Postoperative Negative Pressure Pulmonary Edema

Postoperatif Dönemde Gelişen Negatif Basınçlı Akciğer Ödemi

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Abstract

Negative pressure pulmonary edema (NPPE) can result from the increased intrathoracic and hydrostatic pulmonary pressure associated with forced inspiration against acute closures of the upper respiratory tract in the postoperative period. The associated postoperative complications include atelectasis, pneumonia and embolism. It should be kept in mind that NPPE is the cause of desaturation in the postoperative period in young patients. In patients who have undergone upper respiratory tract surgery, edema in the upper respiratory tract increases the risk of collapse, although rapid clinical response can be achieved in such patients with early diagnosis and intervention. The primary goal of treatment is to ensure the oxygenation of the patient, and non-invasive mechanical ventilation (NIMV) can be used in addition to oxygen support in some cases. NIMV, however, is contraindicated in patients who have undergone upper respiratory tract surgery, in whom full clinical response can be achieved with high-flow oxygen, methylprednisolone and diuretic treatment.

Keywords: Negative pressure, complication, pulmonary edema.

Öz

Negatif basınçlı akciğer ödemi (NBAÖ), postoperatif dönemde üst solunum yolundaki akut kapanmaya karşı yapılan zorlu inspirasyon sonucu artan intratorasik ve hidrostatik pulmoner basınca bağlı gelişir. Preoperatif dönemde en çok konsultasyon istenen branşlardan biri göğüs hastalıklarıdır. Postoperatif komplikasyonlar arasında atelektazi, pnömoni, emboli sıklıkla düşünülür. Genç hastalarda ameliyat sonrası dönemde desatürasyonun nedeni olarak NPPE olduğu akılda tutulmalıdır. Özellikle üst solunum yolu cerrahisi geçirmiş olan hastalarda üst solunum yolunda gelişen ödem, kollaps riskini arttırmaktadır. Bu hastalarda erken dönemde doğru tanı ve müdahale ile hızlı klinik yanıt alınmaktadır. Tedavide en önemli konu hastanın oksijenizasyonunun sağlanmasıdır. Geçirilmiş cerrahinin lokalizasyonuna göre kontrendike olmayan durumlarda oksijen desteğine ilave olarak non-invaziv mekanik ventilatör (NIVM) kullanılabilir. Ancak olgumuzdaki gibi üst solunum yolu cerrahisi geçiren hastalarda NIMV kontrendike olup NBAÖ için yüksek akımlı oksijen, metilprednizolon, diüretik tedavi ile de tam klinik yanıt alınabilir.

Anahtar Kelimeler: Negatif basınç, komplikasyon, akciğer ödemi.

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Submitted (Başvuru tarihi): 24.03.2024 Accepted (Kabul tarihi): 05.05.2024

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Negative pressure pulmonary edema (NPPE) is a rare but significant condition that can cause respiratory distress in the postoperative period, and serious morbidity and mortality can be prevented with early diagnosis and correct treatment (1). Pathophysiology refers to the development of pulmonary edema due to the increased intrathoracic and hydrostatic pulmonary pressure developing as a result of forced inspiration against acute closures of the upper airway, usually from upper airway infection, tumor or laryngospasm (2). Signs and symptoms such as respiratory distress, agitation, pink frothy sputum production, tachypnea, decrease in oxygen saturation and partial oxygen pressure, all of which are typical signs of acute pulmonary edema, usually appear immediately, but can sometimes occur up to 6 hours later. An NPPE diagnosis is not considered if the patient has clinical and radiological findings suggestive of pulmonary edema, has no disease history to explain these findings and has a normal cardiac evaluation.

CASE

The presented case is a 21-year-old male patient with no known history of additional disease. The patient, who had undergone elective rhinoplasty surgery, experienced suspicious aspiration while waking from anesthesia and was advised to consult a pulmonologist. The patient presented to the author due to desaturation in the 2nd postoperative hour and a postero-anterior chest X-ray was requested. The patient was desaturated during the bedside evaluation and distinct rales were noted in the examination findings. An X-ray revealed an appearance consistent with widespread pulmonary edema (Figure 1), and widespread interstitial edema findings were noted on the thorax CT taken while the patient was stable (Figure 2). The patient's saturation was 75% despite 6 L/min oxygen support, and so was transitioned from a cannula to a mask and transferred immediately to the intensive care unit. The bilateral widespread edema appearance on Xray led to negative pressure pulmonary edema being considered associated with the acute closure of the upper airway following upper airway surgery. The absence of a massive fluid transfusion by the anesthetist during surgery and causes that would trigger acute lung injury supported the diagnosis of NPPE. A physical examination revealed bilateral diffuse rales. The patient developed hematemesis, believed to be a result of the postoperative swallowing of blood emanating from the upper respiratory tract, but it abated during follow-up. The appearance of pulmonary edema led to him being referred to cardiology for the exclusion of cardiogenic edema, and he was recommended for diuretic treatment following the cardiology consultation based on the low EF of 60%, while no major heart valve pathology was observed. The goal of treatment in such patients is airway patency and adequate

