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RESEARCH ARTICLE

PREDICTORS OF OUTCOME OF NONINVASIVE VENTILATION IN SEVERE COPD EXACERBATION

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality worldwide. Acute exacerbation of COPD (AECOPD) often leads to respiratory failure requiring ventilatory support. Non-invasive ventilation (NIV) has emerged as an effective treatment strategy, but factors predicting its outcome remain inadequately understood. This study aimed to identify factors that predict NIV outcomes in patients with AECOPD and explore determinants of NIV settings and duration.

Methodology: A hospital-based prospective cohort study was conducted at the Department of Respiratory Medicine, Jaipur National University Hospital, Rajasthan, India. Sixty patients with AECOPD requiring NIV were enrolled. Clinical parameters, arterial blood gas (ABG) analyses and ventilator settings were recorded at baseline and at multiple intervals during treatment. Outcomes were categorized as "success" (clinical stability allowing ward transfer) or "failure" (worsening respiratory parameters requiring intubation or resulting in death).

Results: Of the 60 patients, 43 (71.7%) responded successfully to NIV. Baseline demographic and clinical characteristics were comparable between success and failure groups. However, significant improvements in pH ($p=0.013$), $PaCO_2$ ($p=0.007$) and PaO_2 ($p=0.018$) were observed in the success group after just 2 hours of NIV therapy with continued improvement in subsequent measurements. The mean duration of NIV treatment was significantly longer in the success group (2.69±3.80 days) compared to the failure group (0.92±1.41 days, $p=0.018$). Commonly observed complications included dryness of oral and nasal mucosa (30%), eye irritation (20%) and skin abrasion (13.3%).

Conclusion: Early improvement in arterial blood gas parameters, particularly pH, $PaCO_2$ and PaO_2 within the first 2 hours of NIV initiation, strongly predicts successful outcomes in AECOPD patients. Regular monitoring of these parameters may help identify patients who
would benefit from continued NIV support versus those requiring escalation to invasive ventilation.

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Introduction:-

COPD is the third most common cause of death worldwide, causing 3 million deaths and 63.5 million disability-adjusted life years (DALY) lost globally in 2016, leading to substantial morbidity.[1] One common complication of COPD is acute exacerbation (AECOPD), which can lead to hospitalization and significant expenses to the healthcare system and society, alongside higher rates of morbidity and mortality.[2] People with COPD, especially those with more severe disease, are more likely to experience exacerbations, which often lead to hospitalization. Breathing becomes extremely challenging for patients experiencing a severe episode of COPD. This may result in acute hypercapnic respiratory failure (AHRF), which frequently necessitates immediate hospital-based medical attention. Acute respiratory acidosis brought on by protracted hypercapnia (high carbon dioxide levels) is a typical feature of severe AECOPDs. Despite the adoption of mechanical ventilator support techniques, between one-third and one-fifth of COPD patients admitted to hospitals with AHRF pass away while still in the hospital.[3-8]

In advanced stages of COPD, respiratory muscles operate close to their functional limits, and hyperinflation further hampers their efficiency by altering the mechanics of breathing [9,10]. During acute exacerbations, the added elastic and resistive loads on these muscles may lead to respiratory failure. This deterioration is aggravated by tissue acidosis, which negatively affects muscle performance and accelerates the decline in ventilation [11]. Traditional treatment protocols focus on resolving the underlying trigger while maintaining adequate oxygenation through the use of bronchodilators, corticosteroids, antibiotics and regulated oxygen therapy. In situations where patients do not improve with these interventions, invasive mechanical ventilation is often used. This method involves sedating the patient, performing intubation connecting to a mechanical ventilator and transferring to an intensive care unit (ICU). Although this technique can successfully correct hypercapnic acidosis in some cases, it poses several risks such as barotrauma, oxygen toxicity, vocal cord injury, tracheal narrowing and increased risk of ventilator-associated infections [12–14]. Furthermore, patients with COPD undergoing invasive ventilation often face challenges such as high complication rates and difficulties in weaning off the ventilator [15,16].

