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Review article

Early use of non-invasive ventilation in acute heart failure to reduce catastrophic end results – a prospective study

Surjeet Acharya*, Arun Kaushik, Anita Rawat, Priya Govil, Kishalay Datta

Department of Emergency Medicine, Max Super Specialty Hospital, New Delhi, India

Corresponding author: Surjeet Acharya, ✉ drsa2495@gmail.com, **Orcid Id**: https://orcid.org/0000-0002-3925-1953

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ABSTRACT

Acute heart failure (AHF) syndromes may be accompanied by significant respiratory failure (RF) and is commonly seen in patients with acute cardiogenic pulmonary oedema (ACPE) or cardiogenic shock (CS). Use of non-invasive ventilation (NIV) has been proven to be useful in the treatment of moderate to severe RF in several scenarios. Non-invasive ventilation has shown to be effective in ACPE, by reducing both, the respiratory distress and the rate of endotracheal intubation when compared to conventional oxygen therapy, but the end-result on mortality is still less conclusive. In this article, we will highlight and discuss about the need of early NIV support in patients with AHF depending on - the early selection of patients with AHF, early application of NIV, the correct instructions and achievement of synchronization between patient and the ventilator settings and in some cases mild sedation, may prove the success of the technique.

Keywords: Non-invasive ventilation, acute heart failure, CPAP, IPAP, EPAP, acute cardiogenic pulmonary edema.

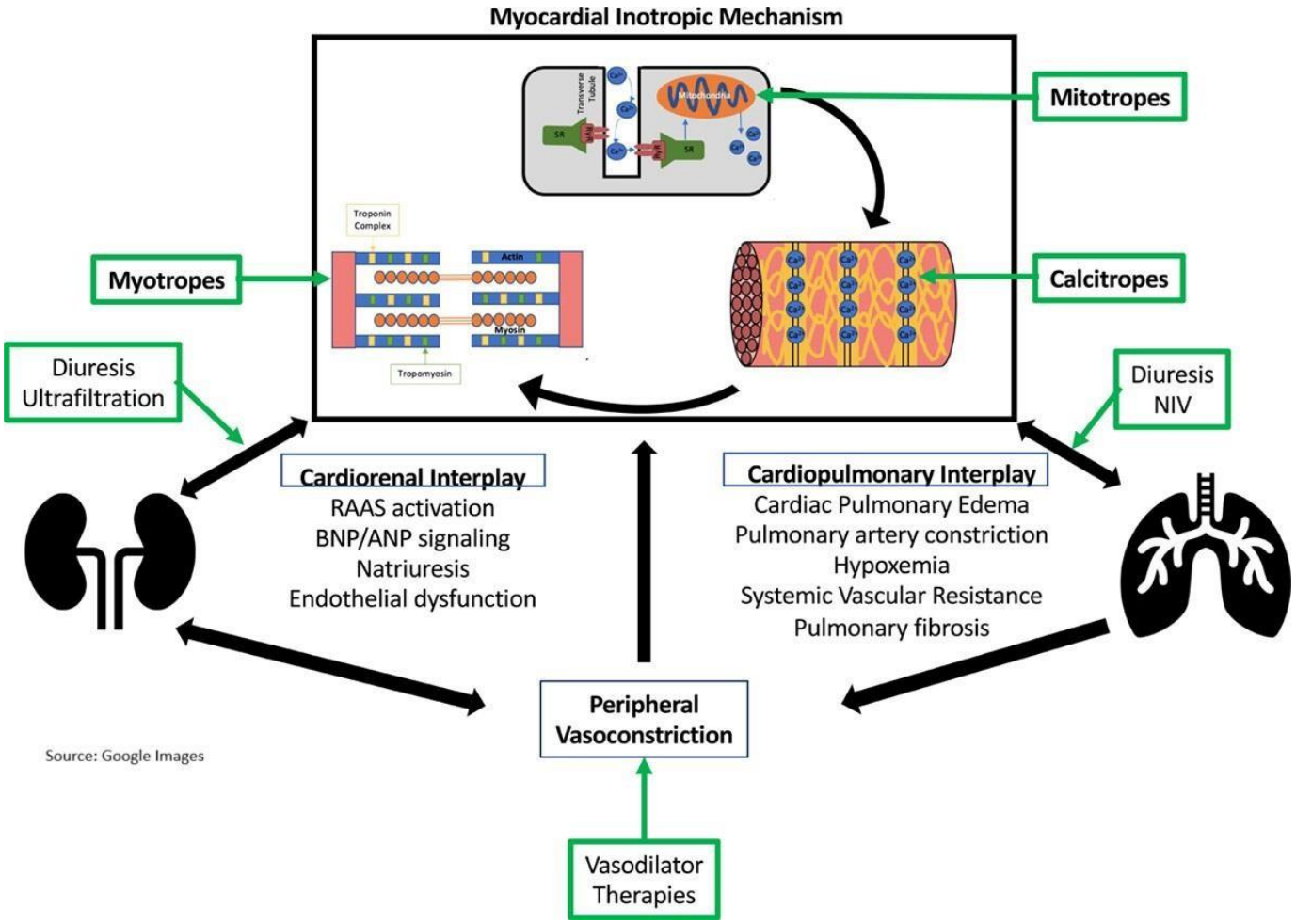
INTRODUCTION

Acute heart failure (AHF) is a clinical syndrome characterized by a majority of symptoms like dyspnea, orthopnea, lower limb swelling and signs like elevated jugular venous pressure and respiratory distress, often caused by a structural or functional abnormality in the myocardium resulting in reduced cardiac output and elevated intracardiac pressures [1]. The presentation and management of patients presenting with AHF is not well understood. These patients present with a sudden rapid onset of disease, often with underlying pre-existing cardiac illness, and their admission in the hospital holds a poor prognosis with a high risk of readmission and death post-discharge. The data available from the United Kingdom National Heart Failure Audit (UKNHF)

