity of obstruction and degree of hypoxia. It has been reported that steroid-derived drugs should be used in the treatment of alveolar damage, and that thereby systemic side effects are avoided, respiratory distress is decreased, and recovery is accelerated. Although the place of loop diuretics in treatment is uncertain, it is thought that they would contribute to the elimination of fluid in the alveoli (Chuang, Wang & Lin, 2007, p. 1113-1116).

Acute NPPE is a complication that usually occurs in patients receiving general anesthesia shortly after extubation (Zhurda et al., 2016, p. 2). It may also occur due to the patient's locked jaw biting the ETT prior to extubation (Dicpinigaitis & Mehta, 1995, p. 1048-1050; Liu &Yih, 1999, p. 174-175). According to Liu et al. (Liu & Yih, 1999, p. 174-175), if an OPA with reinforced bite block is routinely placed after endotracheal intubation, the risk of tube occlusion and NPPE can be avoided. According to some other authors, the use and recommendation of Guedel airway as a bite block may be risky (Kumar, Mullick & Prakash, 2015; King & Lewis, 1996, p. 729-730).

Hereby a rare cause of NPPE following neurosurgery and its management is presented. Carefull planning of the steps prior to extubation of a patient following general anesthesia is crucial to avoid complications. Aspiration and extubation should be performed either under deep anesthesia or when the patient is fully awake. OPA should be placed under adequate depth of anesthesia before extubation. Although OPA reduces the risk of biting of the ETT and subsequent development of NPPE, it may not prevent it completely. Early diagnosis and treatment of NPPE is life-saving.

References

Chuang, YC., Wang, C.H., & Lin, Y.S. (2007). Negative pressure pulmonary edema: Report of three cases and review of the literature. *Eur Arch Otorhinolaryngol, 264*, 1113-1116. doi: 10.1007/s00405-007-0379-9.

Davidyuk, G., Soriano, S.G., Goumnerova, L., & Mizrahi-Arnaud, A. (2010). Acute intraoperative neurogenic pulmonary edema during endoscopic ventriculoperitoneal shunt revision. *Int Anesth Res Soc, 110*, 594–595.

Dicpinigaitis, P.V., & Mehta, D.C. (1995). Postobstructive pulmonary edema induced by endotracheal tube occlusion. *Intensive Care Med*, *21*, 1048-1050.

Gümüş, F., Sevdi, S.M., Erkalp, K., Ülger, G.Ö., Bostan, G., & Alagöl A. (2012). Nörojenik akciğer ödemi (olgu sunumu). *Journal of the Turkish Society of Intensive Care, 10*, 59-62.

King, H.K., & Lewis, K. (1996). Guedel oropharyngeal airway does not prevent patient biting on the endotracheal tube. *Anaesth Intensive Care, 24*, 729-730.

Kono, K., Tomura, N., Okada, H., & Terada, T. (2014). Iatrogenic pneumothorax after ventriculoperitoneal shunt: an unusual complication and a review of the literature. *Turk. Neurosurg, 24*, 123–126. doi: 10.5137/1019-5149.JTN.7928-13.0.

Krodel, D.J., Bittner, E.A., Abdulnour, R., Brown, R., & Eikermann, M. (2010). Case scenario: acute postoperative negative pressure pulmonary edema. *Anesthesiology, 113,* 200-207. doi: 10.1097/ALN.0b013e3181e32e68.

Kumar, A., Mullick, P., & Prakash, S. (2015). Guedel airway: Not a bite block! *BJA, 115, Issue eLetters Supplement*, doi: 10.1093/bja/el_12771

Liu, E.H., & Yih, P.S. (1999). Negative pressure pulmonary oedema caused by biting and endotracheal tube occlusion-a case for oropharyngeal airways. *Singapore Med J, 40*, 174-175.

Oswalt, C.E., Gates, G.A., & Holstrom, M.G. (1977). Pulmonary edema as a complication of acute airway obstruction. *JAMA, 238,* 1833-1835.

Saraswat, V., Madhu, P.V., & Kumar, S.S. (2007). Rapid onset acute epiglottitis leading to negative pressure pulmonary edema. *Indian J Anaesth, 51*, 429-431.

