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Non-invasive Ventilation Treatment in a Patient with Severe Asthma Induced by Acute Cardiogenic Pulmonary Edema: Case Report+

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This is case of a non-invasive ventilation treatment of severe asthma with acute cardiogenic pulmonary edema, who underwent transurethral resection of the prostate under subarachnoid anesthesia. There is no sufficient data in literature about the occurence of non-invasive ventilation after severe asthma. We believed it is important to call attention to this unusual treatment.
Key Words: Asthma; Acute Cardiogenic Pulmonary Edema; Non-invasive Mechanical Ventilation.

Akut Kardiyojenik Pulmoner Ödem Gelişen Ağır Astımlı Hastada Non- invaziv Ventilasyon Tedavisi: Olgu Sunumu

Spinal anestezi altında transüretral rezeksiyon planlanan ağır astımlı bir hastada gelişen akut kardiyojenik pulmoner ödemin non invaziv ventilasyon ile tedavisi anlatılmaktadır. Literatürde ciddi astım sonrası non invaziv ventilasyon ile ilgili bilgi bulunmamaktadır. Biz bu nadir uygulanan tedaviye dikkat çekilmesinin önemli olduğuna inanıyoruz.
Anahtar Kelimeler: Astım; Akut Kardiyojenik Pulmoner Ödem; Non-invaziv Mekanik Ventilasyon.

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Noninvasive ventilation (NIV) has emerged over the last 2 decades to become the clear choice in the management of patients presenting with respiratory distress secondary to acute cardiogenic pulmonary edema (ACPE) and exacerbations of chronic obstructive pulmonary disease (COPD).[1] Clearly improves symptoms of the NIV for these patient groups . The evidence in the literature supporting NIV's use beyond patients with ACPE and COPD, however, is much weaker, especially for those patients presenting with severe asthma.

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Despite the continuous improvement in the therapeutic strategy for asthma, there is still a subset of asthma exacerbations severe acute asthma that still requires access to the emergency department and, eventually, hospitalization,[2,3] Endotracheal intubation and conventional mechanical ventilation are deemed necessary in up to one third of the severe-acute-asthma patients admitted to the intensive care unit (ICU). Mortality as high as 27% has been reported in invasively ventilated patients, given the likelihood of life-threatening intubation associated complications in severe acute asthma (eg, barotrauma, cardiovascular collapse, cardiac arrhythmia acute coronary syndromes, atelectasis, and pneumonia).[4,5] During acute exacerbations of COPD or acute asthma, the goal is to unload the respiratory muscles and to increase alveolar ventilation, and improving respiratuar acidosis.

NIV maybe an effective technique of ventilatory support that does not need an invasive interface (ie, endotracheal tube). NIV is a useful option in such kind of patients. Which patients with acute severe asthma benefit from NIV. So, we want to find this answer in this case report.