Demonstrates mortality rates during the admission of around 10% with a post- discharge 30-day and 1-year mortality of 6.5% and 30%, respectively [2]. In this article, we have tried to emphasize the need of early NIV application in acute heart failure patients for a better clinical outcome and reducing the mortality and morbidity.

Review of Literature

Acute heart failure is broadly defined as a rapid onset of new or worsening signs and symptoms of AHF [3]. It is a life-threatening condition, requiring hospitalization, and emergency treatment is aimed predominantly at managing fluid overload and hemodynamic compromise. This umbrella term includes patients presenting for the first time with typical symptoms and signs of heart failure (de novo AHF) and also those with worsening of their pre-existing cardiomyopathy (acute decompensated heart failure).The understanding of the diseased condition has improved significantly in the past few years, both pathophysiological perspective and the provision of disease-modifying therapies, but, the presentation and management of patients with acute heart failure (AHF) is less well understood. AHF occurs when - there is a sudden increase in cardiac filling pressures with/without acute myocardial dysfunction, which can lead to decreased peripheral perfusion and pulmonary edema. The most common cause is cardiac ischemia along with (sub) total coronary vessel occlusion leading to decreased contractility in myocardium. Other less common cause for AHF is non-ischemic myocardial insults like acute myocardial dysfunction with – toxic insults (e.g drug-induced cardiomyopathy), inflammatory insults (e.g. viral cardiomyopathy), and other per partum cardiomyopathy.


Myocardial Inotropic Mechanism
Mitotropes
Myotropes
Calcitropes
Diuresis
Ultrafiltration
Diuresis
NIV
Cardiorenal Interplay
RAAS activation
BNP/ANP signaling
Natriuresis
Endothelial dysfunction
Cardiopulmonary Interplay
Cardiac Pulmonary Edema
Pulmonary artery constriction
Hypoxemia
Systemic Vascular Resistance
Pulmonary fibrosis
Peripheral
Vasoconstriction
Vasodilator
Therapies
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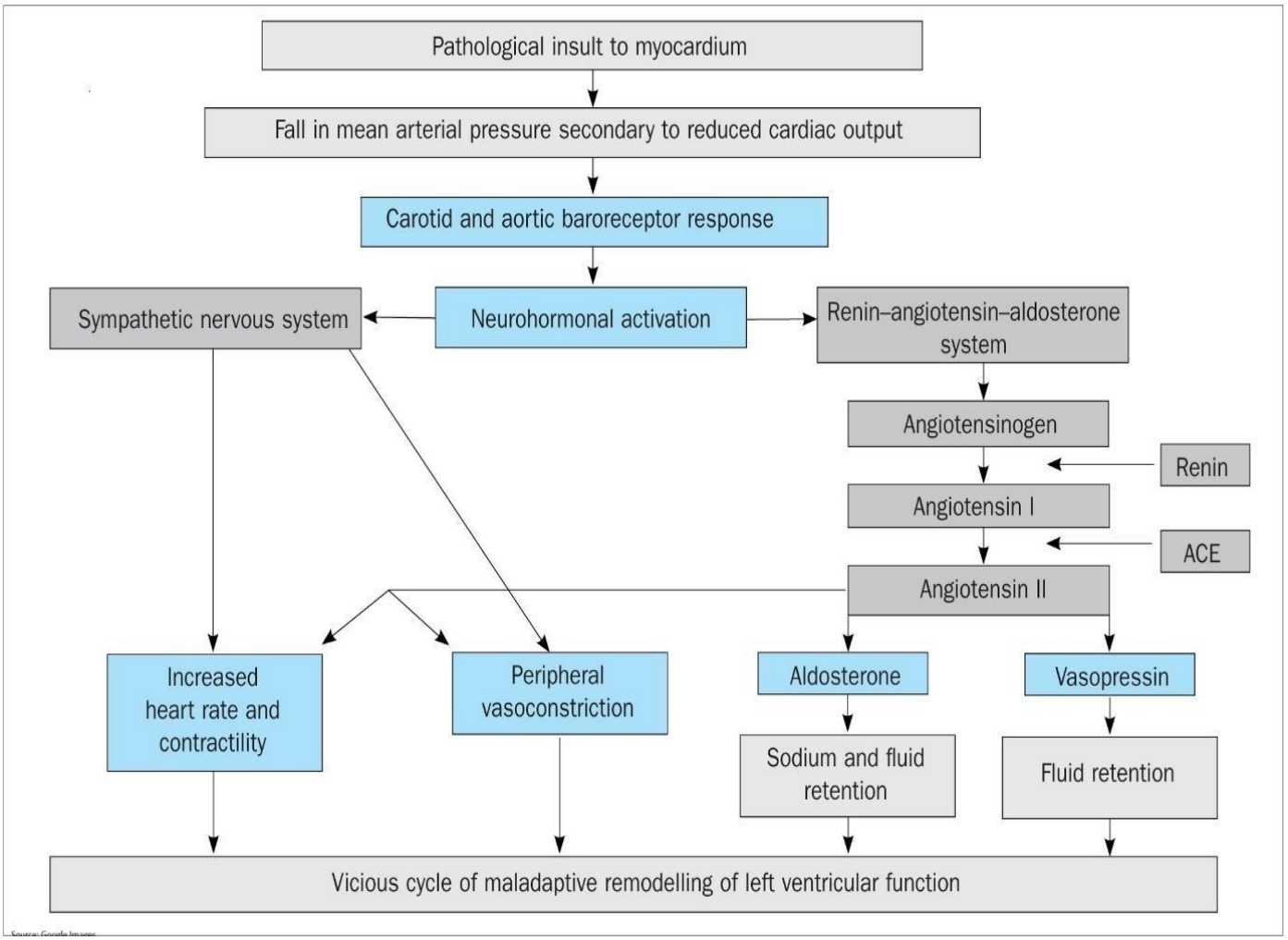
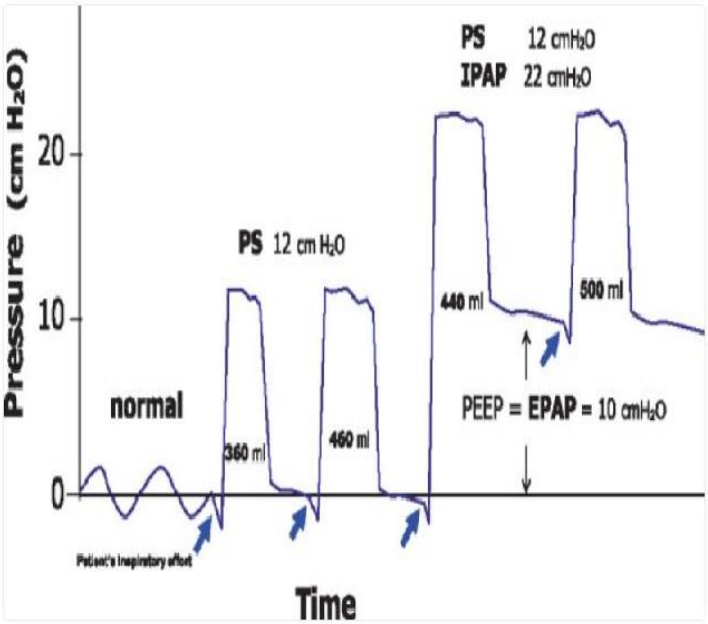