oxygenation, targeting oxygen saturation above 90%. While the provision of oxygen via a mask is sufficient in mild cases, noninvasive mechanical ventilation support should be provided if there is no improvement in the patient's clinic and oxygen values. Our patient was followed up in the ICU with methylprednisolone, diuretics, empirical antibiotic treatment, bronchodilator treatment and mask oxygen, and clinical improvement was noted after 3 hours, with saturation at 85% despite 15 L/min oxygen support with a reservoir mask. NIMV support could not be provided due to the recent respiratory tract surgery. By the 12th hour, the patient's oxygen requirement was 5 L/min and saturation was 90%, while at 24 hours the patient's oxygen requirement was 2 L/min and there was a significant improvement in tachypnea and physical examination findings. Improvement was also noted on an X-ray (Figure 3) taken on the 2nd day of ICU follow-up, and since the patient no longer needed oxygen, he was transferred to the relevant surgical department.



Figure 1: Bilateral pulmonary infiltrations on chest X-ray after surgery (first hour)



Figure 2: Bilateral ground glass opacities after surgery (6th hour)



Figure 3: Chest X-ray after treatment (Second day)

DISCUSSION

Atelectasis, aspiration pneumonia and embolism all come to mind as potential postoperative complications, while NPPE is a rarer condition that can be treated successfully with rapid diagnosis and treatment, and should be kept in mind in cases of non-cardiogenic pulmonary edema, especially in cases of upper airway surgery/infection/tumor. An article published last year stated that although NPPE is rare, the number of cases may be low due to it being overlooked (3). In such patients, shortness of breath, tachypnea, and bloody and foamy sputum can be observed, while desaturation can be observed in vital follow-ups. Physical examinations reveal diffuse crackles in the lungs while chest X-ray can reveal pulmonary edema. Our case was first referred to me by the anesthesiologist after he was found to be desaturated while waking from surgery. Hemopoietic fluid was present during bedside evaluation, and a physical examination revealed widespread rales. The differential diagnosis in such patients includes other causes of pulmonary edema, such as aspiration of stomach contents, hypervolemia and pulmonary thromboembolism. In our patient, since no additional fluid was given during the operation and there was an absence of suspicious aspiration, differential diagnoses were made. The primary goal in the treatment of NPPE patients is adequate oxygenation, and as such, one of the optimal treatment options is the application of non-invasive positive pressure. Chuang et al. (4) recommended the use of steroid-derived drugs for the treatment of alveolar damage, thus avoiding systemic side effects, reducing respiratory distress and accelerating recovery. Although the existing edema is non-cardiogenic in such patients, diuretic treatment may be beneficial due to the accumulation of fluid in the interstitium from capillary leakage, and in one study, diuretic treatment was started with close hemodynamic, electrolyte and urine monitoring in the belief that it would contribute to the removal of fluid from the alveoli, with successful results (2). Although no obstructions occur in the bronchi in the respiratory effort mechanism that develops to counter laryngospasm, studies have shown that beta-agonist treatments facilitate the excretion of fluid from the alveoli (5). Our patient achieved a clinically significant response following rapid diagnosis and treatment, but after the patient was unresponsive to oxygen in the postoperative follow-up and NIMV was contraindicated due to upper respiratory tract surgery, we believed that the additional beta-agonist, methylprednisolone and diuretic treatments we applied, despite the noncardiogenic edema, also contributed to the treatment response. Cases of NPPE usually heal rapidly, both clinically and radiologically within 12-48 hours (2). In our case, complete clinical recovery was observed after approximately 24 hours, and complete radiological recovery after 36 hours.

Although it has been reported in literature that NIMV support can lead to successful results in NPPE cases that develop after upper respiratory tract surgery, clinical response can also be achieved with high-flow oxygen support. Particularly in cases in whom NIMV is contraindicated, priority should be given to alternative treatment options, taking into account the frequency of malpractice lawsuits today.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - E.K.G.; Planning and Design - E.K.G.; Supervision - E.K.G.; Funding - E.K.G.; Materials - E.K.G.; Data Collection and/or Processing - E.K.G.; Analysis and/or Interpretation - E.K.G.; Literature Review - E.K.G.; Writing - E.K.G.; Critical Review - E.K.G.

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