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Update: Acute Heart Failure (VII)

Nonpharmacological Management of Acute Heart Failure

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ABSTRACT

Acute heart failure is a major and growing public health problem worldwide with high morbidity, mortality, and cost. Despite recent advances in pharmacological management, the prognosis of patients with acute decompensated heart failure remains poor. Consequently, nonpharmacological approaches are being developed and increasingly used. Such techniques may include several modalities of ventilation, ultrafiltration, mechanical circulatory support, myocardial revascularization, and surgical treatment, among others. This document reviews the nonpharmacological approach in acute heart failure, indications, and prognostic implications.

Tratamiento no farmacológico de la insuficiencia cardiaca aguda

Palabras clave:
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Apoyo circulatorio mecánico

RESUMEN

La insuficiencia cardiaca aguda constituye un problema de salud pública importante y creciente en todo el mundo, con una morbilidad, una mortalidad y un coste elevados. A pesar de los avances realizados en el tratamiento farmacológico, el pronóstico de los pacientes con insuficiencia cardiaca aguda descompensada continúa siendo malo. Por consiguiente, se están desarrollando abordajes no farmacológicos que se emplean de manera creciente. Estas técnicas pueden incluir varias modalidades de ventilación, ultrafiltración, apoyo circulatorio mecánico, revascularización miocárdica y tratamiento quirúrgico, entre otras. En este artículo se revisan los métodos no farmacológicos de tratamiento de la insuficiencia cardiaca aguda, sus indicaciones y las consecuencias pronósticas.

INTRODUCTION

Acute heart failure (AHF) constitutes a clinical condition with a complex pathophysiology, defined as a heterogeneous syndrome of signs and symptoms of new-onset or gradual/rapidly worsening heart failure (HF), requiring urgent intervention.[1] Despite recent advances in pharmacological management, its morbidity and mortality remain high. Consequently, nonpharmacological approaches are being developed and increasingly used.

POSITIVE AIRWAY PRESSURE THERAPY

Positive airway pressure (PAP) therapy has emerged as an important tool in the treatment of several forms of acute respiratory failure, representing a valuable nonpharmacological tool in the management of AHF. This therapy involves the maintenance of PAP through invasive or noninvasive methods.

In the clinical context of HF, PAP has several effects on hemodynamics: *a)* systemic venous return reduction and right ventricular unloading by increasing intrathoracic pressure,[2] and *b)* changes in total pulmonary vascular resistance, which is the major determinant of right ventricular afterload.[3] Total pulmonary vascular resistance is characterized by a U-shaped curve according to lung volume variation (the lowest pulmonary vascular resistance can be observed in the lung volume around functional residual capacity).

Positive airway pressure also has several effects on the respiratory system in HF: *a)* alveolar recruitment and prevention of alveolar collapse, improving gas exchange and oxygenation; *b)* induction of fluid shifts back from the alveoli and the interstitial space to the pulmonary circulation, and *c)* reduction of the respiratory muscle load and work of breathing.[4]

Types or Modes of Positive Airway Pressure Therapy in Heart Failure Treatment

Several types or modes of PAP therapy can be considered for HF. All of them apply PAP, in particular positive end-expiratory pressure, each type with different purposes. The main available types of PAP for AHF management are presented below.

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Abbreviations

AHF: acute heart failure
CS: cardiogenic shock
ECLS: extracorporeal life support
IABP: intra-aortic balloon pump
MCS: mechanical circulatory support
NPPV: noninvasive positive pressure ventilation
PAP: positive airway pressure
VAD: ventricular assist device

Invasive Ventilation

If invasive ventilation is required, lung protective modes should be performed to prevent pulmonary injury.

Noninvasive Ventilation

Noninvasive positive pressure ventilation (NPPV) has been widely used and its use should be encouraged to alleviate signs and symptoms of respiratory distress due to cardiogenic pulmonary edema. Evidence to date on the potential benefit of NPPV is derived from case series and relatively small, randomized, controlled trials. Most compare continuous PAP (CPAP) or bilevel PAP with standard therapy and suggest that noninvasive ventilation improves symptoms and physiological variables, reducing rates of invasive ventilation and mortality in selected patients.[5–8] Currently, there is still uncertainty regarding the real prognostic impact of NPPV in the context of AHF.

