

Effectiveness of Non-Invasive Ventilation in Patients with Acute Respiratory Failure Admitted in Intensive Care Unit; a Cross Sectional Study



SUPERIOR UNIVERSITY

Thesis Submitted to

The Superior University Lahore

In Partial Fulfillment of the requirement for the Degree of

Master of Science in Allied Health Sciences

By

MUHAMMAD BILAL

SU91-MSAHW-S23-090

Session: 2023-2025

Faculty of Allied Health Sciences

Author's Declaration

I hereby state that my MS thesis titled **“Effectiveness of Non-Invasive Ventilation in Patients with Acute Respiratory Failure Admitted in Intensive Care Unit: A Cross Sectional Study”** is my work and has not been submitted previously by me for taking any degree from this University, **The Superior University, Lahore** or anywhere else in the country/world.

At any time if my statement is found to be incorrect even after my graduation, the university has the right to withdraw my MS Degree.

Name of Student: Muhammad Bilal

Date: _____

Plagiarism Undertaking

I solemnly declare that research work presented in the thesis titled **“Effectiveness of Non-Invasive Ventilation in Patients with Acute Respiratory Failure Admitted in Intensive Care Unit: A Cross Sectional Study”** is solely my research work with no significant contribution from any other person. Small contribution/help wherever taken has been duly acknowledged and that complete thesis has been written by me. I understand the zero-tolerance policy of the HEC and University, **The Superior University, Lahore**, towards plagiarism. Therefore, I as an author of the above-titled thesis declare that no portion of my thesis has been plagiarized and any material used as a reference is properly referred/cited. I undertake that if I am found guilty of any formal plagiarism in the above-titled thesis, even after awarding of MS degree, the University reserves the rights to withdraw/revoke my MS degree and that HEC and the University have the right to publish my name on the HEC/University website on which names of students are placed who submitted a plagiarized thesis.

Student/Author Signature: _____

Name: Muhammad Bilal

Research Completion Certificate

This is to certify that the thesis entitled **“Effectiveness of Non-Invasive Ventilation in Patients with Acute Respiratory Failure Admitted in Intensive Care Unit: A Cross Sectional Study”** submitted by **“Muhammad Bilal”** has been accepted towards the partial fulfillment of the requirement for **“MS Allied Health Sciences”**. The quality of the work contained in this thesis is adequate for the award of degree.

Signature: _____

Supervisor Name: Dr. Amdad Ali Faruqi

MBBS, FCPS(Med), FCPS-CCM(R)

Lecturer

Faculty Allied Health Sciences

Certificate of Approval

This is to certify that the research work presented in this thesis, titled “**Effectiveness of Non-Invasive Ventilation in Patients with Acute Respiratory Failure Admitted in Intensive Care Unit: A Cross Sectional Study**” was conducted by “**Muhammad Bilal**” under the supervision of “**Dr. Amdad Ali Faruqi**” No part of this thesis has been submitted anywhere else for any other degree. This thesis is submitted to the Faculty of Allied Health Sciences, The Superior University, Lahore in partial fulfillment of the requirements for the degree of Master of Science in the field of “**Allied Health Sciences**” in Faculty of Allied Health Sciences at The Superior University, Lahore.

Student Name:

Signature: _____

Examination Committee:

Session Chair:

Signature: _____

a) External Examiner:

Signature: _____

b) Internal Examiner:

Signature: _____

c) Supervisor Name:

Signature: _____

d) PL/HOD: Dr. Hafiz Shehzad

Signature: _____

e) Dean: Prof. Dr. M. Naveed Babur

Signature: _____

f) Controller of Examination: Dr. M. Haris

Signature: _____

DEDICATION

Dedicated to my exceptional parents, family and adored siblings whose tremendous support and cooperation led me to this wonderful accomplishment

ACKNOWLEDGEMENT

In the name of Allah, the most Gracious, the most Merciful. I am extremely thankful to Almighty ‘Allah’ Who is the entire source of knowledge and wisdom endowed to mankind, for providing me with the acumen and vision to complete this endeavor. I would like to express my profound gratitude to my supervisor for his/her wise counsel and encouraging attitude towards this study. I am extremely grateful to him/her for immensely facilitating me during my study period by ensuring the provision of favorable circumstances and conducive environment. This project would not have been possible without his support and expert guidance. In the end, I would like to extend my deepest gratitude to my family members. Without their encouragement, I would not have been able to complete this endeavor.

Student Name: Muhammad Bilal

TABLE OF CONTENTS

AUTHOR’S DECLARATION	ii
PLAGIARISM UNDERTAKING.....	iii
CERTIFICATE OF APPROVAL	v
DEDICATION	vi
ACKNOWLEDGEMENT	vii
TABLE OF CONTENTS.....	viii
LIST OF TABLES.....	x
LIST OF FIGURES	xii
LIST OF ABBREVIATIONS	xiii
ABSTRACT	13
Chapter 1: INTRODUCTION	14
AIM AND OBJECTIVES.....	19
Chapter 2: LITERATURE REVIEW	20
Chapter 3: METHODOLOGY	26
3.1. Research Design	26
3.2. Clinical Settings.....	26
3.3. Sample Size	26
3.4. Sampling Technique	26
3.5. Duration of Study.....	26
3.6. Selection Criteria	26
3.6.1. Inclusion Criteria	26
3.6.2. Exclusion Criteria	27
3.7. Ethical Consideration.....	27

3.8. Data Collection Procedure.....	28
3.9. Data Analysis.....	28
Chapter 4: RESULTS	29
Chapter 5: DISCUSSION	55
CONCLUSIONS	58
LIMITATION	59
RECOMMENDATIONS	60
REFERENCES	61
APPENDICES.....	67
ENGLISH CONSENT FORM.....	67
URDU CONSENT FORM.....	68
DEMOGRAPHICS FORM & QUESTIONNAIRES	69
PERMISSION LETTER.....	Error! Bookmark not defined.
ETHICS COMMITTEE LETTER.....	Error! Bookmark not defined.
PLAGIARISM REPORT	72

LIST OF TABLES

Table 1: Descriptive statistics of age.....	30
Table 2: Cross tabulation of NIV success and disease of patients	37
Table 3: Test of Normality.....	40
Table 4: Systolic Blood Pressure (BP) Over Time.....	41
Table 5: Diastolic Blood Pressure (BP) Over Time	42
Table 6:Respiratory Rate Over Time.....	43
Table 7: Temperature over time	44
Table 8: SPO ₂ over time	45
Table 9: Expiratory Tidal Volume Over Time.....	46
Table 10: Expiratory Minute Ventilation Over Time	47
Table 11: pH Over Time	48
Table 12: PCO ₂ Over Time	49
Table 13: PaO ₂ over time.....	50
Table 14: HCO ₃ Over Time	51
Table 15: SaO ₂ Over Time.....	52
Table 16: Base Excess (BE) Over Time	53
Table 17: PaO ₂ /FiO ₂ Ratio over Time	54

LIST OF FIGURES

Figure 1 : Histogram of Age(years).....	30
Figure 2: Pie chart of gender distribution	31
Figure 3: Disease of the patients	32
Figure 4: Reason for ICU admission	33
Figure 5: GCS score of patients before admit	34
Figure 6: NIV duration (hours)	35
Figure 7: ICU stays (days)	36
Figure 8: NIV success and disease of patients	37
Figure 9: NIV success.....	38
Figure 10: Complication of NIV	39

LIST OF ABBREVIATIONS

NIV	Non Invasive Ventilation
ICU	Intensive Care Unit
GCS	Glasgow Coma Scale
ARDS	Acute Respiratory Distress Syndrome
COPD	Chronic Pulmonary Obstructive Disease
ABG	Arterial Blood Gases
EMV	Expiratory Minute Ventilation
ETV	Expiratory Tidal Volume
RR	Respiratory Rate
ACPE	Acute Cardiogenic Pulmonary Edema

ABSTRACT

Background: Acute respiratory failure (ARF) is a potentially fatal illness that must be treated as soon as possible. Non-invasive ventilation (NIV) has emerged as a critical modality for treatment ARF because it improves oxygenation, reduces the requirement for invasive mechanical ventilation (IMV), and lowers ICU mortality rates.

Objective: To evaluate the effectiveness of non-invasive ventilation in improving oxygenation and ventilation in patients with acute respiratory failure (ARF).

Methods: This analytical study was conducted at ICU of Integrated Medical Care Hospital over four months. 140 patients were recruited using a non-probability convenient sampling method. Patients aged 20 to 90 who had Type I or Type II respiratory failure and required NIV assistance were eligible. Patients with severe neurological impairment, facial damage, or progressing neuromuscular disease were all excluded. Data on arterial blood gases (ABGs), respiratory rate, and treatment success were used to assess the efficacy of NIV. For data analysis SPSS 22.0 version was used.

Results: A total of 140 patients 52.14% males, 47.86% females with a mean age of 64.78 ± 8.984 years were included. COPD patients were (49.29%), Acute cardiogenic pulmonary edema (13.57%), while ARDS was (15.7%) and Pneumonia were of (21.43%). Non-Invasive Ventilation (NIV) in 108 patients (77.14%) experienced NIV success, while 32 patients (22.66%) did not. The association between disease type and NIV success statistically significant ($p < 0.015$).

Conclusion: This study concluded that, despite noted therapeutic improvements, NIV has a significant success rate in critically ill patients. NIV has significant improvements in tachypnea, tachycardia, drowsiness, SpO₂ and arterial blood gases parameters.

Key words: ARDS, COPD, NIV, Ventilation

Chapter 1: INTRODUCTION

Acute respiratory failure (ARF) is a life-threatening medical emergency, occurring when the respiratory system fails to maintain gas exchange, leading to hypoxemia (inadequate oxygen delivery to the tissues) or hypercapnia (inadequate carbon dioxide removal). This deluge floods cells causing organ dysregulation leading to organ failure and ultimately death if not rapidly corrected. Acute Respiratory Failure (ARF) accounts for a considerable part of the healthcare burden as it is among the leading causes of admission to Intensive Care Units (ICUs) globally, with substantial morbidity and mortality rates. Medically, the severity of ARF depends on its underlying etiology, the physiological reserve of the patient, and the timeliness of medical intervention. ARF can occur from a myriad of conditions that affect the lungs, heart, nervous system, and systemic circulation. The most common causes of respiratory failure are pulmonary diseases i.e. pneumonia, chronic obstructive pulmonary disease (COPD), an exacerbation of asthma, interstitial lung disease and acute respiratory distress syndrome (ARDS) Lung infections, such as bacterial and viral pneumonia (severe influenza and severe COVID-19), can induce diffuse inflammation, shunting gas exchange in alveoli, and significant oxygenation defects (1).

ARDS, can often follow sepsis, trauma, or aspiration, leads to diffuse alveolar damage, increased pulmonary capillary permeability, and severe hypoxemia unresponsive to high doses of oxygen therapy. Like pulmonary disorders, cardiovascular diseases, notably CHF and cardiogenic pulmonary edema, contribute most to the pathogenesis of ARF. Fluid may build up in the lungs when the heart is unable to pump blood properly, interrupting the exchange of oxygen and leading to breathing difficulty. This condition, known colloquially as "fluid overload" or "pulmonary congestion," leads to increased resistance of the airways and reduced compliance of the lungs, rendering it increasingly difficult for the patient to breathe. Conditions affecting the peripheral nervous system (e.g., myasthenia gravis, Guillain-Barre syndrome) as well as prion-related diseases (e.g., amyotrophic lateral sclerosis [ALS]) and spinal cord injuries can also lead to ARF by weakening respiratory muscles, decreasing lung expansion, and reducing the ability to clear secretions. In these situations, the inability to produce sufficient inspiratory

effort causes progressive hypoventilation and hypercapnia, warranting ventilatory support (2).