Non-invasive ventilation (NIV) has gained recognition as a viable alternative for treating acute hypercapnic respiratory failure (AHRF) triggered by acute exacerbations of COPD (AECOPD) [4,12,17,18]. Unlike invasive techniques, NIV delivers ventilatory assistance through a nasal or full-face mask, eliminating the need for sedation and intubation. Its advantages include short-duration usage, maintaining the patient's ability to speak, eat, and drink and significantly reducing the risk of hospital-acquired pneumonia [19–21]. NIV functions by delivering pressure-supported airflow that unloads fatigued respiratory muscles helping to improve lung mechanics and gas exchange, ultimately aiding in the correction of acidosis [22]. Clinical evidence from randomized controlled trials and case studies supports the effectiveness of NIV in managing AHRF caused by AECOPD [4,22,23]. However, failure rates between 9% and 50% have been reported indicating that NIV does not always outperform conventional treatments [24,25]. This is concerning, as delayed intubation due to unsuccessful NIV application can worsen patient outcomes [26,27]. This study seeks to identify predictors of NIV success or failure in AECOPD cases and to analyze the factors that affect the selection of NIV settings and the duration of therapy.

Methodology:-

The study employed a hospital-based prospective cohort design, conducted over a period of one year at the Department of Respiratory Medicine, JNU Hospital, Jaipur, Rajasthan. Ethics committee authorization was obtained from the Institutional Review Board for Ethical Clearance. All participants, or their attendants, were thoroughly informed about the study's procedures and its objectives. Written informed consent was obtained from all consenting individuals and patient confidentiality was strictly maintained throughout the research. The study did not alter the standard treatment protocols for the participants, nor did it impose any additional financial burdens on them.

The sample size for the study was determined based on a 95% confidence level, assuming a failure rate of 24% after NIV for AECOPD, as cited by Mostafa Shaheen et al.[28] With an absolute error of 10%, the required sample size was calculated to be 70, using the formula $n = Z\alpha^2\ p\ q\ /\ d^2$, where $Z\alpha$ is 1.96, p is the failure rate (0.24), q is the complement of p (0.76) and d is the absolute error (0.1).

The inclusion criteria for the study included individuals experiencing acute exacerbation of COPD with Type 2 respiratory failure, those with a prior COPD diagnosis per the GOLD 2023 guidelines and patients who were admitted to the ICU, hospital, or casualty and were older than 18 years. Participants had to provide written informed consent. The exclusion criteria eliminated patients with severe upper gastrointestinal hemorrhage, hemodynamic instability, cardiac or respiratory arrest, facial surgery or trauma interfering with mask fitting, pneumonia,

cerebrovascular accidents, or those requiring immediate intubation, among other conditions. After applying these criteria, the study population consisted of 60 patients.

A cohort of 60 patients who met the inclusion and exclusion criteria was enrolled. COPD was diagnosed through clinical history, physical examination, pulmonary function tests and imaging such as chest radiography. Before initiating non-invasive ventilation, all participants underwent conventional medical treatment for 45-60 minutes. Non-invasive ventilation parameters were individualized with patients receiving respiratory support through either pressure support mode or pressure-controlled mode via complete facial coverings or occasionally, helmet devices. The breathing pressure was modified based on patient comfort levels, aiming for an exhaled breath volume of 7-8 mL/kg while maintaining external positive end-expiratory pressure below 6 cmH_2O. Critical care breathing machines or dedicated non-invasive ventilation systems were utilized to track exhaled breath volumes, and oxygen concentration was regulated to sustain oxygen saturation levels above 90%.
The definitions for NIV success and failure were clearly established. Success was defined as achieving a clinical and functional status that allowed the patient to be transferred to the ward, whereas failure was identified by a worsening of arterial blood gas (ABG) tensions, significant dyspnea, or sensory deterioration during mechanical ventilation, or if the patient died in the ICU. For each patient, data was collected, including age, gender, baseline ABG results and measurements taken two hours after NIV initiation, on days one to three and at the time of ICU discharge (for successful cases) or before intubation or death (for failed cases).

The parameters assessed in the study included age, gender, BMI, lung function tests, blood pressure, ABG values, total and differential counts, creatinine clearance and echocardiographic data. Additionally, respiratory rate changes, the need for endotracheal intubation, the duration of NIV during the first 72 hours and the length of hospital stay (LOS) were recorded. The statistical analysis was carried out using SPSS 22.0 for Windows. Means and standard deviations were calculated for each group at various assessment points. One-way ANOVA was applied to analyze the data with a significance threshold set at $p < 0.05$.

Results:-

Table 1:- Demographic and Clinical Characteristics of Study Participants.