Pathological insult to myocardium
Fall in mean arterial pressure secondary to reduced cardiac output
Carotid and aortic baroreceptor response
Sympathetic nervous system
Neurohormonal activation
Renin–angiotensin–aldosterone system
Angiotensinogen
Renin
Angiotensin I
ACE
Angiotensin II
Increased heart rate and contractility
Peripheral vasoconstriction
Aldosterone
Vasopressin
Sodium and fluid retention
Fluid retention
Vicious cycle of maladaptive remodelling of left ventricular function

Main physiologic effects of positive intrathoracic pressure:
Cardiovascular
↓ Venous return → ↓ RV preload → ↓ LV preload
↑ Pulmonary vascular resistance → ↑ RV afterload → RV enlargement →↓ LV Compliance
↓ LV afterload (↓ systolic wall stress)
↓ Systemic blood pressure → ↓ Cardiac output[a]
Respiratory
Recruitment of collapsed alveoli → ↑Functional residual capacity
Maintenance continuously opened alveoli→ Gas exchange during the whole respiratory cycle
Intra-alveolar pressure against oedema
↓ Work of breathing
↑ Oxygenation

In patients with AHF with elevated LV preload and afterload, cardiac output may increase as consequence of the application of positive intrathoracic pressure.
RV, right ventricle; LV, left ventricle.



Pressure support (PS) curves. Pressure curves of a patient breathing on room air, after the application of PS of 12 cmH_2O and after adding positive end expiratory pressure (PEEP) of 10 cmH_2O. Blue arrows indicate patient's inspiratory effort that triggers the ventilator to deliver a decelerating flow to reach the preset PS. Inspiration is interrupted when the patient finishes the inspiratory effort or the flow arrives to a percentage of the peak (usually 25%). Inspiratory positive airway pressure (IPAP) is the sum of PS and PEEP, whereas PEEP is equivalent to expiratory positive airway pressure (EPAP). Note that tidal volumes change in every cycle according to patient's effort.

The rationale of using non-invasive ventilation is an increase in the level of oxygenation and decrease in the work of breathing [4]. It also has an additional improvement in alveolar ventilation leading to reduction in the level of carbon dioxide. In heart failure patients with increased in preload and afterload, use of NIV may improve the cardiac output by reducing both preload and afterload [5, 6] and reducing intrapulmonary shunting [7].

Methodology

In this article, we will try to establish the usefulness of early application of NIV support in patients with acute left ventricular failure in the emergency and the final outcome and 6 months mortality and morbidity score.

This is a prospective study, including patients with acute heart failure visiting the emergency department of our hospital for further management.