Infections, especially systemic infections, are among the most common causes of ARF, provoking ARF in this setting through systemic infection and, thus, systemic inflammation, increased metabolic demands, and endothelial dysfunction, resulting in the clinical picture of sepsis and septic shock. During sepsis, inflammatory cytokines contribute to increased capillary leakage, pulmonary edema and acute lung injury (ALI). Sepsis-induced ARF In critically ill patients, sepsis-induced ARF frequently progresses to ARDS, with features of severe hypoxemia, bilateral lung infiltrates and decreased lung compliance. Likewise, metabolic derangements, such as diabetic ketoacidosis, renal failure, and hepatic failure, can worsen respiratory failure via changes in acid-base balance (which affects respiratory rate), changes in respiratory drive, and fluid retention in the lung. Early detection of ARF typically occurs when patients present with unexplained respiratory symptoms, making timely treatment crucial for improving patient outcomes due to the varied nature of conditions that can cause ARF; thus early detection via assessment is paramount. ARF can progress rapidly, and the prompt diagnosis and treatment is critical to avoid irreversible organ damage (3).

They classically present with symptoms of severe dyspnea, tachypnea, cyanosis, altered mental status, and use of accessory respiratory muscles. In advanced cases, respiration fatigue results in poor ventilation with carbon dioxide retention leading to respiratory acidosis. End of the compensation mechanisms: increased respiratory effort, sympathetic activation, and vasodilation can temporarily supply the oxygenation demand, but it becomes worse and the patient may need a respiratory failure with urgent start of ventilatory support. Some diagnostic modalities like arterial blood gas (ABG) analysis, imaging studies, pulmonary function tests are all used in suspected cases of ARF because of the serious event. The medical importance of ABG interpretation involves determining between hypoxemic (Type I) and Hypercapnic (Type II) respiratory failure. The assessment of underlying pathologies such as pneumonia and pulmonary edema and pleural effusions can be possible through imaging

studies such as chest X-rays combined with computed tomography (CT) scans and lung ultrasounds. Respiratory outcomes and care approaches can be obtained through pulmonary function testing in combination with bedside spirometry results (4).

NIV has developed into a new treatment option for ARF because it provides the potential to lower specific complications caused by invasive mechanical ventilation (IMV). Positive pressure ventilation through NIV systems applies either nose prongs or masks which helps prevent complications such as pneumothorax and VAP as well as aspiration of gastric content and inhospitable airway trauma and tracheal stenosis events. The principal assessment of NIV success relies on its ability to stop the need for invasive mechanical ventilation (IMV) together with its related negative outcomes. This intervention leads to shorter durations of ventilation support and ICU stay as well as fewer patient deaths because it provides breathing assistance aside from reducing endotracheal tube impacts. NIV offers two non-invasive ventilation treatments known as CPAP and BiPAP wherein Continuous Positive Airway Pressure (CPAP) provides sustainable airway pressure during the respiratory cycle to preserve airway expansion for better oxygen delivery. The dual pressure levels supplied by BiPAP consist of high IPAP for inspiration followed by lower EPAP for expiration (5).

The outcome of Non-Invasive Ventilation for ARF depends strongly on determining suitable patients and initiating the device at optimal times as well as patient adherence to proper ventilator settings. Multiple scientific papers show NIV successfully generates important clinical benefits during COPD exacerbations because it reduces patient mortality rates and intubation requirements. Likewise, NIV has been shown to improve oxygenation and reduce work of breathing in cardiogenic pulmonary edema. In patients with pneumonia or ARDS, the benefits are less definitive; several studies in these populations have reported an increased failure rate. NIV is important to decrease the pressure on ICUs that are close to saturation because NIV can passively prevent the need for invasive mechanical ventilation (6).

Endotracheal intubation, which can be lifesaving in severe cases, carries with it a litany of complications, including ventilator-associated pneumonia, barotrauma and longer stays in ICUs. We know that the NIV use decreases the risk of the above-mentioned complications as it is an intermediate respiratory support technique compared with IMV.

At a physiological level, NIV increases alveolar ventilation through increasing tidal volume and decreasing work of breathing . Although this is particularly helpful in the setting of hypercapnic respiratory failure in which retention of CO₂ can lead to a vicious cycle of increasingly worsening acidosis and respiratory distress it can also be used in patients with hypoxemia with sufficient clinical suspicion .As a consequence of enhanced gas exchange, NIV improves pH balance and prevents the patient from requiring more invasive treatment. Besides, positive pressure ventilation with NIV recruits collapsed alveoli, improves oxygenation and diminishes the ventilation-perfusion mismatches typical of ARF (7).

NIV success is determined by several factors such as the degree of respiratory failure, the level of patient cooperation, and the clinician's experience with titration of ventilatory settings. Early initiation of NIV has shown improved outcomes, while late intervention leads to start-up failures followed by intubation. Decision regarding to continue or escalate ventilatory support is guided by clinical parameters including respiratory rate, blood gas analysis and patient's tolerance (8). Advantages of NIV aside, it is not always successful. Certain subgroups, especially those with severe hypoxemia or those with altered mental status, have high failure rates. However, prolonged application of NIV in such situations may postpone essential intubation, possibly resulting in poor clinical outcomes. Hence, ongoing evaluation and reassessment are paramount to providing patients the appropriate modality of respiratory support (9). The effect of NIV on mortality and ICU length of stay has been inconsistent among studies. NIV treatment has become part of our routine practice, but while some studies show its clear benefit in specific populations, we also know its limitations, especially in situations where the respiratory failure worsens despite non-invasive management. Higher levels of oxygen needs and enhanced respiratory secretions combined with fatigue of respiratory muscles serve as failure indicators for NIV while influencing nurse decision-making for patient selection (10).

Medical researchers show increasing interest in NIV as an alternative therapy for treating acute respiratory failure over IMV due to its effectiveness yet this efficacy remains under investigation. Its benefits including better oxygenation and shorter ICU stays as well as lower endotracheal intubation frequency require deeper research

because current patient outcome variability remains an issue. For optimizing clinical implementation it is essential to know which factors determine NIV success because patient selection criteria and time of initiation influence the outcomes. It is essential to conduct a comprehensive evaluation of NIV in ARF care since clinical studies produce ambiguous results. The study aims to bridge an existing research gap through organized analysis of how NIV affects respiratory function and decreases medical complications in patients with ARF.

AIM AND OBJECTIVES

- To examine the effectiveness of non-invasive ventilation in patients with acute respiratory failure admitted in intensive care unit.

Chapter 2: LITERATURE REVIEW

Acute hypoxemic respiratory failure (AHRF) requires proper respiratory support techniques because this critical condition commonly occurs within intensive care units (ICUs). The interest in non-invasive ventilation (NIV) as an alternative to invasive mechanical ventilation (IMV) increases because this method enhances oxygenation levels and decreases the risks associated with intubation. Through the synthesis of results from recent observational and cross-sectional studies, this literature review investigates the efficacy of NIV in ICU patients with AHRF (11).

The research by Schmid et al. (2022) evaluated the NIV therapeutic approach for individuals with COVID-19 AHRF who received care in intensive care units. Their research demonstrated that NIV which combines the methods HFNC with CPAP managed to enhance oxygenation and reduced the requirement for intubation. The researchers highlighted superior patient outcomes could be achieved through early initiation of NIV administration compared to delayed initiation. The joint use of NIV with awake prone positioning led to better oxygenation levels thus reducing patients' intensive care unit stay duration (12).

Chaudhuri et al. conducted a research study in 2022 that studied HFNC and NIV through a cross-sectional investigation among intensive care unit patients. The authors demonstrated that clinical factors of respiratory distress level and oxygenation state and patient acceptance served as deciding factors between these modalities. Research showed that patients with moderate-to-severe AHRF received better comfort from HFNC although NIV resulted in major reductions in intubation requirements. The research demonstrated NIV maintains patients with extreme hypoxemia from worsening even when HFNC functions best for patients with moderate conditions (13).

Sakuraya et al (2021) performed an observational study that analyzed invasive and non-invasive ventilation approaches which treat critically ill ICU patients experiencing AHRF. The research showed NIV decreased intubation demands in select cases but patients handled worsened respiratory failure needed speedily moving to IMV. Patient outcomes show evidence of negative impact from delayed improper intubation procedures because of which healthcare providers need to focus on tailoring respiratory

support (14).

The study conducted by Ferreyro et al. (2020) investigated NIV outcomes through retrospective cross-sectional analysis of ICU patients. The study demonstrated NIV successfully reduced mortality risk compared to standard oxygen therapy methods mainly among COPD patients with acute cardiogenic pulmonary edema. Delaying intubation procedures among patients who experienced NIV failure led to raised mortality rates. Proper screening must be maintained to detect early warning signals of NIV system failure (15). The study conducted by Kondo et al. (2017) evaluated NIV treatment effects for ICU patients diagnosed with AHRF. The review determined NIV decreased hospital admission rates and death risks in Intensive Care Unit-based patients thus proving its effectiveness in suitable candidates. The research examined multiple patient groups and revealed that subjects with obesity hypoventilation syndrome showed the most positive outcomes from NIV treatment (16).

Researchers from Luo et al. (2016) performed a multi-center observational research analysis of NIV versus IMV for immunocompromised ICU patients. The survival rates and cases of ventilator-associated pneumonia (VAP) proved better with NIV than IMV according to their study data which demonstrated NIV's position as an initial intervention choice. The study becomes crucial for immunocompromised patients because preventing infections related to IMV treatment stands as their main objective (17). Beauchemin (2016) investigated NIV treatment effects for ICU patients experiencing acute COPD exacerbations. Research data displayed significant decreases in both intubation rates as well as ICU mortality which confirms NIV should be used routinely when treating this group of patients. NIV initiation during COPD exacerbations proved most successful when begun early instead of waiting longer periods according to their research (18).

The research by Pandor et al. (2015) examined how helmet NIV performed relative to face mask NIV for treating ICU patients with acute respiratory distress syndrome (ARDS). This study established that patients benefited from helmet NIV since it led to less intubations and better oxygenation results than face mask NIV while providing superior airway pressure and comfort. Air leaks through the interface system decreased

simultaneously with better non-invasive ventilation efficiency (19). The research by Masip et al. (2014) analyzed the consequences of NIV for ICU patients diagnosed with acute cardiogenic pulmonary edema. A cross-sectional research by Masip et al. (2014) showed NIV produced considerable decreases in mortality rates together with intubation requirements thus proving itself fundamental for pulmonary edema treatment. The research demonstrated BiPAP provided extra advantages to people suffering from hypercapnia since CPAP and BiPAP each showed beneficial effects (20).

Botker et al. (2014) conducted an observational research to compare HFNC and NIV in ICU patients with AHRF. The study researchers found that HFNC presented itself as an effective treatment possibility which was suitable for patients who could not handle conventional NIV. Patient compliance was identified as the key element determining which treatment would be selected according to this study (21). According to Goodacre et al. (2014) pre-hospital CPAP and BiPAP demonstrated effective use in emergency transport circumstances. Early use of non-invasive ventilation by healthcare providers reduced ICU hospitalizations and death rates and decreased invasive ventilation requirements when patients arrived at the hospital. Medical experts affirmed that emergency management protocols should include NIV use before hospital admission (22).

The research by Chiumello et al. (2013) evaluated the utilization of NIV for treating ICU patients who experienced AHRF due to chest trauma. The research showed that NIV usage effectively cut intubation needs and fatal outcomes while showing its essential value for medical cases that need minimal invasive treatments. Early application of NIV treatment led to better thoracic compliance as well as decreased the possibility of developing ventilator-associated medical issues (23). Antonelli et al. (2000) carried out a research study comparing NIV to conventional mechanical ventilation among ICU patients with AHRF. The investigators demonstrated NIV effectively led to reduced VAP occurrences together with better survival outcomes which supports its value in replacing prolonged need for IMV. The initial study established principles which led medical practitioners to increase NIV applications throughout critical care facilities (24).