Characteristics	Value (N=60)	Percentage (%)
Gender		
Male	32	53.33
Female	28	46.67
Age (years), Mean±SD	65.05±8.71	-
BMI, Mean±SD	24.5±4.28	-
Symptoms		
Cough	55	91.67
Sputum	34	56.67
Fever	22	36.67
Chest Pain	5	8.33
Shortness of Breath	60	100
MMRC Grade of Dyspnea		
II	2	3.33
III	30	50.00
IV	28	46.67
Biomass Exposure		
Yes	25	41.67
No	35	58.33
Smoking Status		
Yes	49	81.67
No	11	18.33
Comorbidities		
Diabetes Mellitus	20	33.33
Hypertension	23	38.33
OSA	7	11.67

CAD	5	8.33
Pulmonary Hypertension	2	3.33
COR Pulmonale		
Yes	36	60
No	24	40

Table 1 summarizes the demographic and clinical profile of patients with AECOPD requiring NIV. The study found a slight male predominance (53.33%) in the gender distribution, which contrasts with the traditionally higher male prevalence in COPD. This could reflect changing epidemiological trends, possibly due to increased smoking among women or exposure to biomass fuels, which was present in 41.67% of the cohort. The mean age of 65.05±8.71 years is typical for COPD exacerbations requiring hospitalization as the disease commonly presents in the sixth and seventh decades of life. The mean BMI of 24.5±4.28 was within the normal range. Dyspnea was the most common symptom reported by all participants (100%), followed by cough (91.67%) with 56.67% of patients having a productive cough, aligning with chronic bronchitis in COPD. Fever was observed in 36.67% of cases, suggesting an infectious trigger, while chest pain was uncommon (8.33%). The majority of patients (50%) had severe breathlessness (MMRC grade III) and 46.67% were in grade IV indicating significant functional limitations.

Tobacco smoking was the primary risk factor, affecting 81.67% of patients with a mean smoking index of 718. Non-smokers (18.33%) were likely affected by other factors, including biomass fuel exposure (41.67%). The comorbidity profile revealed a high prevalence of hypertension (38.33%) and diabetes mellitus (33.33%). Obstructive sleep apnea (11.67%) and coronary artery disease (8.33%) were also present, indicating overlap syndromes and shared risk factors with cardiovascular diseases. Notably, cor pulmonale was present in 60% of patients, signifying advanced pulmonary vascular remodeling and right heart dysfunction. This finding is important as cor pulmonale is associated with poorer COPD outcomes and may influence NIV therapy effectiveness. Most patients (26.7%) had been diagnosed with COPD for 4 years and 35% had experienced a previous exacerbation within the last year indicating either their first severe exacerbation or significant deterioration after a period of relative stability.

Clinical, Laboratory and Radiological Findings

The comprehensive evaluation of clinical parameters revealed significant respiratory compromise in the study population. The mean SPO_2 at presentation was notably low at 76.05±7.11%, indicating substantial hypoxemia despite supplemental oxygen. This was accompanied by tachypnea with a mean respiratory rate of 31.55±4.91 breaths per minute, reflecting the increased work of breathing characteristic of acute exacerbations. The mean total leukocyte count was 9225.17±1814.50 cells/mm^3, suggesting an inflammatory response that often accompanies AECOPD, although not reaching levels typically seen in acute bacterial infections. Cardiovascular assessment through ECG revealed predominant sinus tachycardia in 71.7% of patients, likely a compensatory response to hypoxemia and increased metabolic demands. More concerning were the findings of arrhythmia in 18.3% of patients, which may represent either pre-existing cardiac disease or acute strain on the cardiovascular system. Acute coronary syndrome was identified in 10% of patients, highlighting the significant cardiopulmonary interaction during severe COPD exacerbations and the potential for hypoxemia to precipitate myocardial ischemia.

Radiological evaluation through chest X-rays demonstrated the chronic changes expected in COPD with hyperinflation of the lungs identified in 51.7% of patients (consistent with chronic bronchitis predominant phenotype) and emphysematous changes in 48.3% (consistent with emphysema predominant phenotype). These findings reflect the underlying pathophysiological processes that predispose patients to acute exacerbations. Pulmonary function testing revealed significant airflow limitation that showed measurable improvement during the course of treatment. The mean PEFR at admission was 162.05±47.11 L/min, which improved to 190.53±41.78 L/min at discharge ($p<0.01$). While this improvement was statistically significant, the discharge values remained substantially below predicted normal values, consistent with the irreversible component of airflow limitation characteristic of COPD.