Zhurda, T., Muzha, D., Dautaj, B., Kurti, B., Marku, F., Jaho, E., & Sula,V.E. (2016). Acute postoperative negative pressure pulmonary edema as complication of acute airway obstruction: case report. *J Anesth Clin Res, 7*(2). doi: 10.4172/2155-6148.1000603

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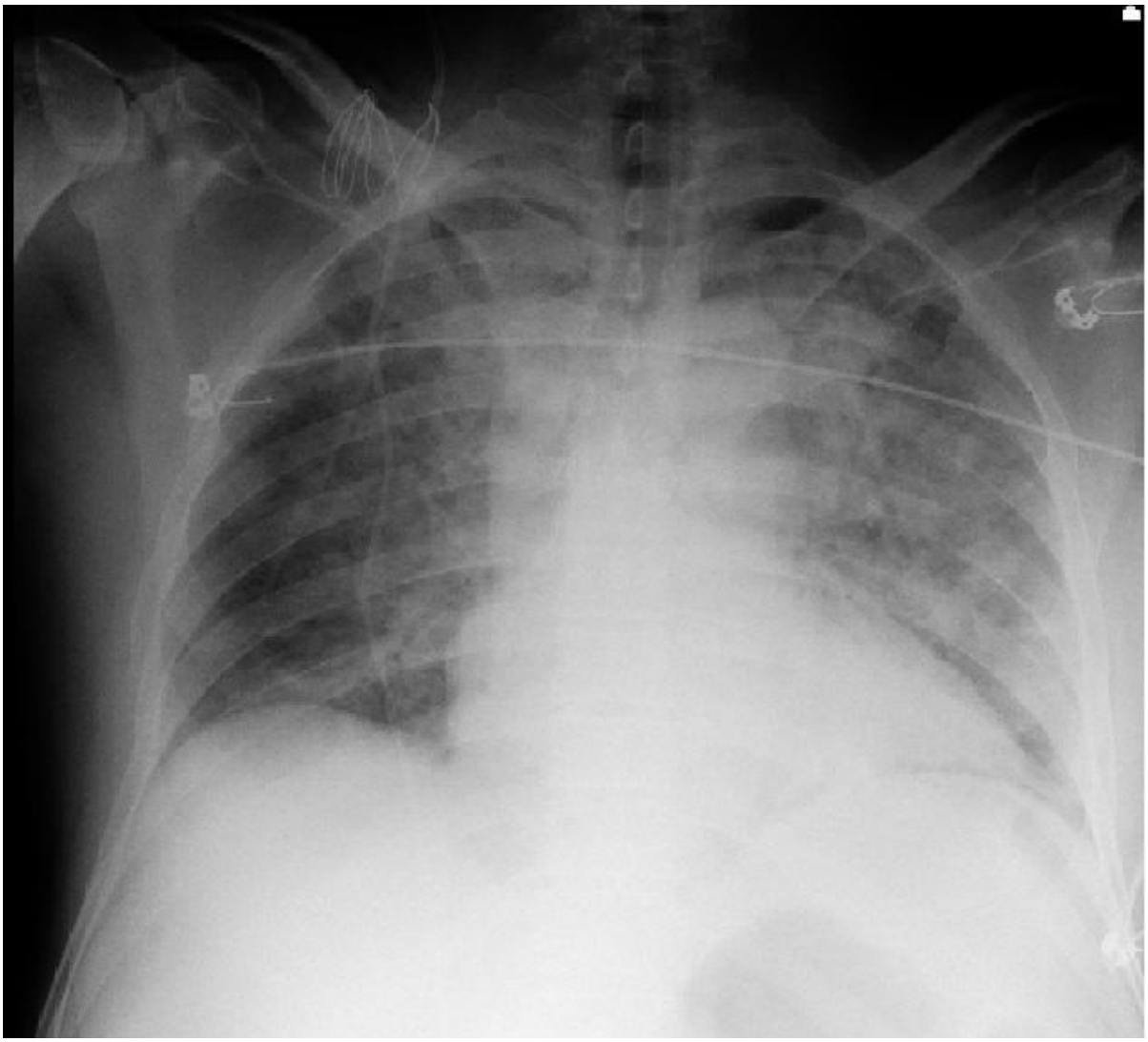