A recent randomized clinical trial of patients with AHF showed that neither type of NPPV reduced short-term mortality or the rate of endotracheal intubation when compared with standard therapy, despite early improvements in symptoms and in surrogate measures of disease severity.[9] However, a recent Cochrane review revealed lower mortality and reduced intubation rate with the use of in-hospital NPPV compared with standard medical treatment alone in patients with AHF.[10]

The question remains whether therapy with NPPV in AHF is of more benefit in patients with systolic dysfunction. Some authors argue that in patients with predominantly diastolic dysfunction (who require a relatively high filling pressure), the effects of positive pressure therapy might compromise venous return.[11]

Continuous Positive Airway Pressure

This therapy is the most widely used mode of PAP therapy in patients with HF. It provides a constant level of positive pressure to maintain airway patency during spontaneous breathing.

Observational studies on the effects of positive pressure on cardiac physiology in the setting of acute pulmonary edema have shown that CPAP improves cardiac output and lung compliance, decreasing lung and airway resistance.[12] Some studies have also shown that CPAP in patients with AHF reduces sympathetic tone, blood pressure, and heart rate.[13] When high-quality, randomized clinical trials were pooled in a meta-analysis, treatment with CPAP was associated with a 26% lower intubation rate and a trend toward overall lower mortality.[14]

Bilevel Positive Airway Pressure

Bilevel PAP provides 2 fixed levels of PAP: a higher level of pressure during inspiration (inspiratory PAP) and a lower level of pressure during expiration (expiratory PAP). Its major difference compared with CPAP is that it provides pressure support (difference between inspiratory PAP and expiratory PAP) during inspiration.

The level of inspiratory PAP plays an important role in unloading respiratory muscles, reducing the work of breathing, controlling obstructive hypopnea, maintaining alveolar ventilation, and reducing $PaCO_2$ (partial pressure of carbon dioxide).

Expiratory PAP produces hemodynamic and respiratory effects similar to those provided by CPAP. A retrospective analysis[15] reported that the use of bilevel PAP in patients with acute pulmonary edema was associated with a low intubation rate and intensive care unit stay.

However, theoretical advantages over CPAP have not been demonstrated in some nonrandomized comparisons with AHF patients, suggesting a possible negative effect.[16] Studies comparing the use of bilevel PAP vs CPAP in acute pulmonary edema reported that bilevel PAP was better than CPAP in increasing the oxygenation, decreasing carbon dioxide and respiratory rate and improving symptoms.[17,18] However, a recent randomized clinical trial showed no differences in treatment efficacy or safety between the 2 noninvasive ventilation treatments.[9] More comparative clinical trials are needed to answer these questions and close monitoring and proper patient selection are essential.

European guidelines generally recommend the use of NPPV therapy in AHF patients with a respiratory rate > 20 breaths/min and signs of pulmonary edema without shock (recommendation IIa B).[19]

Several factors have been reported to be associated with the success of noninvasive ventilation: *a)* patient-ventilator synchrony; *b)* Glasgow coma score > 9; *c)* acceptance of the technique by the patient; *d)* small amount of secretions; *e)* APACHE II (Acute Physiology and Chronic Health Evaluation II) score < 21; *f)* hypercapnia; *g)* initial arterial pH > 7.1; *h)* adequate response in the first hour of treatment, and *i)* high blood pressure at baseline.

Withdrawal is usually progressive, in general under the following conditions: *a)* improvement of dyspnea, without the use of accessory muscles; *b)* heart rate < 100 bpm; *c)* respiratory rate < 30 breaths/min; *d)* FiO_2 (fraction of inspired oxygen) ≤ 50%, spontaneous breathing without NPPV, SaO_2 (arterial oxygen saturation) > 90% and comfortable patient, and *e)* PaO_2 (partial pressure of oxygen) > 70 mmHg or PaO2/FIO2 ratio > 200 mmHg.

Contraindications and Possible Complications of Noninvasive Ventilation

There is no clear consensus on absolute and relative contraindications for the use of NPPV,[20] some of them being described as exclusion criteria in many studies. For possible contraindications and complications see Table 1.

ULTRAFILTRATION

Congestion and fluid retention, the hallmark of HF, cause about 90% of HF hospitalizations,[21] with their severity being associated with worse outcomes. The presence of overt congestion characterizes patients with higher neurohumoral activation, particularly of the renin-angiotensin-aldosterone system, intrarenal microvascular and cellular dysregulation, and oxidative stress. In these patients, glomerular filtration rate is usually decreased, sodium reabsorption in the proximal tubule is increased, and urinary sodium excretion is reduced.

The adverse effect of persistent congestion on outcomes has been shown in several studies. In the ESCAPE study, elevated