NIV Parameters and Outcomes

The NIV settings employed in this study reflect a therapeutic approach tailored to balance patient comfort with effective ventilatory support. The mean IPAP of 14.25±1.56 cm H_2O provided moderate inspiratory assistance to reduce the work of breathing without causing excessive gastric insufflation or patient discomfort. The mean EPAP of 6.49±1.04 cm H_2O was sufficient to counteract intrinsic PEEP and maintain airway patency during exhalation. The overall success rate of NIV therapy was 71.7% (43 patients) with 28.3% (17 patients) experiencing treatment

failure. This success rate is comparable to those reported in similar studies, including those by Brochard L et al. (1995)[5] and Kshatriya RM et al. (2019)[29], which reported success rates of 74%. A notable finding was the significant difference in NIV duration between the success and failure groups (2.69±3.80 days versus 0.92±1.41 days, p=0.018), suggesting that patients who responded positively to NIV were able to tolerate and benefit from longer periods of ventilatory support. The complications associated with NIV therapy were generally mild and manageable. The most common complication was dryness of oral and nasal mucosa (30%), followed by eye irritation (20%) and skin abrasion (13.3%). These interface-related issues are well-recognized challenges in NIV delivery and can often be addressed through mask adjustment, application of protective dressings and appropriate humidification. More concerning complications such as hypotension (6.7%) and abdominal distension (1.7%) were relatively rare. Importantly, 23.3% of patients experienced no complications, suggesting good overall tolerability of the intervention.

Comparison of Arterial Blood Gas Parameters Between Success and Failure Groups

Table 2:- Comparison of pH, $PaCO_2$, PO_2 and HCO_3 Between Success and Failure Groups.

Parameter	Interval	Success Group (Mean±SD)	Failure Group (Mean±SD)	p-value
pH	0 hr	7.10±0.01	7.10±0.02	0.10
	2 hr	7.18±0.01	7.02±0.01	0.013*
	24 hr	7.20±0.02	7.01±0.02	0.009*
	24-48 hr	7.25±0.04	7.05±0.01	0.007*
	48-72 hr	7.30±0.02	7.02±0.02	0.005*
	72-96 hr	7.31±0.03	7.03±0.03	0.004*
$PaCO_2$ (mmHg)	0 hr	71.86±3.51	72.54±2.73	0.79
	2 hr	62.43±2.54	71.19±3.12	0.007*
	24 hr	53.05±2.69	74.06±2.54	<0.01*
	24-48 hr	50.02±2.94	75.28±2.91	<0.01*
	48-72 hr	45.08±2.01	77.55±3.09	<0.01*
	72-96 hr	41.23±2.80	79.38±2.56	<0.01*
PO_2 (mmHg)	0 hr	75.43±3.54	74.68±2.89	0.62
	2 hr	77.81±2.61	71.43±3.06	0.018*
	24 hr	80.26±2.76	66.05±2.48	<0.01*
	24-48 hr	83.19±3.01	61.02±2.85	<0.01*
	48-72 hr	85.40±2.08	59.08±3.03	<0.01*
	72-96 hr	86.83±2.87	58.23±2.50	<0.01*
HCO_3 (mmol/L)	0 hr	30.21±2.81	30.10±2.62	0.83
	2 hr	31.56±1.88	30.78±2.79	0.60
	24 hr	34.19±2.03	32.05±2.21	0.07
	24-48 hr	35.82±2.28	32.69±2.58	0.031*
	48-72 hr	35.54±1.35	33.07±2.76	0.044*
	72-96 hr	35.91±2.14	33.04±2.23	0.048*

*Statistically significant (p<0.05)

Table 2 provides a comprehensive comparison of arterial blood gas parameters between the success and failure groups at multiple time intervals, offering critical insights into the physiological response to NIV therapy. At baseline (0 hr), both groups demonstrated comparable severe respiratory acidosis with mean pH values of 7.10, indicating significant decompensation of the acid-base status. Similarly, baseline $PaCO_2$ levels were markedly elevated in both groups (71.86±3.51 vs. 72.54±2.73 mmHg, p=0.79), reflecting severe alveolar hypoventilation. Initial PO_2 values were also comparable (75.43±3.54 vs. 74.68±2.89 mmHg, p=0.62), as were HCO_3 levels (30.21±2.81 vs. 30.10±2.62 mmol/L, p=0.83), suggesting that the severity of acute respiratory failure at presentation was not predictive of NIV outcome. The divergence in physiological trajectories became evident as early as 2 hours after NIV initiation. The success group demonstrated a significant improvement in pH (7.18±0.01 vs. 7.02±0.01, p=0.013), representing a clear trend toward normalization of acid-base status. This early improvement aligns with findings by Anton A et al. (2000)[30], who identified early pH response as a predictor of NIV success. Concurrently, the success group showed a substantial reduction in $PaCO_2$ levels (62.43±2.54 vs. 71.19±3.12 mmHg, p=0.007) indicating effective alveolar ventilation and CO_2 elimination with NIV support. PO_2 levels also improved

significantly in the success group compared to the failure group at this early time point (77.81±2.61 vs. 71.43±3.06 mmHg, p=0.018).