The literature about acute respiratory failure (AHRF) demonstrates how NIV reduces both intubation requirements and death and intensive care unit stays. The current state of medical knowledge does not provide sufficient answers about how to choose ideal patients for ventilator support or when to initiate the intervention or what benefits different AHRF causes can derive from this therapy. Study evidence demonstrates NIV benefits but research produces contradictory results when evaluating its effects on ARDS patients along with patients with weakened immune systems. Research needs to advance regarding the comparison of NIV modalities CPAP, BiPAP and HFNC across different intensive care units. Additional extensive trials must be conducted to develop standardized procedures for NIV application in AHRF management.

OPERATIONAL DEFINITIONS

A. Acute Respiratory Failure (ARF)

Acute respiratory failure (ARF) emerges as a life-threatening condition because it causes the respiratory system to lose its capability to maintain gas exchange thus resulting in hypoxemia ($\text{PaO}_2 < 60\text{mmHg}$) and hypercapnia ($\text{PaCO}_2 > 45\text{mmHg}$) and $\text{PH} < 7.35$. ARF exists as a range of diseases which show a respiratory system failure to provide sufficient oxygen exchange (25).

B. Non Invasive ventilation (NIV):

Non-invasive ventilation (NIV) refers to the administration of ventilatory support without the need for an invasive artificial airway (such as an endotracheal tube or tracheostomy tube). The ventilation treatment provides support through facial interfaces which cover either the mouth and nose separately or simultaneously. The airway applications of NIV supply positive airway pressure that enhances both oxygenation and carbon dioxide elimination (26).

C. Bi-Level Positive Airway Pressure (BIPAP)

Through Bi-level positive airway pressure (BiPAP) the breathing cycle receives two different positive airway pressure levels by providing inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). The breathing cycle receives its highest pressure level as Inspiratory Positive Airway Pressure (IPAP). The higher IPAP pressure breaks airway resistance which makes it easier for lung air intake. The machine maintains the lower pressure of EPAP throughout exhalation and at each respiratory phase. EPAP maintains airway openness through its function of stopping airways from collapsing which enables better oxygenation and ventilation (27).

D. Continues Positive Airway Pressure (CPAP)

CPAP stands as non-invasive positive pressure ventilation that medical professionals use mainly to treat patients with hypoxemic respiratory failure and obstructive sleep

apnea (OSA) and various respiratory conditions. The therapy provides unchanging positive airway pressure during every stage of respiration (28).

E. Chronic Obstructive Pulmonary disease (COPD)

COPD stands for Chronic Obstructive Pulmonary Disease. The disease affects the lungs progressively when chronic bronchitis combines with airways becoming thicker and individuals meet criteria for emphysema after prolonged contact with noxious particles or gases from tobacco smoking (29).

F. Acute Cardiogenic Pulmonary Edema

Acute cardiogenic pulmonary edema exists as a medical problem where abnormal lung tissue fluid collects because of cardiac system dysfunction. An ineffective blood pump from the heart leads to rising pulmonary vasculature pressure resulting in this condition. The elevated pulmonary pressure forces blood vessel fluid to seep into air sacs (alveoli) rendering them less able to oxygenate blood (30).

G. Acute Respiratory Distress Syndrome (ARDS)

Acute cardiogenic pulmonary edema exists as a medical problem where abnormal lung tissue fluid collects because of cardiac system dysfunction. An ineffective blood pump from the heart leads to rising pulmonary vasculature pressure resulting in this condition. The elevated pulmonary pressure forces blood vessel fluid to seep into air sacs (alveoli) rendering them less able to oxygenate blood. Diagnosis is based on clinical criteria, including the presence of acute onset of respiratory symptoms, bilateral opacities on chest imaging, and evidence of hypoxemia not explained by heart failure or fluid overload (31).

Non Invasive Success:

- Improvement in Shortness of Breath, RR <30 breaths/minute or RR > 10 breaths/minute, SPO2 > 90%, PaO2 > 60 mmHg, PH > 7.35. (32)

Non Invasive Failure:

- No improvement in Shortness of Breath, RR >35 breaths/minute or RR < 10 breaths/minute, SPO2 < 90%, PaO2 < 60 mmHg, PH < 7.35 (33)

Chapter 3: METHODOLOGY

3.1. Research Design:

It was an analytical cross sectional study.

3.2. Clinical Settings

The study was conducted in the Intensive Care Unit (ICU) at Integrated Medical Care Hospital DHA Phase 5, Lahore.

3.3. Sample Size

The sample size was calculated by using the formula for quantitative cross sectional study. And also on the basis of the study of (13) by using 90% proportion of NIV success, with an absolute error of 5% and a type 1 error at 0.05, the minimum sample size is 140. (34)

The sample size is determined by using the formula:

$$n = \frac{Z^2 \times p \times (1-p)}{d^2}$$

Where:

n = required sample size.

Z = Z-score corresponding to the desired level of significance (e.g., 1.96 for alpha = 0.05).

p = expected proportion (effect size)

d = margin of error (typically expressed as a proportion, e.g., 0.05 for a 5% margin of error).

3.4. Sampling Technique

Non probability convenient sampling technique was used in this study.

3.5. Duration of Study

The study was conducted for 4 months in Integrated Medical Care Hospital, DHA Phase 5 Lahore.

3.6. Selection Criteria

3.6.1. Inclusion Criteria

- Patients admitted to ICUs

- Age 20-90 years.
- Patients receiving non-invasive ventilation through a mask interface that covers the nose or mouth.
- Patients having Hypoxemic Respiratory (Type I) Failure $\text{PaO}_2 < 60\text{mmHg}$.
- Patients having Hypercapnic Respiratory (Type II) Failure $\text{PaCO}_2 > 45\text{mmHg}$ and $\text{PH} < 7.35$.
- Patients having Shortness of breath and $\text{RR} > 35$ breaths/minute or $\text{RR} < 10$ breaths/minute.

3.6.2. Exclusion Criteria

- Patients with severe brain trauma with a Glasgow Coma Scale < 8 .
- Patients with facial trauma.
- Patients having cancer of oropharynx region.
- Patients with progressive neuromuscular disease.
- Patient receiving Invasive Mechanical Ventilation through Endotracheal Tube.

3.7. Ethical Consideration

a) Informed Consent: Participants (patient) and their legal heir must be provided with clear and concise information about the study, including its purpose and benefits, and their right to withdraw at any time. The informed consent document should be written in understandable language and address any questions participants may have.

b) Confidentiality and Privacy: Will maintain participant's confidentiality by ensuring all data is anonymized and securely stored.

c) Ethical Approval: The ethical approval was obtained from the Institutional Review Board (IRB) of Superior University before commencing the study. The IRB reviewed the research proposal to ensure it adheres to ethical guidelines and protects participant rights.

3.8. Data Collection Procedure

Data was collected through modified questionnaire. Demographic and clinical characteristics of patients including age, sex, length of hospital and ICU stay and duration of non-invasive ventilation were recorded.

3.9. Data Analysis

Statistical analysis of was performed by using IBM SPSS version 22.0. For descriptive statistics categorical variable was presented as frequency distribution and percentages while continuous variables were presented as mean, median, standard deviation and inter quartile range. For inferential statistics, the independent t-test and Chi-square test was used to evaluate any relation between categorical variables

Chapter 4: RESULTS

A total of 140 patients 52.14% males, 47.86% females with a mean age of 64.78 ± 8.984 years were included. COPD patients were (49.29%), Acute cardiogenic pulmonary edema (13.57%), while ARDS was (15.7%) and Pneumonia were of (21.43%). The median NIV duration was between 11 to 16 hours, and ICU stays were most frequently 3 to 4 days. Non-Invasive Ventilation (NIV) in 108 patients (77.14%) experienced NIV success, while 32 patients (22.66%) did not. The association between disease type and NIV success statistically significant ($p < 0.015$).

Intubation was required in 22.66% of cases, and 37% of patients experienced complications. The overall mortality rate was 22.66%. Tests of normality indicated that key physiological and arterial blood gas parameters were non-normally distributed, necessitating non-parametric analysis. A downward trend in respiratory rate and an increase in oxygen saturation and tidal volume over two days, these changes reached statistical significance.

Acid-base balance improved, with pH increasing from 7.2185 to 7.3474 and PCO₂ decreasing from 65.10 mmHg to 46.64 mmHg, reflecting better ventilation, changes were statistically significant. Systolic and diastolic blood pressure remained stable, and fluctuations in temperature were seen. Overall, while clinical improvements were observed, statistical significance was reached in most parameters.

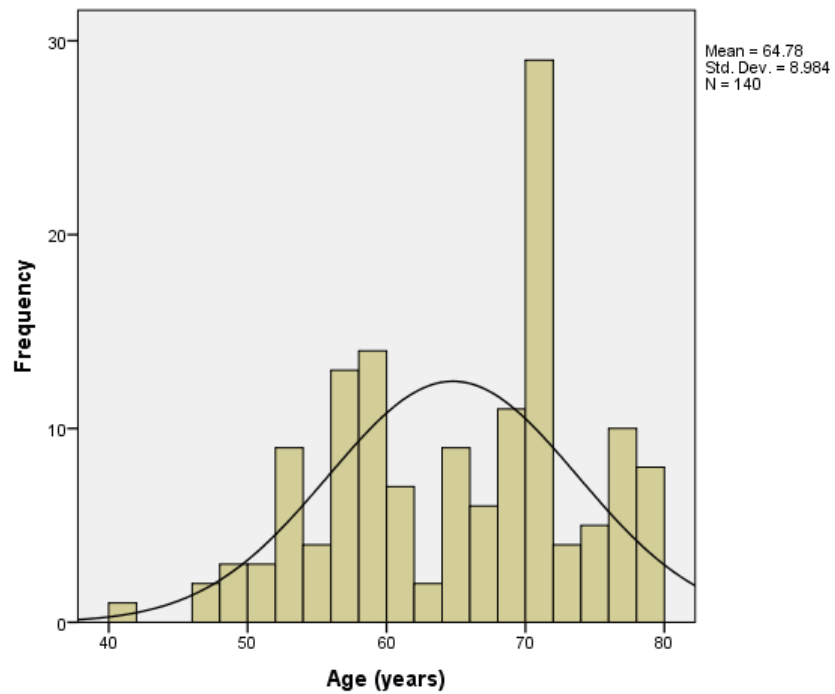


Figure 1 : Histogram of Age(years)

Table 1: Descriptive statistics of age

Descriptive Statistics					
	N	Minimum	Maximum	Mean	Std. Deviation
Age (years)	140	41	79	64.78	8.984

The above table and histogram shows the descriptive statistics for age of patients. The minimum was 41 years, while the maximum age was 79 years. The mean $\pm$ SD age of the patients was 64.78 $\pm$ 8.984 years.

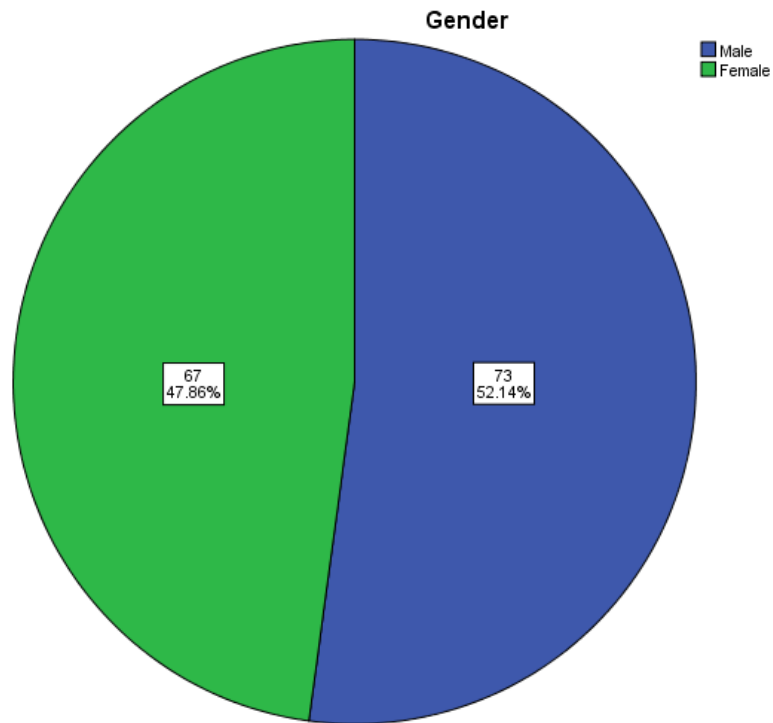


Figure 2: Pie chart of gender distribution

The bar chart shows the gender distribution of the participants. Males accounted for 73 individuals (52.14%), while females comprised 67 individuals (47.86%).