Study Population

The patients presenting to the emergency department of Max Super Specialty Hospital, Shalimar Bag, New Delhi, with acute heart failure.

Study Duration

Duration of the study is 6 months (March 2024 – August 2024).

Inclusion Criteria

Adult patients visiting the Emergency Department with acute heart failure.

Exclusion Criteria

Age < 18

Underlying respiratory disorder

Not consenting for the participation in the study

Study Process

Patients will be divided into 2 groups – one group will be having NIV application in the emergency department and the second group will be treated as per conventional treatment of acute heart failure. All the tests and diagnostic process will be followed same for both groups. The final result will be on – outcome of the patient during discharge and 6 months follow-up.

RESULT

A total of 184 patients were enrolled for this study qualifying the inclusion and exclusion criterions.

Groups were made:

The intervention group (A - early NIV applications): 92

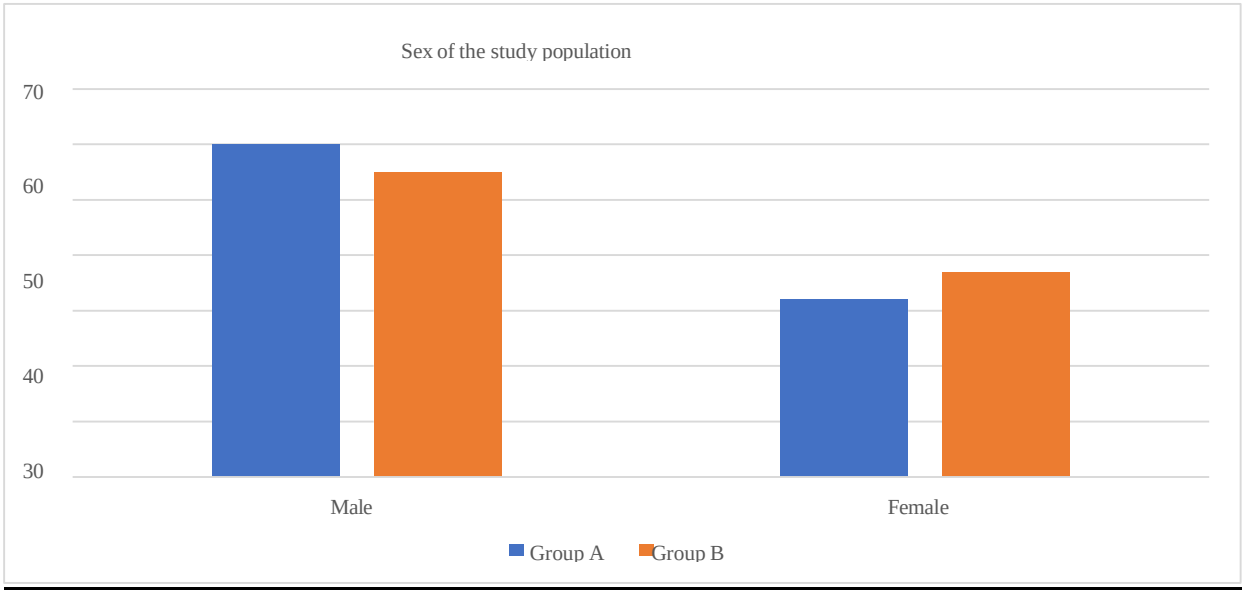
The non-interventional group (B - following the conventional treatment protocol): 92

The following were the results noted:

