Arch Bronconeumol. 2017;xxx(xx):xxx–xxx

Sociedad Española de Neumología y Cirugía Torácica SEPAR

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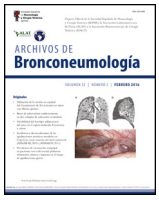

Original article

Non-Invasive Mechanical Ventilation Versus Continuous Positive Airway Pressure Relating to Cardiogenic Pulmonary Edema in an Intensive Care Unit☆

Alberto Belenguer-Muncharaz[a,b,*], Lidón Mateu-Campos[a,b], Rubén González-Luís[a], Bárbara Vidal-Tegedor[a], Amparo Ferrándiz-Sellés[a,b], Joaquín Árguedas-Cervera[a], Susana Altaba-Tena[a], Patricia Casero-Roig[a], Ester Moreno-Clarí[a]

[a] *Intensive Care Unit, Hospital General Universitario de Castelló, Spain*
[b] *Unidad Predepartamental Medicina, Facultad Ciencias de la Salud, Universitat Jaume I de Castelló, Spain*

ARTICLE INFO

Article history:
Received 7 October 2016
Accepted 8 February 2017
Available online xxx

Keywords:
Noninvasive ventilation
Continuous positive airway pressure
Cardiogenic pulmonary edema
Acute respiratory failure

ABSTRACT

Background: To compare the application of non-invasive ventilation (NIV) versus continuous positive airway pressure (CPAP) in the treatment of patients with cardiogenic pulmonary edema (CPE) admitted to an intensive care unit (ICU).
Methods: In a prospective, randomized, controlled study performed in an ICU, patients with CPE were assigned to NIV (n=56) or CPAP (n=54). Primary outcome was intubation rate. Secondary outcomes included duration of ventilation, length of ICU and hospital stay, improvement of gas exchange, complications, ICU and hospital mortality, and 28-day mortality. The outcomes were analyzed in hypercapnic patients ($PaCO_2$ > 45 mmHg) with no underlying chronic lung disease.
Results: Both devices led to similar clinical and gas exchange improvement; however, in the first 60 min of treatment a higher PaO_2/FiO_2 ratio was observed in the NIV group (205±112 in NIV vs. 150±84 in CPAP, P=.02). The rate of intubation was similar in both groups (9% in NIV vs. 9% in CPAP, P=1.0). There were no differences in duration of ventilation, ICU and length of hospital stay. There were no significant differences in ICU, hospital and 28-d mortality between groups. In the hypercapnic group, there were no differences between NIV and CPAP.
Conclusions: Either NIV or CPAP are recommended in patients with CPE in the ICU. Outcomes in the hypercapnic group with no chronic lung disease were similar using NIV or CPAP.

Ventilación mecánica no invasiva frente a presión positiva continua en la vía aérea en el manejo del edema pulmonar cardiogénico en una unidad de cuidados intensivos

Palabras clave:
Ventilación no invasiva
Presión positiva continua en la vía aérea
Edema agudo de pulmón cardiogénico
Insuficiencia respiratoria aguda

RESUMEN

Introducción: Comparar la efectividad de la ventilación no invasiva (VNI) frente a la presión positiva continúa en la vía aérea (CPAP) en pacientes ingresados por edema agudo de pulmón (EAP) cardiogénico en una unidad de cuidados intensivos (UCI).
Métodos: Ensayo clínico donde 56 pacientes fueron asignados a VNI y 54 pacientes a CPAP. El objetivo primario fue la tasa de intubación. Los objetivos secundarios fueron: duración de ventilación, estancia en UCI y en el hospital, mejoría gasométrica, complicaciones y mortalidad en UCI, hospitalaria y a los 28 días.

☆ Initial results of the study were presented in XLIV Congreso Nacional de la Sociedad Española de Medicina Intensiva y Unidades Coronarias (SEMICYUC), Valladolid 2009, Spain. Med Intensiva 2009; 33 (espec cong): 81–111.

* Corresponding author.
E-mail address: belengueralberto8@gmail.com (A. Belenguer-Muncharaz).

http://dx.doi.org/10.1016/j.arbres.2017.02.005
0300-2896/

Los objetivos fueron analizados en pacientes hipercápnicos ($PaCO_2$ >45 mmHg) sin patologia pulmonar.
Resultados: Ambos dispositivos obtuvieron similar mejoría clínica y del intercambio gaseoso, sin embargo, la VNI mostró un aumento más rápido de la oxigenación (medido por el cociente PaO_2/FiO_2) en los primeros 60 minutos de aplicación (205 ± 112 en VNI vs. 150 ± 84 en CPAP, p= 0,02). La tasa de intubación fue similar en ambos grupos (9% en VNI vs. 9% en CPAP, p= 1,0). No hubo diferencias en la duración de la ventilación, ni en la estancia en UCI ni hospitalaria. Tampoco hubo diferencias significativas en la mortalidad en UCI, hospitalaria y a los 28 días entre ambos grupos. En el subgrupo de pacientes hipercápnicos tampoco se observaron diferencias significativas en los objetivos analizados.
Conclusiones: La VNI como la CPAP se pueden emplear en pacientes con EAP en la UCI. En pacientes hipercápnicos sin patología pulmonar no se observa beneficio de la VNI sobre la CPAP.