The physiological disparity between groups became progressively more pronounced over subsequent time intervals. By 24 hours, the success group had achieved a mean pH of 7.20±0.02 compared to 7.01±0.02 in the failure group (p=0.009) with further improvement to near-normal values (7.31±0.03) by 72-96 hours. The failure group, in contrast, remained persistently acidotic. This pattern is consistent with findings by Confalonieri M et al. (2005)[31], who identified persistent acidosis as a marker of NIV failure. Perhaps most striking was the divergent trend in $PaCO_2$ levels. While the success group showed progressive reduction in $PaCO_2$ reaching near-normal values by 72-96 hours (41.23±2.80 mmHg), the failure group demonstrated not only persistence but worsening of hypercapnia (79.38±2.56 mmHg at 72-96 hours, p<0.01). This deterioration in the failure group likely reflects progressive fatigue of respiratory muscles, worsening ventilation-perfusion mismatch, or increasing airway resistance despite NIV support - processes that eventually necessitate invasive ventilation or may lead to mortality if left unaddressed. Oxygenation parameters (PO_2) showed similar divergence with the success group achieving progressive improvement (reaching 86.83±2.87 mmHg by 72-96 hours), while the failure group experienced deterioration (falling to 58.23±2.50 mmHg, p<0.01). This suggests that NIV not only improved ventilation but also oxygenation in responsive patients, likely through recruitment of collapsed alveoli and improvement in ventilation-perfusion matching. The HCO_3 response, representing renal compensation, showed a delayed pattern compared to the respiratory parameters. No significant difference was observed at 2 hours or 24 hours. However, by 24-48 hours, the success group demonstrated significantly higher HCO_3 levels (35.82±2.28 vs. 32.69±2.58 mmol/L, p=0.031), a difference that persisted through subsequent measurements. This delayed response is physiologically consistent with the slower time course of renal bicarbonate retention compared to the more rapid respiratory compensation facilitated by NIV.

Discussion:-

In this study, 60 patients meeting the inclusion criteria were selected. Numerous studies have demonstrated the effectiveness of non-invasive ventilatory assistance as a therapy option for acute exacerbations of COPD with respiratory failure.[32,33] Prompt implementation of NIV during COPD flare-ups accompanied by elevated carbon dioxide respiratory insufficiency can prevent the need for airway intubation and related adverse outcomes. The majority of participants in this study were men (53.33%) with the remainder (46.67%) being women. These results were in line with those of Steriade AT et al. (2019)[34], who also noted that 50.56% of patients in their study were male. Similar to the current study, the majority of the subjects in the study by Vaudan S et al. (2015)[35] were men. The subjects' mean BMI in this study was 24.5±4.28. Barbé F et al. (1996)[24] found similar results, reporting that the subjects' mean BMI was 24.9±1.3.

In the present study, 81.67% of subjects had a positive history of smoking with a mean smoking index of 718. This high prevalence of smoking history is consistent with the known strong association between tobacco smoking and COPD development. The most common comorbidities observed in this study were hypertension (38.33%), diabetes mellitus (33.33%), OSA (11.67%) and CAD (8.33%). According to a study by Ongel EA et al. (2014)[36], cardiovascular comorbidities are the most prevalent comorbidities in COPD with incidence rates of 35%, 14% and 13%, respectively. This finding aligns closely with the current study.

Of the 60 patients who participated in the research, 71.7% received successful NIV treatment, avoiding the need for endotracheal intubation, while 28.3% experienced NIV failure. This outcome is nearly in line with a study by Kshatriya RM et al. (2019)[29], which reported a success rate of 74%. Our study's NIV success rate was comparable to that of Singh VK et al. (2006)[37]. Similar to our study, 74% of patients with COPD exacerbations placed on NIV in a multicentric study conducted in Europe between 1990 and 1991 by Brochard L et al. (1995)[5] did not require intubation and invasive ventilation. Plant PK et al. (2000)[32] reported a success rate of 84.7%, which is higher than the current trial, while Ambrosino N et al. (1995)[3] reported a success rate of 78% in the NIV group, which aligns more closely with our findings.

Both the successful and failed groups in the current investigation had the same baseline pH. The successful group's acidosis improved more statistically significantly than the failed group's after two hours of treatment (p=0.013). The successful group's pH improved more statistically significantly than the failure group's during all subsequent intervals. Numerous investigations of acute exacerbations of COPD have demonstrated that acidosis predicts death and is a measure of the degree of decompensation in acute hypercapnic respiratory failure.[37,38] There was no discernible difference between the successful and failure groups' baseline mean $PaCO_2$ values. Following two hours of therapy, the successful group's $PaCO_2$ levels were considerably lower than those of the failure group (p=0.007).