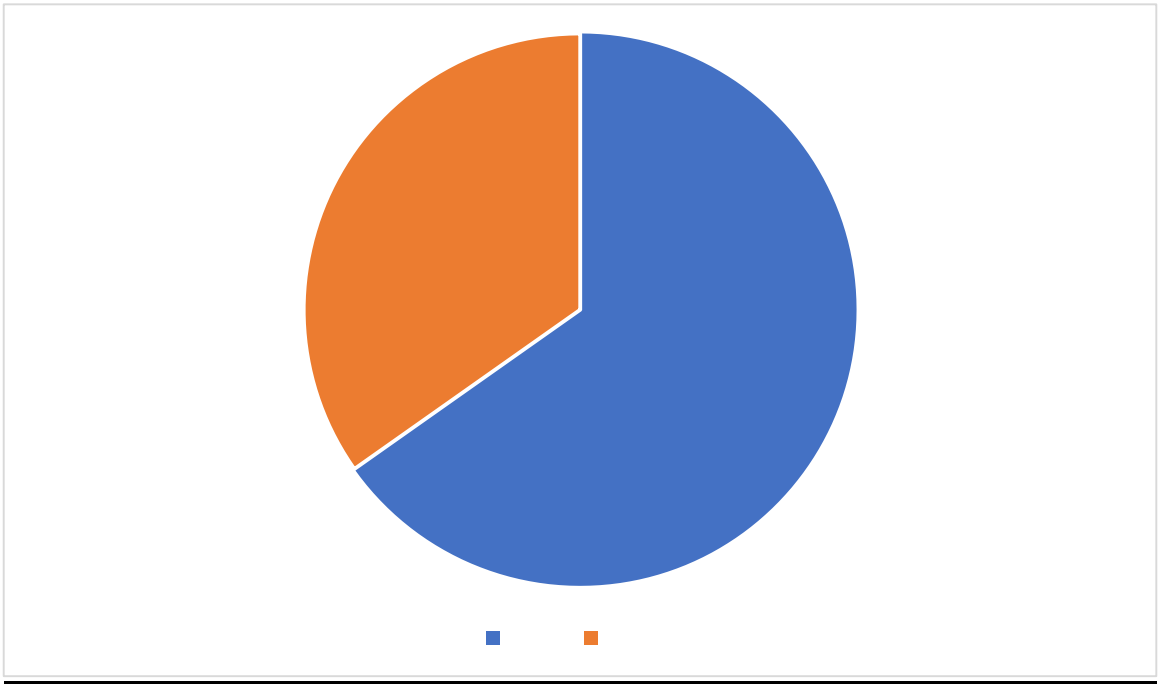
Age-wise distribution

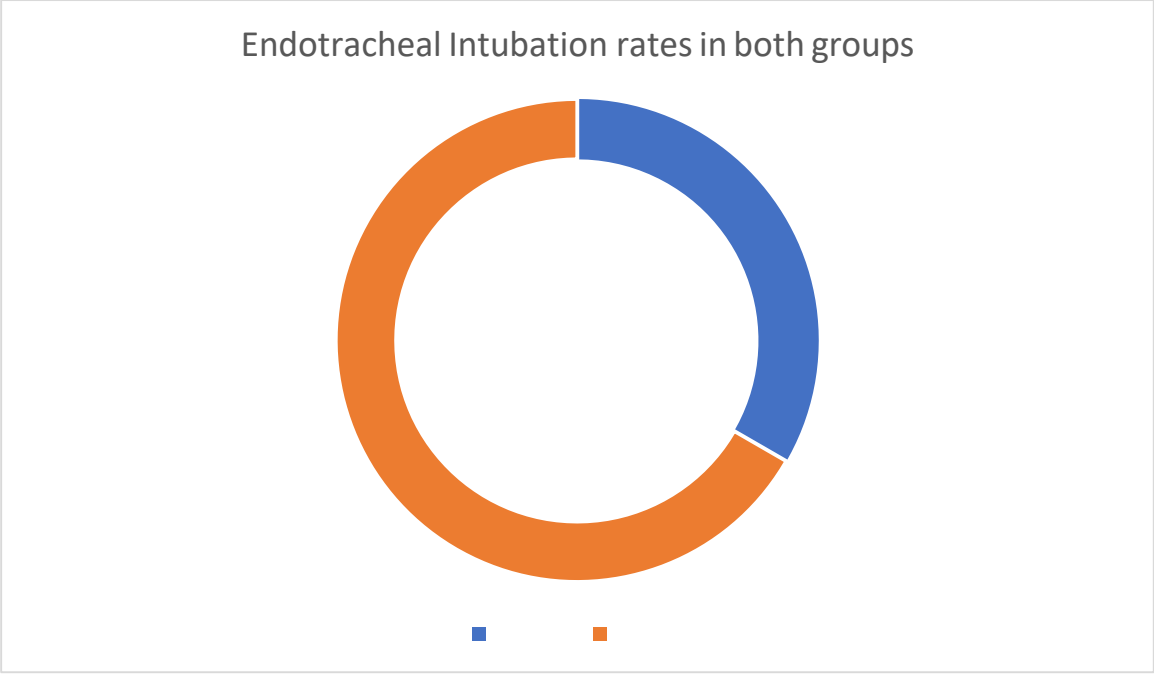
	Group A	Group B
20-30	0	0
30-40	5	4
40-50	11	12
50-60	34	38
60-70	26	22
70-80	10	8
>80	6	8

Sex

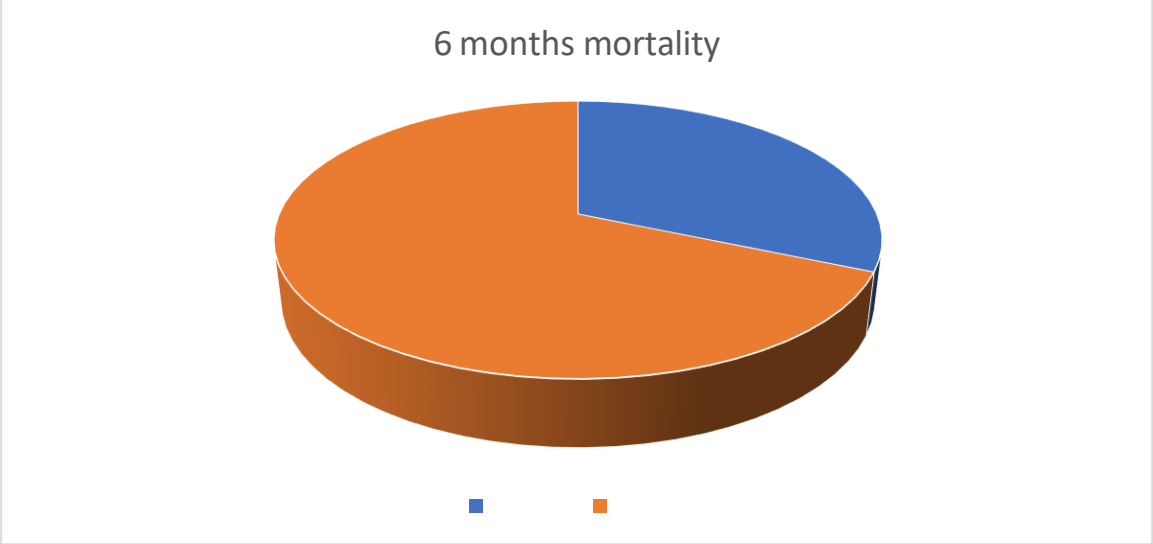


Early NIV applications





Months- mortality



Condition on discharge

Condition of discharge	**Group A**	**Group B**
Stable	82	68
Require special nursing at home	8	16
Dead	2	8

It was seen that higher number of patients succumbed to their ill-ness in group B category when compared to group A in a 6 months mortality frame.