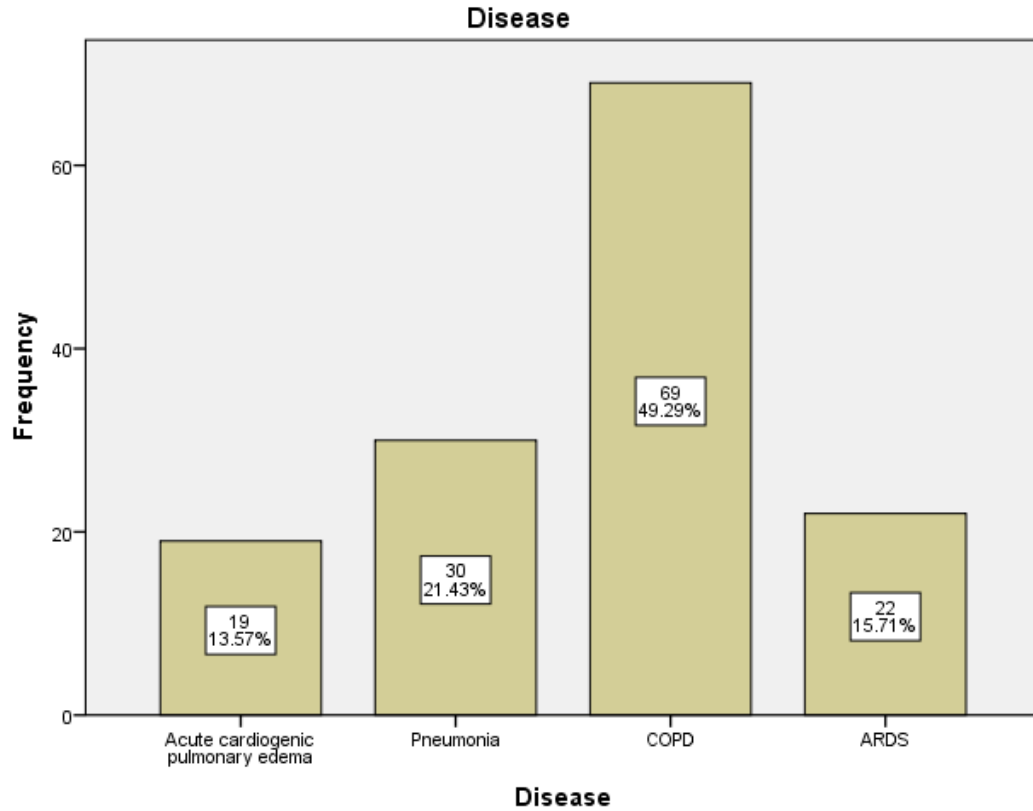


Figure 3: Disease of the patients

The distribution of diseases among patients shows that COPD was the most common condition, affecting 69 individuals (49.29%). Acute cardiogenic pulmonary edema with 19 patients (13.57%), while acute respiratory distress syndrome (ARDS) was present in 22 cases (15.7%). Pneumonia with 30 patients (21.43%).

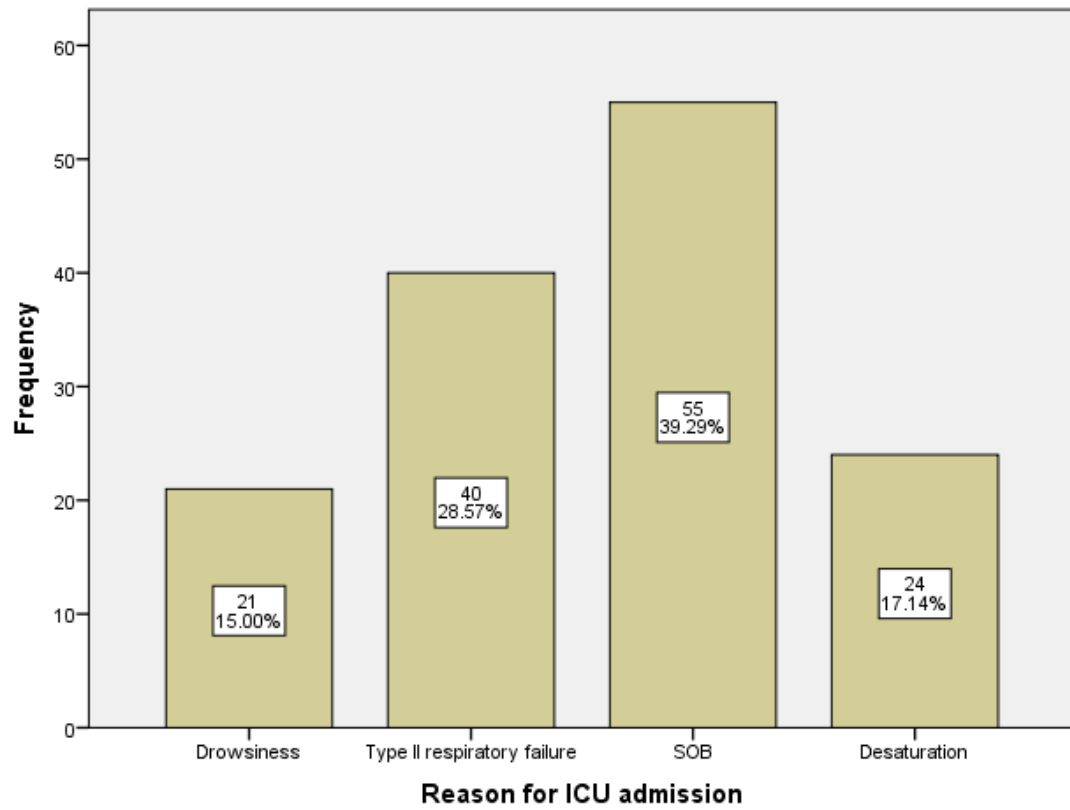


Figure 4: Reason for ICU admission

The table presents the reasons for ICU admission, with shortness of breath (SOB) being the most common cause (39.3%), followed by type II respiratory failure (28.6%), desaturation (17.1%), and drowsiness (15.0%).

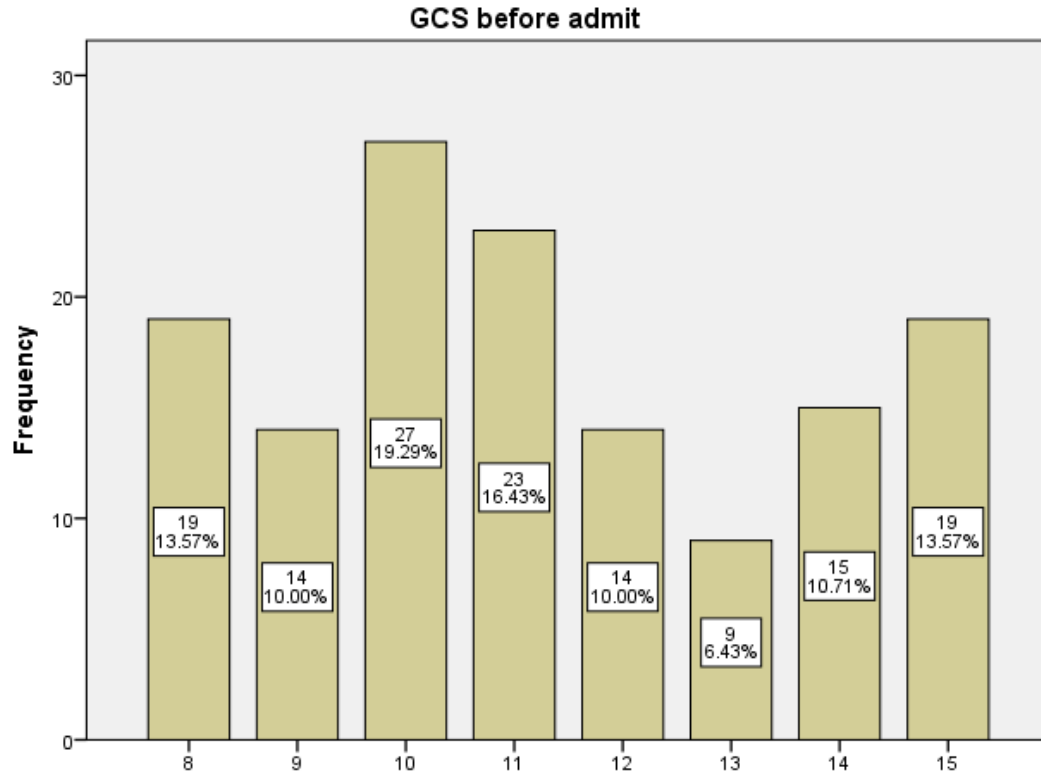


Figure 5: GCS score of patients before admit

The Glasgow Coma Scale (GCS) distribution before admission shows that the highest number of patients had a GCS score of 10, with 27 individuals (19.29%). This is followed by a GCS score of 11, recorded in 23 patients (16.43%). Both GCS scores of 8 and 15 had the same frequency, with 19 patients each (13.57%). Meanwhile, GCS scores of 9, 12, were observed in 14 patients each (10.00%) and 15(10.71%). The lowest frequency was noted for a GCS score of 13, with only 9 patients (6.43%).

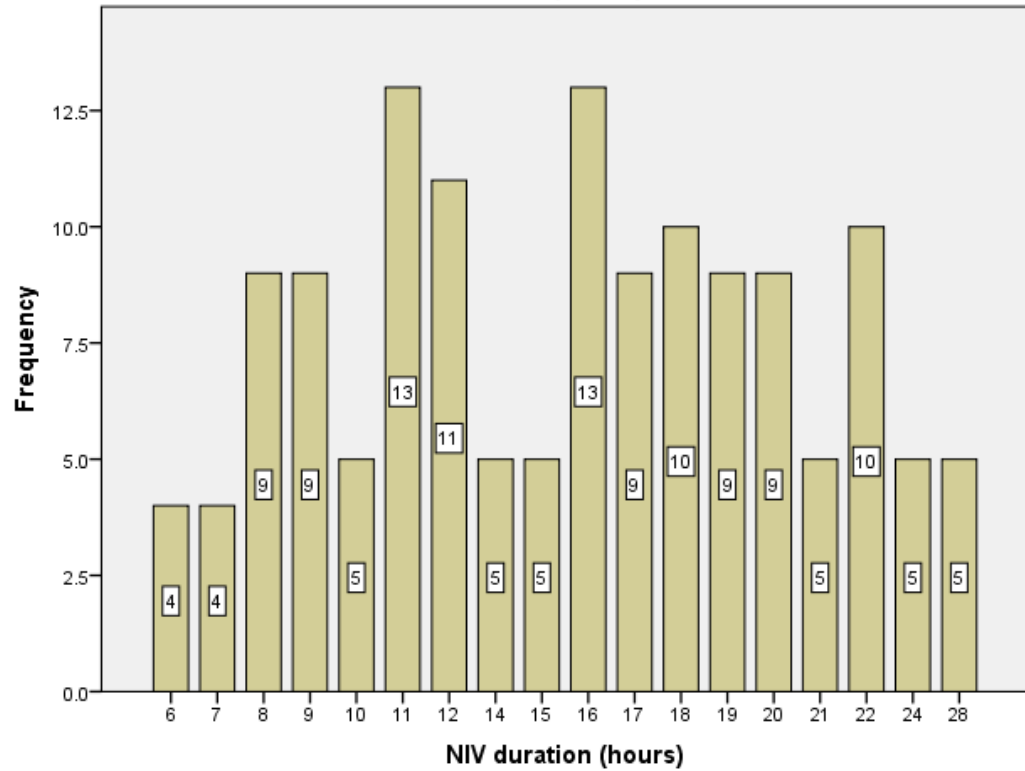


Figure 6: NIV duration (hours)

The bar chart presents the frequency distribution of non-invasive ventilation (NIV) duration in hours among patients. The data shows that NIV durations are widely distributed, with peaks around 11,12,16,18, 22 hours. Other common durations include 8,9, 19,20 hours. There are fewer cases with NIV durations exceeding 24 hours.