Throughout all subsequent intervals, the successful group's $PaCO_2$ values were considerably lower than those of the failure group ($p<0.01$). The baseline PO_2 level did not significantly differ between the successful and failure groups. The successful group's PO_2 level improved more statistically significantly than the failed group's after two hours of treatment ($p=0.018$). Over the course of the subsequent periods, the successful group's PO_2 level improved more statistically significantly than the failure group's ($p<0.01$).

In the current investigation, it was found that patients receiving non-invasive ventilatory support showed a considerable improvement in pH, $PaCO_2$ and PO_2. According to several authors, improvements in pH, PCO_2 and consciousness level during the first hour or two after NIV initiation are excellent markers of success (166). Similar findings were made in a study by Celikel T et al. (1998)[39], which indicated that the baseline values for pH, $PaCO_2$, PO_2 and respiratory rate had significantly improved with NIV. In the study by Bott J et al. (1993)[4], the pH increased while the controls decreased and the $PaCO_2$ decreased more in the NIV group. In the comparison of efficacy and mortality, the NIPPV group experienced a decrease in mortality. They therefore concluded that NIPPV significantly increased pH, decreased $PaCO_2$ and dyspnea and decreased mortality in patients with acute ventilatory failure brought on by COPD. According to a study by Kshatriya RM et al. (2019)[29], a favorable outcome was significantly correlated with improvements in baseline ABG parameters like pH, PCO_2 and PO_2 during or after 24 hours of NIV support. The pH levels of the two groups at the start and end of the trial differed significantly according to Abdelfattah RA et al. (2023)[40] with the success group having higher pH levels, just like in our study.

The poor result of NIV support was significantly influenced by a low pH and a high starting PCO_2. These findings corresponded with research by Ambrosino N et al. (1995)[3], which showed that carbon dioxide levels and acid-base imbalance during initial non-invasive ventilation trials determine treatment success probability and guide continuation decisions. Confalonieri M et al. (2005)[31] found that pH below 7.25 after one hour of non-invasive ventilation predicted higher failure rates. Supporting studies by Agarwal R et al. (2008)[41] and Anton A et al. (2000)[30] similarly advised considering intubation when non-invasive ventilation fails to improve pH and breathing rate within two hours. This evidence indicates that carbon dioxide elevation and acidemia severity after one hour serve as predictive indicators for non-invasive ventilation success in chronic obstructive pulmonary disease patients.

In the current investigation, there was no discernible difference in the mean HCO_3 level between the successful and failure groups at baseline, two hours later and twenty-four hours later. The successful group's mean HCO_3 level improved more statistically significantly than the failure group's after 24 to 48 hours of therapy ($p=0.031$). The successful group's mean HCO_3 level improved more statistically significantly than the failure group's at 48–72 and 72–96 hours (p values of 0.044 and 0.048, respectively). This was consistent with findings by Corrêa TD et al. (2015)[42], who found that one indicator that could indicate NIV failure was lower arterial bicarbonate levels. The mean number of NIV days in the present study was 2.69 ± 3.80 in the successful group and 0.92 ± 1.41 in the failure group ($p=0.018$). According to a study by Kshatriya RM et al. (2019)[29], patients in the success group received NIV for 2.72 ± 1.19 days. This result is nearly in line with their findings.

Conclusion:-

COPD represents a significant health burden for individuals above 40 years and poses ongoing future challenges. Disease flare-ups contribute substantially to illness severity and death rates. This investigation demonstrated that non-invasive respiratory support serves as an effective therapeutic approach for chronic obstructive pulmonary disease episodes accompanied by breathing failure, successfully preventing numerous patients from needing mechanical ventilation and related complications. Following two hours of non-invasive ventilation treatment, patients with favorable outcomes showed marked enhancement in pH, carbon dioxide partial pressure, and oxygen partial pressure values. The overall effectiveness rate of non-invasive ventilation reached 71.7%, consistent with published research findings. The most dependable indicators of treatment success were rapid improvements in blood gas measurements, specifically pH, carbon dioxide levels, and oxygen levels within the initial 2-hour period after starting non-invasive ventilation. Ongoing assessment of these indicators enables healthcare providers to distinguish patients who will benefit from continued non-invasive support from those requiring advancement to invasive ventilation. When implemented promptly with careful blood gas monitoring to direct treatment choices, non-invasive ventilation can minimize complications and fatalities associated with elevated carbon dioxide respiratory failure by decreasing the need for endotracheal intubation.