DISCUSSSION

Our study was done and evaluated for patients with acute heart failure arriving to the emergency department. Acute cardiogenic pulmonary oedema or acute heart failure is the second most common indication for NIV application [8]. In the patients getting admitted in the cardiac care unit (CCU). Based on the results found above, we can clearly define the age, sex predominance for acute heart failure. It was seen that early NIV application had good outcome in patients with acute heart failure than the other group (of non-applicant). The first randomized control trial performed at the end of the 1980's using CPAP, showed faster improvement of RF than conventional therapy [9]. With a reduction in endotracheal intubation rate [10].

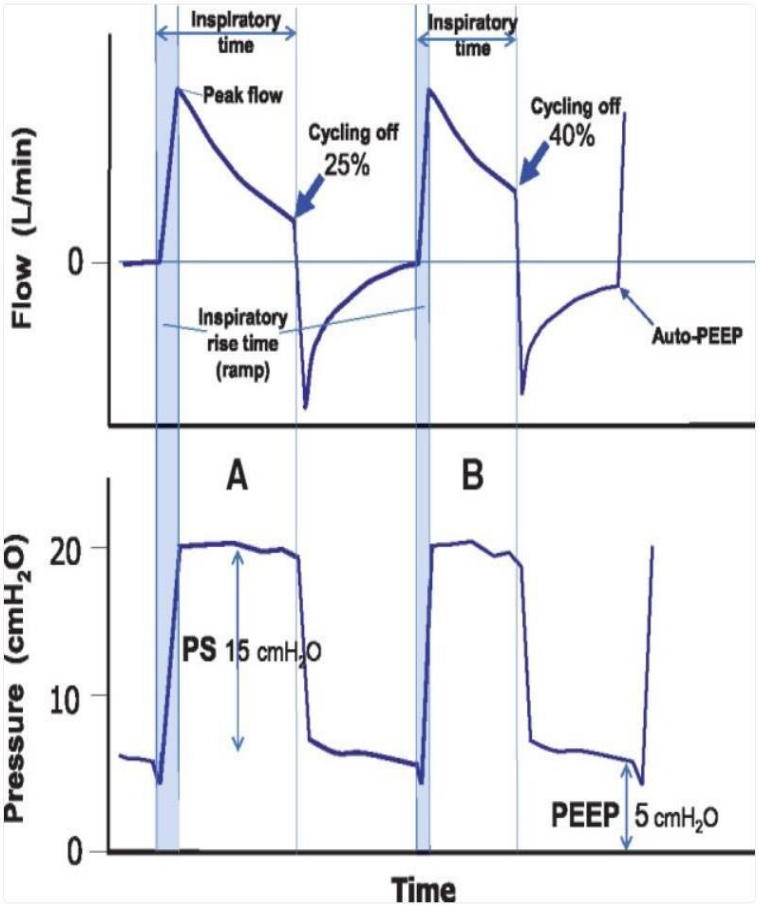
The rate of early intubation or need for invasive ventilation was higher in group B than in group A. The final outcome also varied hugely. Several studies over the last few years have shown that the early application of CPAP/NIV in the pre-hospital care of patients with AHF improved faster than conventional treatment therapy, with a tendency to reduce the rate of endotracheal intubation [11, 12]. The latest guidelines by European society for critical care medicine have given NIV Class IIa recommendation [13]. In patients with respiratory distress (respiratory rate > 25 breaths/min, SpO_2 <

90%) in acute heart failure. The NICE guidelines in AHF recommended NIV in patients with ACPE with severe dyspnoea and acidaemia [14].

We would like to recommend that NIV should be used in patients with ACPE, as defined above, in order to reverse RF faster, reduce chances of endotracheal intubation and with lower evidence, potentially reduce risk of mortality in critically ill patients. The usage of CPAP may be the best option in the pre- hospital setting. Before initiating NIV, the contraindications for NIV should be considered. Communication plays a vital role in successful NIV therapy. Empathic communication between nurses/physicians and the patient is essential, with clear instructions about what to expect and frequent encouragement thereafter.

Contraindications of NIV	
Absolute	Cardiac or respiratory arrest
	Anatomical abnormality (unable to fit the interface)
	Inability to keep patent airway (uncontrolled agitation, coma[a] or obtunded mental status)
	Refractory hypotension
Relative	Mild agitation or poor cooperation
	Mild hypotension
	Upper gastrointestinal haemorrhage or vomiting
	Inability to expectorate copious secretions
	Recent frail upper gastrointestinal or airway surgery
	Multiorgan failure
	Isolated right ventricular failure
[a]Modalities have been	like NIV with volume controlled or 'Average volume assured pressure support' used in hypercapnic encephalopathy.