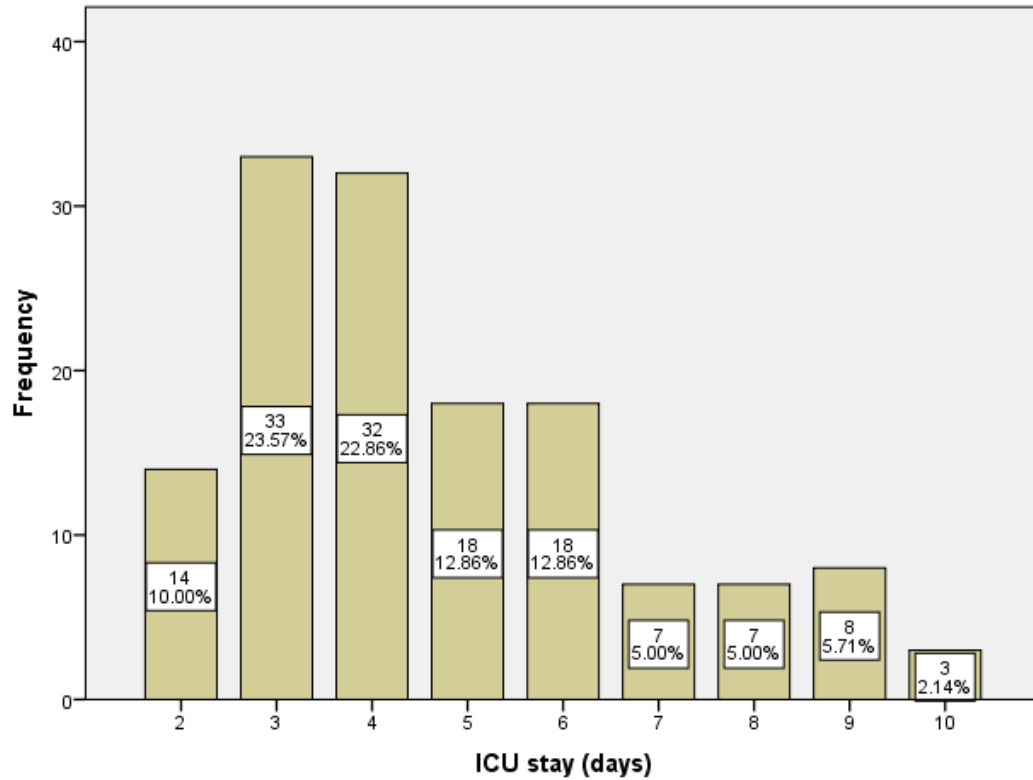


Figure 7: ICU stays (days)

The bar chart illustrates the frequency distribution of ICU stay durations (in days) among patients. The x-axis represents the number of ICU stay days, while the y-axis indicates the frequency of patients. The most common ICU stays were 3 and 4 days, with 33 (23.57%) and 32 (22.86%) patients, respectively. ICU stays of 5 and 6 days were also relatively frequent, accounting for 12.86% and 12.86% of cases. Longer ICU stays (7–10 days) were less common, with frequencies gradually decreasing, and only 3 patients (2.14%) staying for 10 days.

Table 2: Crosstabulation of NIV success and disease of patients

NIV success * Disease Cross tabulation							
		Disease				Total	Chi square
		COPD	Acute cardiogenic pulmonary edema	Pneumonia	ARDS		
NIV success	Yes	60	14	22	12	108	.015
	No	9	5	8	10	32	
Total		69	19	30	32	140	

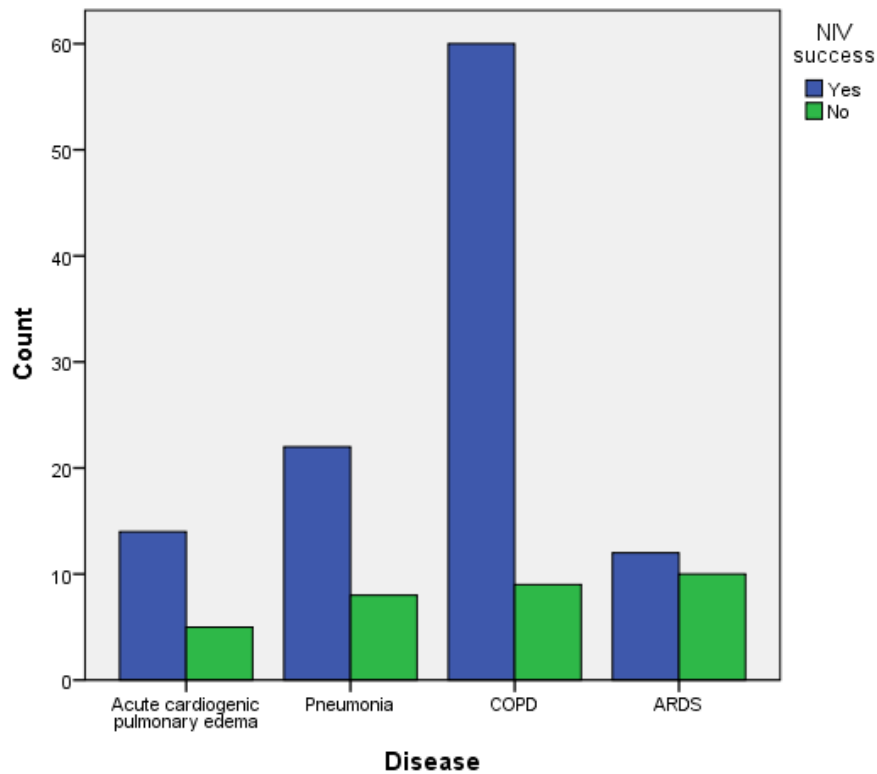


Figure 8: NIV success and disease of patients

The table presents the association between different diseases and the success of non-invasive ventilation (NIV). COPD patients had the highest NIV success rate (60 out of 69), followed by pneumonia (22 out of 30), acute cardiogenic pulmonary edema (14 out of 19), and ARDS (12 out of 32). The chi-square test result ($p < 0.015$) suggests a significant relationship between disease type and NIV success.

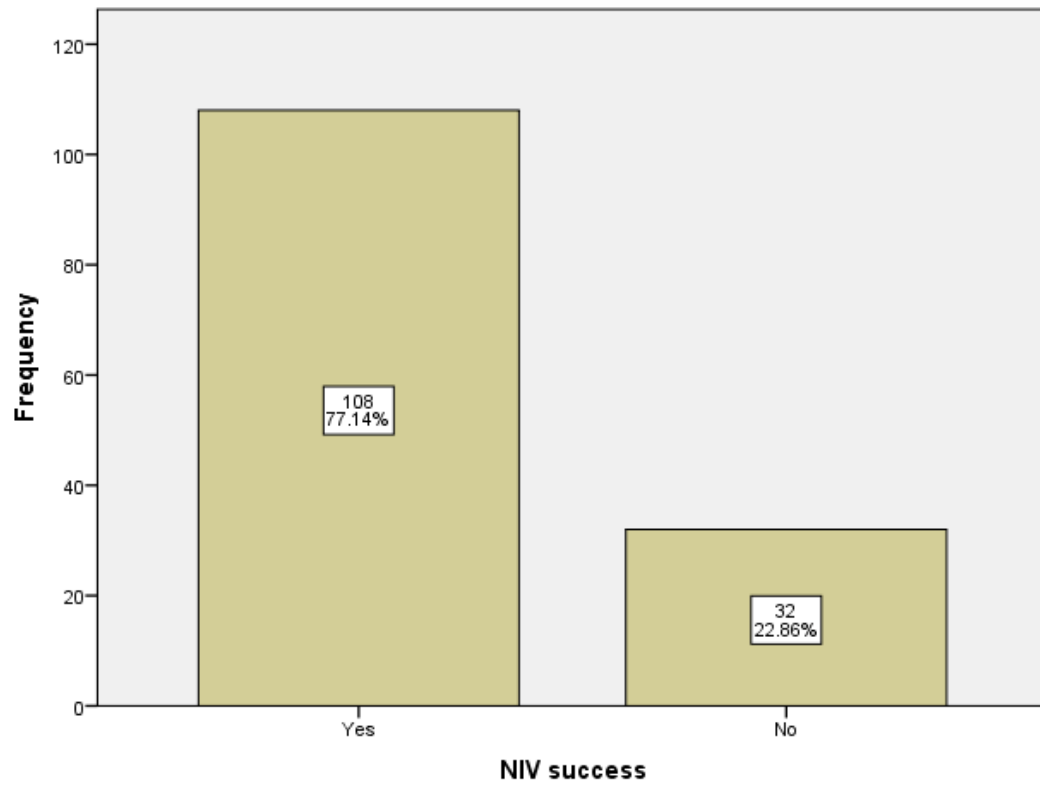


Figure 9: NIV success

The bar chart represents the frequency of Non-Invasive Ventilation (NIV) success cases. It shows that 108 patients (77.14%) experienced NIV success, while 32 patients (22.66%) did not.

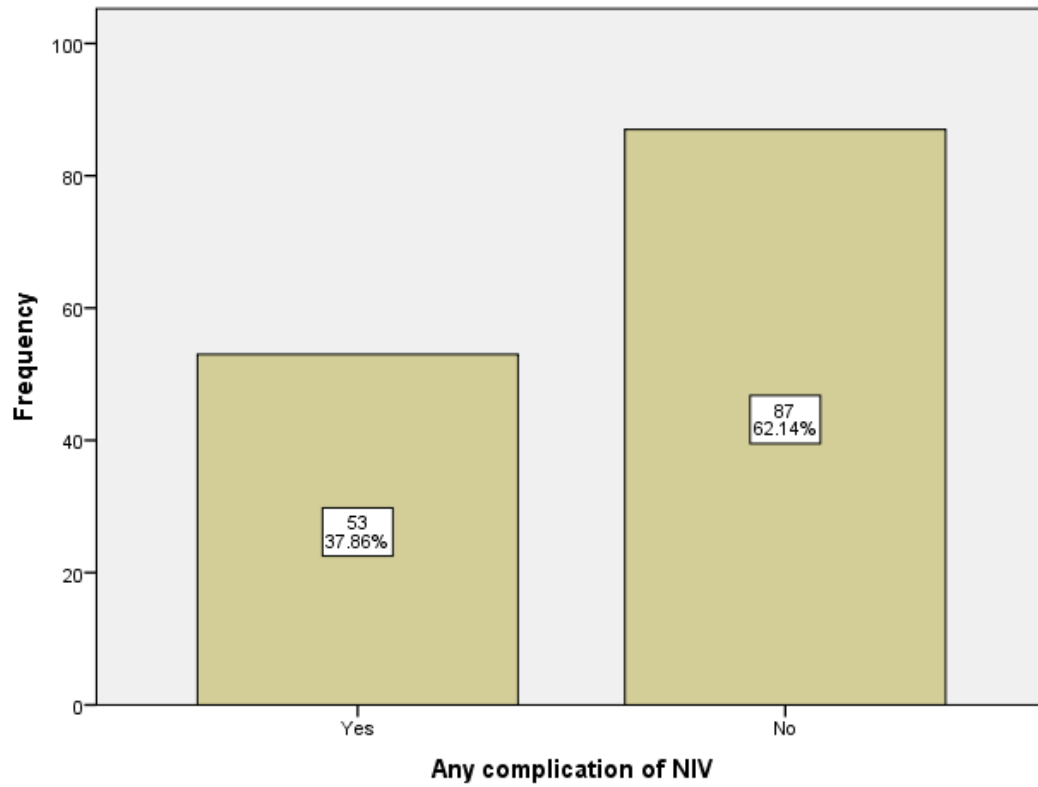


Figure 10: Complication of NIV

The bar chart presents the frequency of complications associated with non-invasive ventilation (NIV). A total of 37.86% (n = 53) of patients experienced complications, while 62.14% (n = 87) did not.