Figure 1: Lung edema image

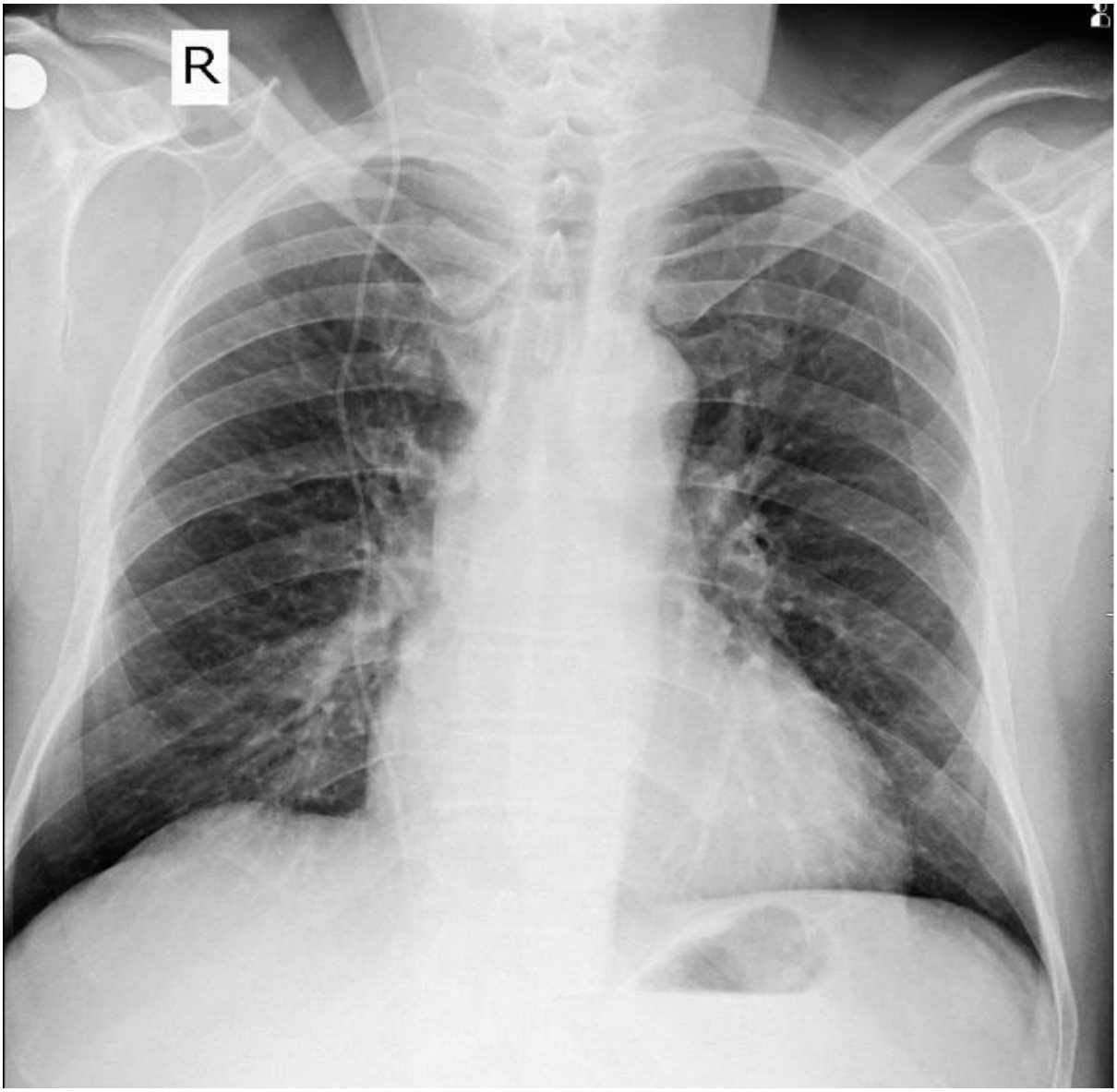

Figure 2: Chest X-ray post op 2. Day