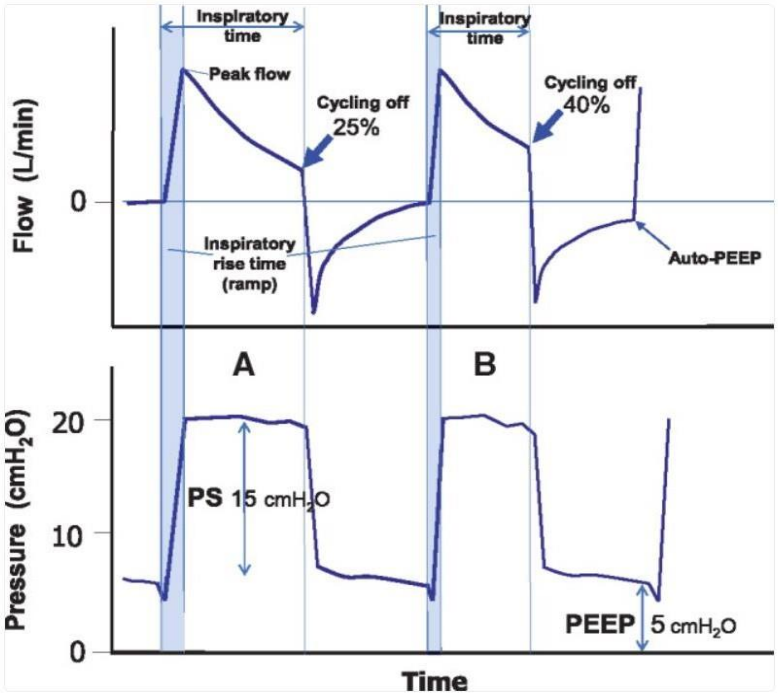
Settings of ventilator in NIV/CPAP



Pressure and flow curves in non-invasive pressure support ventilation (NIPSV). (*A*) Non-invasive pressure support ventilation delivered with a cycling off of 25% of the maximal peak flow. (*B*) Decrease of the inspiratory time after the reduction of the ramp and the increase of the cycling off to 40%. Example of flow curve with Auto-PEEP (the expiratory flow does not arrive to 0). PEEP, positive end expiratory pressure; PS, pressure support.

For NIV, it is recommended to start with low levels of PEEP (3–4 cmH2O) and pressure support of 7–8 cmH2O, increasing it progressively according to the patient adaptation and response. Target of tidal volumes are 4–7 mL/kg (often lower in COPD patients). With pressure support of 10–18 cmH2O and PEEP of 4–7 cmH2O (IPAP 15–20 cmH2O/EPAP 4–7 cmH2O), a suitable ventilation is generally achieved. High pressures may cause excessive air leakage, asynchrony (especially in patients with high respiratory rate) and discomfort.

While using CPAP, it is advised to start with 5 cmH2O, increasing soon to 7.5 or 10 cmH2O, according to the response. Later, FIO2 and flow rate can be decreased according to SpO2 [16] and patient's demand. In less severe cases, it is usually started with lower flow and FIO



Pressure and flow curves in non-invasive pressure support ventilation (NIPSV). (*A*) Non-invasive pressure support ventilation delivered with a cycling off of 25% of the maximal peak flow. (*B*) Decrease of the inspiratory time after the reduction of the ramp and the increase of the cycling off to 40%. Example of flow curve with Auto-PEEP (the expiratory flow does not arrive to 0). PEEP, positive end expiratory pressure; PS, pressure support.

While on NIV, it is necessary to monitor the patients during the period of NIV therapy. As per the recommendation below

Patient
Respiratory rate
Other vital signs
Dyspnoea/accessory muscle use/abdominal paradoxical breathing
Level of consciousness
Comfort with the interface
Collaboration
Ventilator parameters
Tidal volume (>4 mL/Kg: 6–7 mL/Kg) and minute ventilation
Air leakage volume (<0, 4 L/s or < 25 L/min)
Pressure support and PEEP settings
Asynchrony (ineffective efforts, auto-triggering, double-triggering, short/long cycle)[a]
Trigger/slope (ramp)/Inspiration time/expiration settings
Auto-PEEP
Alarms (apnoea or high respiratory rate, low/high minute ventilation, others)
Gas exchange
Continuous pulse-oximetry (SpO_2)
Arterial or venous blood gas samples[b]
Risk factors of failure
Before initiation
Lung infection
Altered mental status
Hypotension
High severity scores
Copious secretions
Extremely high respiratory rate
Severe hypoxaemia in spite of high F_IO_2
After initiation
Inappropriate ventilator settings
Unfitting interface
Excessive air leakage
Asynchrony with the ventilator
Poor tolerance to NIV
After 60–90 min
No reduction in respiratory rate or carbon dioxide
No improvement in pH or oxygenation (↓SpO_2 or ↓PaO_2/FiO_2)

Signs of fatigue
Neurological or underlying disease impairment
Criteria for endotracheal intubation
Cardiac or respiratory arrest
Progressive worsening of altered mental status
Progressive worsening of pH, $PaCO_2$, or PaO_2 despite NIV
Progressive signs of fatigue during NIV
Need to protect the airway
Persistent haemodynamic instability
Agitation or intolerance to NIV with progressive respiratory failure