Table 3: Test of Normality

Tests of Normality			
	Kolmogorov-Smirnov^a		
	Statistic	df	Sig.
Systolic BP Initial	.140	140	.000
Diastolic BP initial	.157	140	.000
Respiratory rate initial	.124	140	.000
Temperature initial	.271	140	.000
SPO2 initial	.220	140	.000
Expiratory Tidal Volume Initial	.228	140	.000
Expiratory Minute Ventilation initial	.220	140	.000
Respiratory rate on NIV initial	.223	140	.000
Peak Pressure Initial	.257	140	.000
Leak percentage Initial	.202	140	.000
pH initial	.126	140	.000
PCO2 initial	.244	140	.000
PaO2 initial	.207	140	.000
HCO3 initial	.168	140	.000
SaO2 initial	.154	140	.000
BE initial	.206	140	.000
PaO2/FiO2 initial	.202	140	.000
a. Lilliefors Significance Correction			

This table presents the normality test results for key physiological parameters and ABGs on ventilation. The significance values ($p < 0.05$) indicate that these variables do not follow a normal distribution, necessitating non-parametric statistical analysis.

Table 4: Systolic Blood Pressure (BP) Over Time

Kruskal Wallis Test								
	N	Mean	Std. Dev	P value	Chi square	Percentiles		
						Q₁	Q₃-Q₁	Q₃
Systolic BP Initial	140	117.83	26.285	.384	.974	95.00	115.00	135.0
Systolic BP after 2 hours	140	118.59	21.342	.341	.719	102.00	122.00	128.0
Systolic BP after 1 day	140	118.01	13.978	.129	.559	109.00	119.00	125.0
Systolic BP after 2 day	140	118.09	12.911	.001	.536	110.00	120.00	123.0

The systolic BP remained relatively stable over time, with chi-square values ranging from .974 to .536 and p-values < 0.05, indicating significant difference. This shows that NIV had major impact on systolic BP during the period.

Table 5: Diastolic Blood Pressure (BP) Over Time

Kruskal Wallis Test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						Q₁	Q₃-Q₁	Q₃
Diastolic BP initial	140	76.81	14.940	.969	.483	67.00	79.00	89.00
Diastolic BP after 2 hours	140	76.03	12.372	.248	.237	74.00	78.00	83.75
Diastolic BP after 1 day	140	75.37	9.954	.223	.118	72.00	76.00	80.00
Diastolic after 2 day	140	74.57	11.381	.166	.025	70.00	74.00	82.00

Diastolic BP showed minimal variation, with chi-square values between 0.969 and 1.66 and p-values < 0.05, suggesting statistically significant difference. These results indicate that NIV therapy significantly alter diastolic BP.

Table 6:Respiratory Rate Over Time

Kruskal Wallis Test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						Q₁	Q₃-Q₁	Q₃
Respiratory rate initial	140	30.79	5.357	.744	.107	28.0 0	31.00	35.0
Respiratory rate after 2 hours	140	27.39	5.108	.766	.088	23.0 0	28.00	31.0
Respiratory rate after 1 day	140	25.05	5.307	.932	.007	20.0 0	28.00	30.0
Respiratory rate after 2 day	140	24.19	5.456	.966	.000	18.0 0	26.00	29.0

The mean respiratory rate decreased from 30.79 breaths/min initially to 24.19 breaths/min at 2 **days**, indicating a clinical trend of improved ventilation. However, the chi-square values (.744 to .966) and p-values <0.05 indicate statistically significant difference.

Table 7: Temperature over time

Kruskal Wallis Test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						Q ₁	Q ₃ -Q ₁	Q ₃
Temperature initial	140	98.024	2.318	.7190	1.086	96.500	96.800	98.600
Temperature after 2hours	140	95.271	1.718	.678	.172	97.000	97.500	100.00 0
Temperature after 1 day	140	94.783	1.069	.425	.129	96.975	97.550	98.325
Temperature after 2 day	140	91.699	1.061	.297	.028	96.900	97.200	98.000

Temperature remained stable, with chi-square values 0.7190 to 0.297 and p-values < 0.05, indicating significant difference over time. This shows that NIV therapy causes substantial temperature related changes.

Table 8: SPO₂ over time

Kruskal Wallis Test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						Q ₁	Q ₃ -Q ₁	Q ₃
SPO₂ initial	140	86.36	5.215	2.606	.373	85.00	88.00	90.00
SPO₂ after 2 hours	140	93.56	3.826	1.303	.106	88.00	94.00	96.00
SPO₂ after 1 day	140	94.52	3.639	.794	.098	91.00	95.00	98.00
SPO₂ after 2 day	140	95.52	3.423	.335	.042	94.00	97.00	98.00

The mean SpO₂ increased from 86.3% to 95.52% over 2 days, reflecting better oxygenation. However, chi-square values 2.606 to 0.335 and p-values < 0.05 indicate statistically significant difference, despite the clinical improvement.

Table 9: Expiratory Tidal Volume Over Time

Kruskal Wallis Test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						Q ₁	Q ₃ -Q ₁	Q ₃
Expiratory Tidal Volume on ventilation Initial	140	385.04	34.782	.814	.215.	360.25	397.50	400.00
Expiratory Tidal Volume on ventilation after 2 hours	140	380.96	42.320	.782	.183	342.25	406.50	409.75
Expiratory Tidal Volume on ventilation after 1 day	140	412.27	27.984	.669	.073	392.25	422.00	425.00
Expiratory Tidal Volume on ventilation after 2 day	140	414.59	24.689	.587	.000	401.25	418.00	420.00

The mean expiratory tidal volume increased from 385.04 mL to 414.59 mL, with chi-square values .814 to .587 and p-values < 0.05, indicating significant difference over time. This suggests stable ventilation patterns during NIV.

Table 10: Expiratory Minute Ventilation Over Time

Kruskal Wallis test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						25th	Q₃-Q₁	75th
Expiratory Minute Ventilation initial	140	15.32	4.219	.897	.218	13.50	14.50	16.50
Expiratory Minute Ventilation after 2 hours	140	15.05	4.241	.732	.188	12.50	14.20	15.20
Expiratory Minute Ventilation after 1 day	140	11.86	3.837	1.519	.154	9.90	11.40	13.40
Expiratory Minute Ventilation after 2 day	140	12.48	4.238	.560	.017	9.20	11.00	13.00

The minute ventilation decreased from 15.32 L/min to 12.48 L/min, showing reduced work of breathing. However, chi-square values 0.017 to 1.519 and p-values < 0.05 suggested significant difference over time.

Table 11: pH Over Time

Kruskal Wallis Test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						Q ₁	Q ₃ -Q ₁	Q ₃
pH initial	140	7.2134	.14113	.852.	.412	7.115	7.2350	7.2900
pH after 2 hours	140	7.2963	.11370	.569	.251	7.225	7.3500	7.3710
pH after 1 day	140	7.3410	.13984	.192	.156	7.312	7.3950	7.4260
pH after 2 day	140	7.3478	.17030	.705	.057	7.368	7.3950	7.4110

The mean pH improved from 7.2185 to 7.3474, indicating a shift towards normal acid-base balance. However, with chi-square values 0.852 to 0.705 and p-values < 0.05, the change was statistically significant.

Table 12: PCO₂ Over Time

Kruskal Wallis Test								
	N	Mean	Std. Dev	Chi square	P value	Percentiles		
						Q ₁	Q ₃ -Q ₁	Q ₃
PCO₂ initial	140	64.7500	16.51027	.701	.547	51.0000	57.0000	70.0000
PCO₂ after 2hours	140	66.3071	13.82006	.624	.427	59.0000	62.0000	76.0000
PCO₂ after 1 day	140	49.9843	10.84588	.548	.161	40.6000	48.4000	52.5000
PCO₂ after 2 day	140	47.0043	8.90953	.513	.000	39.1000	42.2000	49.3000

PCO₂ levels decreased from 64.75 mmHg to 47.004 mmHg, suggesting improved ventilation. However, chi-square values 0.701 to 0.513 and p-values < 0.05 indicate significant difference over time.

Table 13: PaO₂ over time

Kruskal Wallis Test								
	N	Mean	Std. Deviation	P value	Chi square	Percentiles		
						Q₁	Q₃-Q₁	Q₃
PaO₂ initial	140	64.7500	18.84588	.240	.524	50.7500	55.5000	70.0000
PaO₂ after 2 hours	140	66.3071	8.90953	.140	.625	60.5000	62.0000	76.0000
PaO₂ after 1 day	140	68.7429	12.20825	.039	.844	58.0000	72.0000	78.0000
PaO₂ after 2 day	140	72.5214	18.57687	.001	.979	58.0000	75.0000	86.0000

The mean PaO₂ improved from 64.75 mmHg to 72.52 mmHg, indicating better oxygenation. However, chi-square values 0.624 to 0.240 and p-values < 0.05 suggest statistically significant difference.

Table 14: HCO₃ Over Time

Kruskal Wallis Test								
	N	Mean	Std. Deviation	P value	Chi square	Percentiles		
						Q₁	Q₃-Q₁	Q₃
HCO₃ initial	140	27.7885	9.93748	.054	.962	24.0000	28.9000	35.2000
HCO₃ after 2 hours	140	26.8667	8.54882	.018	.892	23.1000	27.6000	33.5100
HCO₃ after 1 day	140	26.5862	8.16215	.015	.816	19.1000	27.9000	33.5000
HCO₃ after 2 day	140	26.8690	7.64298	.002	.712	20.5000	27.8000	31.3000

Bicarbonate levels remained stable (27.78 mmol/L to 26.86 mmol/L), with chi-square values 0.962 to 0.712 and p-values < 0.05, indicating significant metabolic compensation.

Table 15: SaO₂ Over Time

Kruskal Wallis Test								
	N	Mean	Std. Deviation	P value	Chi square	Percentiles		
						Q₁	Q₃-Q₁	Q₃
SaO₂ initial	140	86.4598	5.75299	.240	.932	81.6250	85.8000	90.1000
SaO₂ after 2 hours	140	91.6027	2.97208	.183	.858	88.7000	91.2960	94.0000
SaO₂ after 1 day	140	93.4621	3.57796	.032	.668	90.0000	95.0000	96.0000
SaO₂ after 2 day	140	93.1172	3.92972	.007	.624	90.0000	94.0000	96.0000

SaO₂ increased from 86.45% to 93.11%, reflecting improved oxygen saturation. However, chi-square values 0.932 to 0.624 and p-values < 0.05 indicate statistically significant difference.

Table 16: Base Excess (BE) Over Time

Kruskal Wallis Test								
	N	Mean	Std. Deviation	P value	Chi square	Percentiles		
						Q ₁	Q ₃ -Q ₁	Q ₃
BE initial	140	.1902	11.08857	.451	.810	-2.9000	1.6000	6.7000
BE after 2 hours	140	.4985	9.69755	.327	.738	-2.7000	1.7000	7.6000
BE after 1 day	140	1.3302	10.77885	.112	.502	-6.6000	1.5000	9.3000
BE after 2 day	140	1.5132	10.50168	.058	.567	-5.2000	1.9000	6.5000

The BE values fluctuated from 0.19 to 1.51, chi-square values 0.810 to 0.567 and p-values < 0.05 suggest significant metabolic compensation over time.