Introduction

Cardiogenic pulmonary edema (CPE) is a cause of hypoxemic acute respiratory failure (ARF) due to acute heart failure. Traditionally, the standard medical treatment for CPE has been morphine, nitroglycerin, oxygen therapy and diuretics, and endotracheal intubation.[1]

Development of ventilatory support devices, such as continuous positive airway pressure (CPAP) and non-invasive ventilation (NIV), has played a decisive role in the treatment of ARF secondary to CPE. The use of either CPAP[2–7] or NIV[8–11] has resulted in greater clinical improvements compared with standard medical therapy. Hypercapnia without chronic lung disease has been associated with poor outcomes in patients with CPE,[12,13] particularly when $PaCO_2$ is higher than 60 mmHg.[13] Although there is a strong indication for NIV in hypercapnic patients,[11,12] the superiority of NIV over CPAP remains unclear, and hence, both have been recommended.[14–31]

NIV and CPAP have both been successfully used in CPE patients admitted to an intensive care unit (ICU).[8–10,28] However, few trials have been published in the ICU setting.[10,28] In addition, acute coronary syndrome (ACS) has been considered to be an exclusion criterion in several trials.[10,11,18,19,28–31]

The aim of the present study was to demonstrate that NIV performs better than CPAP in the management of CPE in an ICU setting. The primary outcome was a reduction in the need for endotracheal intubation in the NIV group. The secondary outcomes were duration of ventilation, ICU and hospital stay, ICU and hospital mortality, and 28-day mortality. The clinical and gasometric improvements, together with the rate of complications (renal failure, nosocomial infections), were all recorded. We also assessed the role of hypercapnia ($PaCO_2$ > 45 mmHg) on primary and secondary outcomes in patients with no underlying chronic lung disease.

Methods

A prospective, randomized study was conducted in a medical-surgical ICU from July 2007 to December 2010. The study protocols were approved by the local clinical research ethics committee. Written consent was required from all patients, or from their next of kin, before inclusion in the study. CPE patients aged 18 years or older admitted to the ICU from the emergency department (ED), a hospital ward, or the cardiac catheterization laboratory were included in the study. Cardiogenic pulmonary edema is defined as the presence of dyspnea, respiratory rate >25 breaths/minute, the use of accessory respiratory muscles, cyanosis, cold sweats, bilateral crackles and/or wheezing on pulmonary auscultation, hypoxemia, hypertension, and a predominance of bilateral pulmonary infiltrates on chest radiography (if available).[1] The potential causes of CPE are understood to be ACS with or without persistent ST-elevation, hypertensive emergency, valvulopathy, acute arrhythmia, endocarditis, or decompensation due to chronic heart failure.[1] The exclusion criteria were: refusal to give informed consent, inability to cooperate, severe encephalopathy (Glasgow coma score <10), anatomical difficulty when adjusting the face mask, non-cardiogenic ARF (pneumonia, blunt chest trauma, or chronic obstructive pulmonary disease), respiratory or cardiac arrest on admission, together with the need for immediate intubation.[15] Specific cardiac contraindications were also considered, including: cardiogenic shock on admission established by systolic blood pressure (SBP) <90 mmHg, or dependence on vasoactive drugs (norepinephrine >0.5 μg/kg/min). Hypercapnia was defined as partial pressure of carbon dioxide ($PaCO_2$) > 45 mmHg.[11,13] Patients with chronic obstructive pulmonary disease (COPD) or obstructive sleep apnea syndrome (OSA) were excluded for the analysis in the hypercapnic group.

Methodology

Patients were continuously monitored via electrocardiography and invasive or non-invasive blood pressure measurements. Blood oxygen was monitored using pulse oximetry, which estimates transcutaneous arterial oxygen saturation (SaO_2), together with arterial blood samples for ABG analyses using the ABL 800 Flex (Radiometer™, Denmark, Copenhagen) blood gas analyzer which measures partial pressure of oxygen (PaO_2), $PaCO_2$, partial pressure of oxygen to fraction of oxygen ratio (PaO_2/FiO_2), and pH. Demographic data, comorbidities, and predicted mortality using the Simplified Acute Physiology Score 3 (SAPS 3), were all collected on admission. All vital signs and arterial blood gases (if available) were recorded at baseline and at 1, 2 and 8 h after randomization. All complications arising during ICU stay were recorded, and patients were followed up for 28 days or until hospital discharge.

At the time of onset of CPE, either in the ED or on the ward, all patients received standard medical therapy (oxygen through a Venturi mask, morphine, intravenous nitroglycerin if SBP >160 mmHg, together with loop diuretics) at the discretion of the attending physician. In the absence of clinical improvement (dyspnea, respiratory rate >25 bpm, SaO_2 < 90%), the patient was admitted to the ICU and assigned to the NIV group or the CPAP group, regardless of the treatment received in the ED. Patients already in the ICU at onset of CPE were randomized without preliminary medical treatment. Patients were assigned to each group using computer-randomized treatment allocations contained in a sealed envelope.

Protocol

The NIV or CPAP procedure was explained to the recumbent patient. The oronasal mask was selected according to the patient's anatomy and subsequently adjusted using straps.[15] Two alternative procedures were used. In the first, the CPAP was applied using a flow generator (WhisperFlow™, Caradyne, Ireland) capable of delivering 140 L/min, with adjustable fractional inspired oxygen

Please cite this article in press as: Belenguer-Muncharaz A, et al. Non-Invasive Mechanical Ventilation Versus Continuous Positive Airway Pressure Relating to Cardiogenic Pulmonary Edema in an Intensive Care Unit. Arch Bronconeumol. 2017. http://dx.doi.org/10.1016/j.arbres.2017.02.005