References:-

1. GBD Results Tool | GHDx [Internet]. Available from: http://ghdx.health data.org/gbd-results-tool
2. Soler J, Sánchez L, Latorre M, Alamar J, Román P, Perpiñá M. The impact of COPD on hospital resources: the specific burden of COPD patients with high rates of hospitalization. Arch Bronconeumol. 2001;37(9):375–381.
3. Ambrosino N, Foglio K, Rubini F, Clini E, Nava S, Vitacca M. Non-invasive mechanical ventilation in acute respiratory failure due to chronic obstructive pulmonary disease: correlates for success. Thorax 1995;50(7):755-7.
4. Bott J, Carroll MP, Conway JH, et al. Randomised controlled trial of nasal ventilation in acute ventilatory failure due to chronic obstructive airways disease. Lancet 1993;341(8860):1555-7.
5. Brochard L, Mancebo J, Wysocki M, et al. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. New England Journal of Medicine 1995;333(13):817-22.
6. Foglio C, Vitacca M, Quadri A, Scalvini S, Marangoni S, Ambrosino N. Acute exacerbations in severe COLD patients. Treatment using positive pressure ventilation by nasal mask. Chest 1992;101(6):1533-8.
7. Jeffrey AA, Warren PM, Flenley DC. Acute hypercapnic respiratory failure in patients with chronic obstructive lung disease: risk factors and use of guidelines for management. Thorax 1992;47(1):34-40.
8. Roberts CM, Stone RA, Buckingham RJ, Pursey NA, Lowe D. Acidosis, non-invasive ventilation and mortality in hospitalised COPD exacerbations. Thorax 2011;66(1):43-8.
9. Macklem PT. Hyperinflation. American Review of Respiratory Disease 1984; 129:1-2.
10. Tobin MJ, Perez W, Guenther SM, et al. The pattern of breathing during successful and unsuccessful trials of weaning from mechanical ventilation. American Review of Respiratory Disease 1986;134(6):1111-8.
11. Jaun G, Calverley P, Talamo C, Schnader J, Roussos C. Effect of carbon dioxide on diaphragmatic function in human beings. New England Journal of Medicine 1984;310:874-9.
12. Fagon JY, Chastre J, Hance A, Montravers P, Novara A, Gibert C. Nosocomial pneumonia in ventilated patients: a cohort study evaluating attributable mortality and hospital stay. American Journal of Medicine 1993;94:281-7.
13. Koenig SM, Truwit JD. Ventilator-associated pneumonia: diagnosis, treatment, and prevention. Clinical Microbiology Reviews 2006;19(4):637-57.
14. Waters B, Muscedere J. A 2015 update on ventilator-associated pneumonia: new insights on its prevention, diagnosis, and treatment. Current Infectious Disease Reports 2015;17(8):496.
15. Brochard L, Rauss A, Benito S, et al. Comparison of three methods of gradual withdrawal from ventilatory support during weaning from mechanical ventilation. American Journal of Respiratory and Critical Care Medicine 1994;150:896-903.
16. Esteban A, Frutos F, Tobin MJ, et al. A comparison of four methods of weaning patients from mechanical ventilation. Spanish Lung Failure Collaborative Group. New England Journal of Medicine 1995;332:345-50.
17. Kramer N, Meyer TJ, Meharg J, Cece RD, Hill NS. Randomized, prospective trial of noninvasive positive pressure ventilation in acute respiratory failure [see comments]. American Journal of Respiratory and Critical Care Medicine 1995;151(6):1799-1806.
18. Meduri GU, Conoscenti CC, Menashe P, Nair S. Noninvasive face mask ventilation in patients with acute respiratory failure. Chest 1989;95(4):865-70.
19. Guerin C, Girard R, Chemorin C, Varax R, Fournier G. Facial mask noninvasive mechanical ventilation reduces the incidence of nosocomial pneumonia. A prospective epidemiological survey from a single ICU. Intensive Care Medicine 1997;23:1024-32.
20. Kramer B. Ventilator-associated pneumonia in critically ill patients. Annals of Internal Medicine 1999;130:1027-8.
21. Nourdine K, Combes P, Carton MJ, Beuret P, Cannamela A, Ducreux JC. Does noninvasive ventilation reduce the ICU nosocomial infection risk? A prospective clinical survey. Intensive Care Medicine 1999;25:567-73.
22. Appendini L, Patessio A, Zanaboni S, et al. Physiologic effects of positive end-expiratory pressure and mask pressure support during exacerbations of chronic obstructive pulmonary disease. American Journal of Respiratory and Critical Care Medicine 1994;149(5):1069-76.
23. Nava S, Hill N. Non-invasive ventilation in acute respiratory failure. Lancet. 2009;374:250-259.
24. Barbé F, Togores B, Rubí M, Pons S, Maimó A, Agustí AG. Noninvasive ventilatory support does not facilitate recovery from acute respiratory failure in chronic obstructive pulmonary disease. Eur Respir J. 1996;9(6):1240-5.