[a] Asynchrony: *Ineffective efforts*: inspiratory efforts not followed by a cycled response from the ventilator. *Auto-triggering* or *double-triggering*: cycled respirations out of patients' demand. These asynchronies should be managed by reducing the leakage, tuning the inspiratory trigger, and adjusting the level of pressure support. *Prolonged cycle (delayed cycling off)*: cycled mechanical inspiratory time longer than patient's inspiratory time. It may be compensated by reduction of

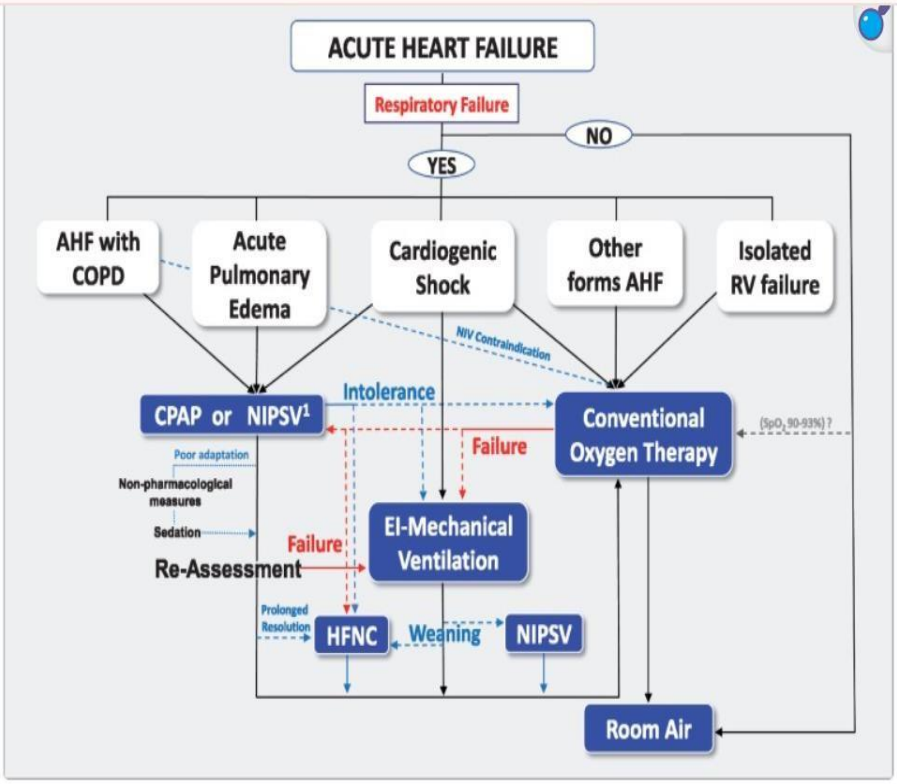
Leakage, decrease of pressure support, inspiratory time or ramp, and when available, titration of expiratory trigger. *Auto-PEEP*: air trapping due to a limitation of the expiratory airflow. Observed in COPD and cases with high respiratory frequency. Treated with measures to extend expiratory time and decrease respiratory rate, titrating PEEP (compensate 80% of the auto-PEEP in COPD patients). [b] Baseline and after 60–90 min of NIV for: PaO_2/FiO_2, pH, $PaCO_2$, and bicarbonate; venous samples are suitable for pH, bicarbonate, and SvO_2.

PEEP, positive end-expiratory pressure; PaO_2, arterial partial oxygen pressure; $PaCO_2$, arterial partial carbon dioxide pressure; FiO_2, fraction of inspired oxygen; SpO_2, oxygen saturation by pulse-oximetry; NIV, noninvasive ventilation.

Non-invasive ventilation is usually terminated when: a satisfactory recovery has been achieved (usually 2–5 h in AHF), there are signs of NIV failure, the need for endotracheal intubation. After mid- or long-term use of NIV (>24 h), a weaning [17] period is often carried out, by decreasing F_IO_2, PEEP, and ventilation settings progressively. Early mobilization may shorten this process.

The number of deaths in our study was found higher in group B than group A. Please note, these deaths were not related to our study but due to the complications of the disease process *per se* [15].

CONCLUSION



Algorithm for non-invasive ventilation in acute heart failure syndromes. After any NIV technique, patients should receive conventional oxygen therapy (COT) before switching to room air. The administration of COT in patients with SpO_2 ranging 91–93% is not clear. [1]Continuous positive airway pressure may be preferred in pre-hospital and low equipped areas, whereas non-invasive pressure support ventilation may be chosen by experienced teams, in patients with significant hypercapnia or COPD. Proportional assist ventilation, adaptive servoventilation, and HFNC have also been used in some trials as first line therapy in ACPE. COPD, chronic obstructive pulmonary disease; HFNC, high-flow nasal cannula; EI, endotracheal intubation; COT, conventional oxygen therapy; ACPE, acute cardiogenic pulmonary oedema.

In patients with AHF, NIV plays an important role in the management of ACPE. It may be also considered in patients with RF associated with lung disease and in some cases of cardiogenic shock (after stabilizing the blood pressure) in AHF. CPAP is a simpler technique that is recommended as first line therapy in such patients, especially in the pre-hospital setting or in less well-equipped areas. NIV support ventilation is equally effective in ACPE and may be preferable, by experienced teams and cooperative patients.

This study still could not explain the mortality in patients with early NIV / CPAP usage in AHF and we suggest further studies for having a better overall accountability.

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