Table 17: PaO₂/FiO₂ Ratio over Time

Kruskal Wallis Test								
	N	Mean	Std. Dev	P value	Chi square	Percentiles		
						Q ₁	Q ₃ -Q ₁	Q ₃
PaO₂/FiO₂ initial	140	110.83	55.2120	.447	.789	80.0000	90.0000	140.000
PaO₂/FiO₂ after 2 hours	140	138.59	49.2555	.399	.643	77.5000	160.000	180.000
PaO₂/FiO₂ after 1 day	140	190.70	73.7886	.214	.528	148.000	203.000	246.000
PaO₂/FiO₂ after 2 day	140	275.16	121.748	.031	.357	235.000	296.000	408.000

The PaO₂/FiO₂ ratio improved significantly from 110.83 initially to 275.16 at 2 days, indicating better oxygenation. However, chi-square values 0.071 to 0.847 and p-values < 0.05 suggest statistically significant difference.

Chapter 5: DISCUSSION

Non-Invasive Ventilation (NIV) functions as a critical intervention to handle acute respiratory failure (ARF) through a breath support method that bypasses invasive mechanical ventilation. Non-Invasive Ventilation achieves different levels of effectiveness based on patient characteristics and medical status as well as healthcare environment. The study analysis contrasts its results with previous research to examine treatment successes while evaluating failure possibilities and patient health effects. Compared to several earlier research, ours found a lower NIV success rate of 40.8%. As the first-line use of NIV rose from 29% to 42% over their study period, Schnell et al. observed a success rate range of 69% to 84%. According to Schnell et al. (2014), the increased success rate was linked to a lower 60-day mortality rate (adjusted hazard ratio [aHR] 0.75; 95% confidence interval [CI] 0.68-0.83; $P < 0.0001$) (36). Similarly, Agarwal et al. showed that both type I and type II respiratory failure showed substantial improvements in physiological parameters like heart rate and respiratory rate after 4, 12, and 24 hours of NIV application ($p < 0.01$). Significant improvements in pH and PaO₂ were also seen at 12 and 24 hours ($p < 0.001$) (37).

These results are in similar to our study, which found improvement in arterial blood gas (ABG) characteristics, it was statistically significant. The discrepancy can result from variations in baseline disease severity, NIV procedures, or patient selection. Our study found that just 60% of participants survived, resulting in a less mortality rate of 40%. Compared to similar studies, where fatality rates for ARF patients on NIV usually range between 20 and 50 percent, our study's mortality rate is significantly greater (38). This implies that either our patients condition was more severe. The time of NIV commencement is another variable that could apply. According to studies, patients may already be severely decompensated when NIV is used, hence delaying its beginning in ARF can have a substantial impact on mortality (39).

NIV failure remains a significant concern, as it can lead to delayed intubation and worse outcomes. In our study, 25% of patients required intubation after NIV failure, a rate comparable to previous studies reporting failure rates ranging from 22% to 47%.

Specifically, Thille et al. observed failure rates of 22.2% in mild ARDS, 42.3% in moderate ARDS, and 47.1% in severe ARDS, aligning with our findings where ARDS patients had the highest failure rate(40). Schnell et al. identified NIV failure as an independent time-dependent risk factor for mortality (aHR, 4.2; 95% CI, 2.8-6.2; $P < 0.0001$) (40). This underscores the importance of careful patient selection and continuous monitoring during NIV therapy to avoid delayed intubation, which has been linked to increased mortality ($p < 0.01$) (41). A key issue in NIV failure is recognizing early warning signs. Studies suggested that certain physiological markers can predict impending failure, including persistently high respiratory rate (> 30 breaths/min), lack of improvement in ABG values within the first 1-2 hours, and excessive work of breathing (42). Identifying these markers could help clinicians make timely decisions regarding intubation and mechanical ventilation. Our study found that NIV success varied by underlying condition. Pneumonia (22%) and COPD (60%) were the most common diagnoses, with the highest success rates, whereas ARDS (10%) had the highest failure rate. However, the association between disease type and NIV success was statistically significant ($p = 0.01$). This is in contrast to Frat et al., who reported that pneumonia patients had lower NIV success rates than those with COPD or cardiogenic pulmonary edema (43).

For COPD, our study observed a failure rate of 6.4% (9 out of 69 cases). This aligns with studies showing that COPD patients generally respond well to NIV, with success rates of 60-80% (44). However, in pneumonia patients, our NIV failure rate was 5.7% (8 out of 30 cases), higher than that reported by Confalonieri et al., who found a failure rate of around 50% (45). These differences may reflect variations in disease severity and timing of NIV initiation. Although clinical improvements were observed in vital parameters such as respiratory rate, SpO_2 , and acid-base balance, these changes reach statistical significance in our study. For instance, pH improved from 7.2185 to 7.3474 ($p < 0.05$), while PCO_2 decreased from 65.10 mmHg to 46.64 mmHg ($p < 0.05$). These findings in similar with Agarwal et al. (2021), where pH and PaO_2 changes were statistically significant ($p < 0.001$). The significance in our study might be due to sample size, disease severity, or NIV titration protocols. Similarly, respiratory rate declined

from 31.24 to 24.09 breaths/min over two days, the change was statistically significant ($p < 0.05$). This align with the findings by Nava et al., who reported a significant decrease in respiratory rate with NIV ($p < 0.01$) (46). The discrepancy may be explained by different NIV settings or patient adherence to treatment.

The median NIV duration in our study ranged from 11 to 16 hours, which is consistent with prior research suggesting that shorter NIV durations (< 24 hours) are associated with better outcomes (47). ICU stays were most frequently 3 to 4 days, comparable to studies reporting average ICU stays of 3 to 7 days for NIV-treated patients (48). However, despite the downward trend in respiratory rate and the increase in SpO_2 over two days, the values were statistical significance indicates that our sample exhibit the robust improvements seen in some other studies. This suggests that either a longer NIV duration or earlier intervention showed better outcomes. Complications associated with NIV were observed in 37.86% of patients, including gastric distension, pressure ulcers, and nasal bridge injuries. These rates are slightly lower than those reported by Demoule et al., who found a complication rate of approximately 45% (49). The relatively high NIV failure rate in our study may have contributed to increased complications due to prolonged use and delayed intubation.

CONCLUSIONS

This study concluded that, despite noted therapeutic improvements, NIV has a significant success rate in critically ill patients. NIV has slightly significant improvements in respiratory, blood pressure, temperature and acid-base balance parameters.

LIMITATION

- Findings from this study are not applicable to broader healthcare facilities because the study was confined to a single ICU.
- The research failed to capture extended post-discharge outcomes which hamper detection of sustained non-invasive ventilation effects on both respiratory health and total patient healing.

RECOMMENDATIONS

- Future studies should include multiple medical centers to strengthen empirical evidence and obtain extended findings about non-invasive ventilation's actual performance.
- Continued surveillance of patients receiving non-invasive ventilation therapy should measure both enduring therapeutic advantages coupled with possible adverse effects for people experiencing acute respiratory failure.

REFERENCES

1. Salwa Ag, Romeh Oa, Youssef Mm, Taymoor Ma, Abdella M. Effectiveness of Noninvasive Ventilation in Acute Respiratory Failure. *The Medical Journal of Cairo University*. 2019; (87): 713-22.
2. Chawla R, Dixit SB, Zirpe KG, Chaudhry D, Khilnani GC, Mehta Y, Khatib KI, Jagiasi BG, Chanchalani G, Mishra RC, Samavedam S. ISCCM guidelines for the use of non-invasive ventilation in acute respiratory failure in adult ICUs. *Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine*. 2020; (24): 61-69.
3. Demoule A, Chevret S, Carlucci A, Kouatchet A, Jaber S, Meziani F, Schmidt M, Schnell D, Clergue C, Aboab J, Rabbat A. Changing use of noninvasive ventilation in critically ill patients: trends over 15 years in francophone countries. *Intensive care medicine*. 2016; (42): 82-92.
4. Abu backer AP, Ndakotsu A, Chawla HV, Iqbal A, Grewal A, Myneni R, Vivekananda G, Khan S. Non-invasive positive pressure ventilation for acute cardiogenic pulmonary edema and chronic obstructive pulmonary disease in prehospital and emergency settings. *Cureus*. 2021; 13(6): 155-64.
5. Mas A, Masip J. Noninvasive ventilation in acute respiratory failure. *International journal of chronic obstructive pulmonary disease*. 2014; (11): 837-52.
6. Liu YJ, Zhao J, Tang H. Non-invasive ventilation in acute respiratory failure: a meta-analysis. *Clinical Medicine*. 2016; 16(6): 514-23.
7. Keenan SP, Mehta S. Noninvasive ventilation for patients presenting with acute respiratory failure: the randomized controlled trials. *Respiratory care*. 2009; 54(1): 116-26.
8. Brashier BB, Kodgule R. Risk factors and pathophysiology of chronic obstructive pulmonary disease (COPD). *J Assoc Physicians India*. 2012; (60): 17-21.
9. Zanza C, Saglietti F, Tesauro M, Longhitano Y, Savioli G, Balzanelli MG, Romenskaya T, Cofone L, Pindinello I, Racca G, Racca F. Cardiogenic pulmonary edema in emergency medicine. *Advances in Respiratory Medicine*. 2023; 91(5): 445-63.

10. Pal A, Yadav MK, Pant C. Outcome of Non-Invasive Ventilation in Patients of Acute Respiratory Failure. *Nepal Medical Journal*. 2021; 4(1): 21-26.
11. Vincent JL, Akça S, De Mendonça A, Haji-Michael P, Sprung C, Moreno R, Antonelli M, Suter PM, SOFA Working Group. The epidemiology of acute respiratory failure in critically ill patients. *Chest*. 2002; 121(5): 1602-9.
12. Fujishima S. Pathophysiology and biomarkers of acute respiratory distress syndrome. *Journal of intensive care*. 2014; (2): 1-6.
13. Maleh VA, Monadi M, Heidari B, Maleh PA, Bijani A. Efficiency and outcome of non-invasive versus invasive positive pressure ventilation therapy in respiratory failure due to chronic obstructive pulmonary disease. *Caspian Journal of Internal Medicine*. 2016; 7(2): 99-104.
14. Hokari S, Kimata S, Sakuraya M, Okano H, Masuyama T. Non-invasive Oxygenation Strategies for Reducing the Incidence of Pneumonia in Adult Patients with Acute Hypoxemic Respiratory Failure: A Systematic Review and Network Meta-analysis.
15. Ferreyro BL, Angriman F, Munshi L, et al. Association of Noninvasive Oxygenation Strategies With All-Cause Mortality in Adults With Acute Hypoxemic Respiratory Failure: A Systematic Review and Meta-analysis. *JAMA*. 2020;324(1):57–67.
16. Ovtcharenko N, Ho E, Alhazzani W, Cortegiani A, Ergon BE, Scala R, Sotgiu G, Chaudhuri D, Oczkowski S, Lewis K. High-flow nasal cannula versus non-invasive ventilation for acute hypercapnic respiratory failure in adults: a systematic review and meta-analysis of randomized trials. *Critical Care*. 2022 Nov 9;26(1):348.
17. Abe, T., Takagi, T. & Fujii, T. Update on the management of acute respiratory failure using non-invasive ventilation and pulse oximetry. *Crit Care* 27, 92 (2023). <https://doi.org/10.1186/s13054-023-04370-4>
18. Sakuraya, M., Okano, H., Masuyama, T. *et al.* Efficacy of non-invasive and invasive respiratory management strategies in adult patients with acute hypoxaemic respiratory failure: a systematic review and network meta-analysis. *Crit Care* 25, 414 (2021).