25. Soo Hoo GW, Santiago S, Williams AJ. Nasal mechanical ventilation for hypercapnic respiratory failure in chronic obstructive pulmonary disease: determinants of success and failure. Critical Care Medicine 1994;22(8):1253-61.
26. Ambrosino N. Noninvasive mechanical ventilation in acute respiratory failure. European Respiratory Journal 1996;9:795-807.
27. Wood KA, Lewis L, Harz B, Kollef MH. The use of noninvasive positive pressure ventilation in the emergency department. Chest 1998;113(5):1339-46.
28. Shaheen M, Daabis RG, Elsoucy H. Outcomes and predictors of success of noninvasive ventilation in acute exacerbation of chronic obstructive pulmonary disease. Egyptian Journal of Bronchology. 2018 Sep;12:329-39.
29. Kshatriya RM, Khara NV, Oza N, Paliwal RP, Patel SN. A study of outcome of noninvasive ventilatory support in acute respiratory failure. Indian J Respir Care 2019;8:107-10.
30. Anton A, Guell R, Gómez J, et al. Predicting the result of noninvasive ventilation in severeacute exacerbations of patients with chronic airflow limitation. Chest 2000;117:828–833.
31. Confalonieri M, Garuti G, Cattaruzza MS, et al. Italian noninvasive positive pressure ventilation (NPPV) study group. A chart of failure risk for noninvasive ventilation in patients with COPD exacerbation. Eur Respir J. 2005;25(2):348-55.
32. Plant PK, Owen JL, Elliott MW. Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial. Lancet. 2000;355(9219):1931-5.
33. Ferrer M, Esquinas A, Leon M, et al. Non-invasive ventilation in severe hypoxemic respiratory failure: a randomized clinical trial. Am J Respir Crit Care Med 2003;168:1438.
34. Steriade AT, Johari S, Sargarovschi N, et al. Predictors of outcome of noninvasive ventilation in severe COPD exacerbation. BMC Pulm Med. 2019;19(1):131.
35. Vaudan S, Ratano D, Beuret P, Hauptmann J, Contal O, Garin N. Impact of a Dedicated Noninvasive Ventilation Team on Intubation and Mortality Rates in Severe COPD Exacerbations. Respir Care. 2015;60(10):1404-8.
36. Ongel EA, Karakurt Z, Salturk C, et al. How do COPD comorbidities affect ICU outcomes?. International journal of chronic obstructive pulmonary disease. 2014; 17:1187-96.
37. Singh VK, Khanna P, Rao BK, Sharma SC, Gupta R. Outcome prediction for noninvasive positive pressure ventilation in acute respiratory failure. JAPI. 2006;54:361–365.
38. Lightlower JV, Wedzicha JA, Elliott MW, et al. Non-invasive positive pressure ventilation to treat respiratory failure resulting from exacerbation of chronic obstructive pulmonary disease: Cochrane systematic review and meta-analysis. BMJ. 2003;326:185– 90.
39. Celikel T, Sungur M, Ceyhan B, Karakurt S. Comparison of noninvasive positive pressure ventilation with standard medical therapy in hypercapnic acute respiratory failure. Chest 1998;114:1636-42.
40. Abdelfattah RA, Ali YM, Abdel Aziz MO, Abdelaziz AO, Mohamed BI. Predictors of Success of Noninvasive Ventilation in Patients with COPD Exacerbations (Role of Clinical Parameters and Arterial Blood Gases). The Egyptian Journal of Hospital Medicine. 2023;91(1):3850-4.
41. Agarwal R, Gupta R, Aggarwal AN, Gupta D. Noninvasive positivepressure ventilation in acute respiratory failure due to COPD vs. othercauses: effectiveness and predictors of failure in a respiratory ICU in NorthIndia. Int J Chron Obstruct Pulmon Dis 2008;3:737–743.
42. Corrêa TD, Sanches PR, de Morais LC, et al. Performance of noninvasive ventilation in acute respiratory failure in critically ill patients: a prospective, observational, cohort study. BMC Pulm Med 2015;15:144.