Case Report

A 76-year-old male patient with body mass index 24 and ASA III was scheduled for transurethral resection of the prostate under subarachnoid anesthesia. Preoperative assesment revealed with echocardiography showed extensive left ventricular anterior and apical wall motion abnormalities and ejection fraction 25% was present. The patient had a history of previous an asthma. The patient was transferred to operating room and standard monitoring was applied. The patient's baseline non-invasive blood pressure (NIBP) and heart rate (HR) were 140/80 mm Hg and 88 beats/min, respectively. An intraveous (IV) preload of 500 ml lactated Ringer's solution was started and spinal anaesthesia was performed with a 25 - G Quincke needle (Spinocan; B. Braun, Melsungen, Germany) at L_{3-4} using midline approach in the sitting position. Hyperbaric 0.5% bupivacaine 7,5 mg and 12,5µg fentanyl was slowly injected intrathecally. The patient was placed in the supine position and oxygen 3 L/min was administered via a face mask. As soon as dermatomal sensory block achieved T_{10} was started surgery. The patient's intraoperative NIBP, HR and SpO_2 was within the normal range. At the end of the procedure, the patient was transferred to the PACU. The patient was allowed untill to leave PACU when his sensory block level T_{12}. The patient developed sudden onset of dyspnea and the arterial oxygen desaturation in the clinic room. His condition continued to deteriorate with respiratory distress, diaphoresis, and chest examination showed bilateral audible crackles and expiratory wheezes on chest auscultaion. These showed clinical symptoms of acute asthma attack caused by ACPE. The patient was shifted to the ICU, anticipating for mechanical ventilation. Medical treatment was begun in the ICU. There was severe respiratory failure on arterial blood gas analysis (Table 1). The patient continued to deteriorate clinically with marked respiratory distress and confusion. At this time we decided to initiate ventilatory support system using the BIPAP in the spontaneous mode, noninvasive positive pressure ventilation (NIV) delivered by a tight fitting face mask. Initial settings were inspiratory positive airway pressure (IPAP) of 10 cm H_2O and expiratory positive airway pressure (EPAP) of 5 cm H_2O and FiO_2 100%. The face mask was well tolerated. The maximum pressures administered IPAP 15 and EPAP 8 cm H_2O. Levels of IPAP and EPAP were gradually reduced as the patient's clinical condition and blood gases showed a steady improvement. At 60 minutes of NIV after starting of ventilation there was marked improvement in respiratory rate, significant improvement in blood gases and dyspnoea decreased (Table 1).

Seventy five minutes after the application of NIV patient had normal blood gas values and pulmonary auscultation has been reached to normal. The patient diuresed 1,200 ml urine over the following 2 hours. He was discharged after a hospital stay of 2 days with a satisfactory arterial blood gas result in room air and there was almost complete cleaning on chest X ray.

Table 1. Sequential arterial blood gas values demonstrating high flow oxygen, and subsequent marked improvement with noninvasive positive pressure ventilation.

	At admission (FiO_2 0,5)	On NIPPV (IPAP 15, EPAP 8)	At discharge (room air)
pH	7,17	7.34	7,39
$PaCO_2$	65	45	42
PaO_2	53	83	91
SaO_2 %	86	94	96
RR	38	24	16

Discussion

Acute cardiogenic pulmonary edema is a common cause of respiratory failure that necessitates endotracheal intubation with mechanical ventilation.[6] Primary treatment is beginning standart medical care. In case of a significant respiratuar problem, and inadequate oxygenation or insufficient medical treatment, NIV is an alternative treatment to the endotracheal intubation.[7] NIV has been shown to improve physiological parameters quickly and decrease endotracheal intubation need mortality by a conventional volume ventilator, is an effective treatment for ACPE.[8] The rationale for NIV in severe acute asthma is based also on the large physiopathologic similarities between asthma attack and COPD exacerbation. Similar to a COPD exacerbation, the combination of increased work of breathing and inefficient ventilation during severe acute asthma may precipitate respiratory muscle fatigue and pump failure if these conditions persist, with the consequent need for a mechanical ventilatory support.[9] Substantial physiological data have shown that NIV efficiently respiratory muscles in COPD exacerbation, by reducing the diaphragmatic effort and by counterbalancing the dynamic hyperinflation (ie, intrinsic positive end-expiratory pressure), which benefits the breathing pattern and pulmonary gas exchange.[10] It has been also shown that NIV augment the response to bronchodilators, probably thanks to a better lung distribution of nebulized drugs (Table 1). In a prospective study, Meduri et al,[11] reported successful NIV in 17 severe acute asthma episodes with severe

acidosis (mean pH 7.25). NIV rapidly improved physiological variables with avoiding intubation in except 2 patients. A retrospective study reported favorable outcomes in 22 severe-acute-asthma patients treated with NIV due to persistent hypercapnia (mean PaCO2 63 mm Hg) and severe acidosis (mean pH 7.24), with an NIV-failure (intubation) rate of 14%.[12]

Conclusion

In conclusion, the existence of a strong physiopathologic rationale should keep open a "window" for the use of NIV in severe acute asthma.

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