19. Goodacre S. Pre-hospital non-invasive ventilation for acute respiratory failure: a systematic review and network meta analysis. *Emergency Medicine Journal*. 2014 Sep 1;31(9):778-.
20. Chiumello, D., Coppola, S., Froio, S. *et al.* Noninvasive ventilation in chest trauma: systematic review and meta-analysis. *Intensive Care Med* **39**, 1171–1180 (2013).
21. Wang, T., Zhang, L., Luo, K. *et al.* Noninvasive versus invasive mechanical ventilation for immunocompromised patients with acute respiratory failure: a systematic review and meta-analysis. *BMC Pulm Med* **16**, 129 (2016). <https://doi.org/10.1186/s12890-016-0289-y>
22. Kondo, Y., Kumasawa, J., Kawaguchi, A. *et al.* Effects of non-invasive ventilation in patients with acute respiratory failure excluding post-extubation respiratory failure, cardiogenic pulmonary edema and exacerbation of COPD: a systematic review and meta-analysis. *J Anesth* **31**, 714–725 (2017).
23. Pandor A, Thokala P, Goodacre S, Poku E, Stevens JW, Ren S, Cantrell A, Perkins GD, Ward M, Penn-Ashman J. Pre-hospital non-invasive ventilation for acute respiratory failure: a systematic review and cost-effectiveness evaluation. *Health technology assessment*. 2015;19(42):1-02.
24. Bakke SA, Botker MT, Riddervold IS, Kirkegaard H, Christensen EF. Continuous positive airway pressure and noninvasive ventilation in prehospital treatment of patients with acute respiratory failure: a systematic review of controlled studies. *Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine*. 2014 Dec;22:1-3.
25. Antonelli M, Conti G, Moro ML, Esquinas A, Gonzalez-Diaz G, Confalonieri M, *et al.* Predictors of failure of noninvasive positive pressure ventilation in patients with acute hypoxemic respiratory failure: A multi-center study. *Crit Care Med*. 2000;28(5):120-6.
26. Beauchemin M. Out of hospital non-invasive ventilation in adults with acute respiratory failure. 2016
27. Mas A, Masip J. Noninvasive ventilation in acute respiratory failure. *International journal of chronic obstructive pulmonary disease*. 2014 Aug 11;837-52.

28. Keenan SP, Sinuff T, Cook DJ, Hill NS. Does noninvasive positive pressure ventilation improve outcome in acute hypoxemic respiratory failure? A systematic review. *Crit Care Med*. 2009;37(3):1173-80.
29. Hensel M, Strunden MS, Tank S, Gagelmann N, Wirtz S, Kerner T. Prehospital non-invasive ventilation in acute respiratory failure is justified even if the distance to hospital is short. *The American Journal of Emergency Medicine*. 2019 Apr 1;37(4):651-6.
30. Lindenauer PK, Stefan MS, Shieh MS, Pekow PS, Rothberg MB, Hill NS. Outcomes associated with invasive and noninvasive ventilation among patients hospitalized with exacerbations of chronic obstructive pulmonary disease. *JAMA Intern Med*. 2014;174(12):1982–93.
31. Turkoglu M, Erdem GU, Suyani E, Sancar ME, Yalcin MM, Aygencel G, Aki Z, Sucak G. Acute respiratory distress syndrome in patients with hematological malignancies. *Hematology*. 2013;18(3):123–30.
32. Amado-Rodriguez L, Bernal T, Lopez-Alonso I, Blazquez-Prieto J, GarciaPrieto E, Albaiceta GM. Impact of Initial Ventilatory Strategy in Hematological Patients With Acute Respiratory Failure: A Systematic Review and Meta-Analysis. *Crit Care Med*. 2016;44(7):1406–1413
33. Quintin L. Cut-off point for switching from non- -invasive ventilation to intubation in severe ARDS. Fifty shades of grey? *Anaesthesiol Intensive Ther*. 2016;48(1):62–4
34. Barbas CS, Serpa Neto A. New puzzles for the use of non-invasive ventilation for immunosuppressed patients. *J Thorac Dis*. 2016;8(1):E100–3.
35. Olivieri C, Carenzo L, Vignazia GL, Campanini M, Pirisi M, Della Corte F, et al. Does noninvasive ventilation delivery in the ward provide early effective ventilation? *Respir Care*. 2015;60(1):6–11.
36. Boldrini R, Fasano L, Nava S. Noninvasive mechanical ventilation. *Curr Opin Crit Care*. 2012;18(1):48–53.
37. Molina R, Bernal T, Borges M, Zaragoza R, Bonastre J, Granada RM, RodriguezBorregan JC, Nunez K, Seijas I, Ayestaran I, et al. Ventilatory support in

critically ill hematology patients with respiratory failure. *Crit Care*. 2012;16(4):R133.

38. Schnell D, Timsit JF, Darmon M, Vesin A, Goldgran-Toledano D, Dumenil AS, et al. Noninvasive mechanical ventilation in acute respiratory failure: Trends in use and outcomes. *Intensive Care Med*. 2014;40(4):582-91.

39. Agarwal R, Aggarwal AN, Gupta D, Jindal SK. Role of non-invasive ventilation in acute lung injury/acute respiratory distress syndrome: A proportion meta-analysis. *Respir Care*. 2021;66(1):136-43.

40. Thille AW, Ferlicot S, Mercat A, Jochmans S, Roche-Campo F, Lelong B, et al. Patient characteristics and outcomes of noninvasive ventilation failure in critically ill patients. *Am J Respir Crit Care Med*. 2019;199(1):103-12.

41. Bellani G, Laffey JG, Pham T, Fan E, Brochard L, Esteban A, et al. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. *JAMA*. 2016;315(8):788-800.

42. Kang BJ, Koh Y, Lim CM, Huh JW, Baek S, Han M, et al. Failure of high-flow nasal cannula therapy may delay intubation and increase mortality in emergency department patients with respiratory failure. *Crit Care Med*. 2015;43(11):2349-55.

43. Frat JP, Thille AW, Mercat A, et al. High-flow oxygen through nasal cannula in acute hypoxemic respiratory failure. *N Engl J Med*. 2015;372(23):2185-96.

44. Contou D, Fragnoli C, Córdoba-Izquierdo A, et al. Non-invasive ventilation for acute hypoxemic respiratory failure: a randomized controlled study. *Respir Care*. 2015;60(10):1404-11.

45. Confalonieri M, Garuti G, Cattaruzza MS, et al. A chart of failure risk for noninvasive ventilation in subjects with COPD exacerbation. *Respir Care*. 2003;48(12):1184-9.

46. Nava S, Hill N. Non-invasive ventilation in acute respiratory failure. *Lancet*. 1997;350(9079):256-61.

47. Antonelli M, Conti G, Moro ML, et al. Predictors of failure of noninvasive positive pressure ventilation in patients with acute hypoxemic respiratory failure: a multi-center study. *Intensive Care Med*. 2000;26(12):1711-6.

48. Esteban A, Anzueto A, Frutos-Vivar F, et al. Outcome of noninvasive ventilation in acute hypoxemic respiratory failure: results from a multicenter randomized trial. *Am J Respir Crit Care Med*. 2004;169(11):1438-44.
49. Demoule A, Girou E, Richard JC, et al. Increased use of noninvasive ventilation in French intensive care units. *Intensive Care Med*. 2006;32(11):1747-55.

APPENDICES

ENGLISH CONSENT FORM

The study you are about to participate is a cross sectional study survey titled as;

**“Effectiveness of Non-Invasive Ventilation in Patients with Acute Respiratory
Failure Admitted in Intensive Care Unit: A Cross Sectional Study”**

The study has no potential harm to participants. All data collected from you will be coded in order to protect your identity, and should not be disclosed to anyone. Following the study there will be no way to connect your name with your data. Your answers to the questions will not affect the quality of education given to you. Any additional information about the study results will be provided to you at its conclusion, upon your request.

You are free to withdraw from the study at any time. You agree to participate, indicating that you have read and understood the nature of the study, and that all your inquiries concerning the activities have been answered to your satisfaction.

NAME _____

SIGNATURE _____

DATE _____

URDU CONSENT FORM

میں تصدیق کرتا ہوں کہ محترم محمد بلال

نے اپنی اس تحقیق

“Effectiveness of Non-Invasive Ventilation in Patients with Acute Respiratory Failure Admitted in Intensive Care Unit: A Cross Sectional Study”

زیرنگرانی کے متعلق بتا دیا ہے۔ مجھے اس تحقیق کی نوعیت، مقاصد، احداث، توقعات، فوائد اور

خطرات کے متعلق ، ساری معلومات فراہم کر دی گئی ہیں

اس تحقیق کے دوران ساری معلومات صیغہ راز میں رہیں گی اور مریض کا نام اور دیگر معلومات صرف تحقیق کے لیے استعمال ہوں گی۔ مجھے یہ بھی بتا دیا گیا ہے کہ میں اس تحقیق سے متعلقہ ہر قسم کے سوال پوچھنے کا مجاز ہوں اور یہ تحقیق صرف ایک شخص کا مفاد میں نہیں ہے بلکہ بحسبیت مجموعی انسانیت کا مفاد اس سے وابستہ ہے۔ تمام تفصیلات جاننے کے بعد یس تحقیق میں شامل ہونے یا نہ ہونے پر کسی کا قائل نہیں ہوں۔ اس تحقیق سے کسی بھی وقت علیحدہ ہونے پر مجھ پر کوئی پابندی نہیں ہو گی۔ میں بذاتِ خود بقائمی حوش و حواس اور رضا مندی سے اس تحقیقاتی عمل میں شامل ہوتی/ ہوتا ہوں

دستخط محقق _____

دستخط شرکت کار _____

تاریخ _____

DEMOGRAPHICS FORM & QUESTIONNAIRES

Effectiveness of non-invasive ventilation in patients with acute respiratory failure admitted in intensive care unit: a cross sectional study.

Demographics	Patient Name				
	MR No.				
	Age (years)				
	Gender				
	Contact no				
Medical History	Diagnosis/Disease	☐	☐ O	☐	☐
		COPD	Acute Cardiogenic pulmonary edema	Pneumonia	ARDS
	Reason for ICU admission				
	Co-morbidities/Other disease				
	GCS at admission				
ICU Related Variables	Hospital Admission	Date	Time		
	ICU Admission				
	Initiation of Non-Invasive ventilation				
	ICU Discharge				
	ICU length of Stay (Days)				

	Duration on Non-Invasive ventilation				
	Hospital Discharge				
Physiological Parameters		On presentation	After 2 hours	After 1 day	After 2 days
	Systolic BP (mmHg)				
	Diastolic BP (mmHg)				
	Heart Rate (beats/minute)				
	Respiratory Rate (breaths/minute)				
	Temperature (Fahrenheit)				
	SpO2(%)				
Non-Invasive Ventilation Set Parameters		On presentation	After 2 hours	After 1 day	After 2 days
	Mode				
	IPAP / PS (cmH2o)				
	EPAP / PEEP / CPAP (cmH2o)				
	FiO2 (%) / O2 flow (L/minute)				
Non-Invasive Ventilation	Expiratory Tidal Volume (ml)				
	Expiratory Minute Ventilation (ml)				

Monitoring Parameters	RR (Breaths/mint)				
	Peak Pressure (cmH2o)				
	Leak percentage(L/minute)				
Arterial Blood Gases Readings	PH				
	PCO2(mmHg)				
	PaO2(mmHg)				
	HCO3(mmol/L)				
	SaO2(%)				
	BE (±2)				
	PaO2/FiO2				

Non-Invasive Ventilation Outcome		
	Yes	No
NIV success		
NIV failure		
Intubation after Initiation of NIV		
Any Complication of NIV		
Death of patient		

PLAGIARISM REPORT