ISSN: 0975-3583,0976-2833 VOL11, ISSUE 03, 2020

# Ventilator Associated Lung Injury in Pulmonary ICUs in Ain Shams University Hospital

**Tamer M. Ibraheem, MD; Mona Mansour Ahmed, MD; Dalia Abd El Sattar El Embaby, MD**

Department of Pulmonary Medicine, Faculty of Medicine, Ain Shams University, Cairo, Egypt

**Corresponding author:** Dalia Abd El Sattar El Embaby, **E-Mail:** dr.daliaelembaby333@gmail.com

## ABSTRACT

**Background:** Damage known as ventilator-associated lung injury (VALI) can occur when the lung is subjected to mechanical ventilation with the application of pressure, whether positive or negative. VALI may occur in previously normal lungs or worsen pre-existing ARDS. VALI can occur due to volutrauma, barotraumas, atelectrauma, biotrauma; Ventilator associated pneumonia (VAP) or ARDS.

**Objective**: To measure the incidence of ventilator associated lung injury in Pulmonary ICUs in Ain Shams University Hospitals in one year period span.

**Patients and methods:** This prospective observational study was conducted on all patients who were admitted to Pulmonary ICUs in Ain Shams University Hospitals in one year span and underwent mechanical ventilation for more than 48 hours. Data for factors related to the occurrence of ventilator associated lung injuries were collected after 48 hours of intubation.

**Results:** 120 patients were included in present study of which 45% females and 55% males with mean age of 56.14±13.32. Duration of MV was 11.14±12.04 days, and most of the modes of MV ACV and BIPAP modes. VALI occurred in 12.5% Ventilation related complications in 34.1%, with Volutrauma 40%, Biotrauma 26.7%, Atelactotrauma 13.3% and barotrauma 20%.

**Conclusion:** protective ventilation strategy is used routinely in Ain Shams University Pulmonary ICUs, making the incidence of VALI low despite the underlying lung pathology in the MV patients; high plateau Pressure and long duration of MV are important risk factors for development of VALI

**Keywords:** ARDS, VALI, Volutrauma**,** Biotrauma**,** barotraumas.

## INTRODUCTION

Damage known as ventilator-associated lung injury (VALI) can occur when the lung is subjected to mechanical ventilation with the application of pressure, whether positive or negative. VALI may aggravate pre-existing ARDS or affect previously healthy lungs. Patients on mechanical ventilation who develop VALI are more likely to get ARDS [(1)].

VALI can originate from either volutrauma, which is essentially lung injury brought on by an abnormally high tidal volume on a mechanical ventilator, or from a disease resembling adult respiratory distress syndrome [(2)]. Barotraumas, which include the rupture of the alveoli as a result of high transalveolar pressure. Pneumothorax, pneumomediastinum, pneumoperitoneum, and subcutaneous emphysema are frequent side effects [(3)].

Patients who use a mechanical ventilator for a prolonged amount of time might develop biotrauma, a severe inflammatory reaction, in their lungs [(4)]. Atelectotrauma, which is caused by the frequent, transitory closure and reopening of alveoli throughout the breathing cycle and results in damage to the alveoli [(5)]. Finally, there is ventilator-associated pneumonia (VAP), a serious consequence that has a death rate of 33-50%. It is said to happen in 10 to 25 percent of individuals who get mechanical ventilation. The risk of VAP is greatest very after upon intubation. According to estimates, VAP will happen at a rate of 3% per day for the first five days, 2% per day for the following five days, and 1% per day after that [(6)].

ISSN: 0975-3583,0976-2833 VOL11, ISSUE 03, 2020

Aim of the work was to measure the incidence of ventilator associated lung injury in Pulmonary ICUs in Ain Shams University Hospitals in one year period span.

## PATIENTS AND METHODS

This prospective observational study was conducted on all patients who were admitted to Pulmonary ICUs in Ain shams university hospitals in one year span and underwent mechanical ventilation for more than 48 hours.

Data for factors related to the occurrence of ventilator associated lung injuries were collected after 48 hours of intubation. Correlation between ventilation associated lung injury and ventilator mood, parameters and patient characteristics.

Radiology used in the study included CXR and chest US, Clinical Pulmonary Infection Score (CPIS) was used to serve as a surrogate tool to facilitate the diagnosis of ventilator-associated pneumonia (VAP) [(7)].

**Ethical consent:**

An approval of the study was obtained from Ain SHAMS University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of participation in the study.

**Statistical analysis:**

Statistical Package for Social Sciences (SPSS) version 22. Frequencies and relative percentages were used to depict qualitative data. To determine differences between two or more sets of qualitative variables, use the chi square test ($X^2$). Quantitative information was presented as mean±SD. Two independent groups of normally distributed variables were compared using the independent samples t-test (parametric data). P value less than 0.05 was regarded as significant.

## RESULTS

The study included 55% males and 45% females with Mean age 56.14±13.32, with duration of mechanical ventilation 11.14±12.04 and most of the modes of MV ACV and BIPAP modes. VALI occurred in 12.5% Ventilation related complications in 34.1%, with Volutrauma 40%, Biotrauma 26.7%, Atelactotrauma 13.3% and barotrauma 20%. Complications of mechanical ventilation among the studied cases was shown in figure (1) while of VALI among the studied cases was shown in figure (2). The factors that is related directly to development of VALI shows high statically significance between weight and development of VALI as shown in table (1)

**Table (1):** Correlation between VALI and socio-demographic data of the studied cases

| | **VALI (n=15)** | **Non VALI (n=105)** | **Test** | **P** |
|---|---|---|---|---|
| **Age (years)**<br>Minimum- maximum<br>Mean± SD | <br>32-72<br>52.73±13.56 | <br>18-73<br>56.62±13.28 | t 1.05 | 0.2 |
| **Gender** N (%)<br>Female<br>Male | <br>9 (60)<br>6 (40) | <br>45 (42.9)<br>60 (57.1) | $X^2$ 1.55 | 0.2 |
| **Weight (Kg)**<br>Minimum- maximum<br>Mean± SD | <br>50-90<br>70±15.11 | <br>40-140<br>93.28±24.85 | t 3.5 | 0.0006* |

Table (2) shows reveals a statically significant correlation between bronchiectasis as well as lung abscess and development of VALI.

ISSN: 0975-3583,0976-2833 VOL11, ISSUE 03, 2020

**Table (2):** Relation between comorbidites of the studied cases and VALI

| | VALI (n=15) | Non VALI (n=105) | $X^2$ | p |
|---|---|---|---|---|
| **Pulmonary (n=)** | | | | |
| BA | 0 | 5 (4.8) | 0.74 | 0.3 |
| Bronchiectasis | 2 (13.3) | 2 (1.9) | 5.3 | 0.02* |
| COPD | 0 | 15 (14.3) | 2.58 | 0.1 |
| Effusion | 1 (6.7) | 1 (0.9) | 2.61 | 0.1 |
| H1N1 | 0 | 3 (2.9) | 0.44 | 0.5 |
| ILD | 2 (13.3) | 6 (5.7) | 1.22 | 0.2 |
| Abcess | 2 (13.3) | 0 | 14.2 | 0.0002* |
| Pneumonia | 0 | 4 (3.8) | 0.59 | 0.4 |
| Pulmonary TB | 0 | 2 (1.9) | 0.29 | 0.5 |
| OHV | 0 | 22 (20.9) | 3.8 | 0.04* |
| Lung cancer | 1 (6.7) | 2 (1.9) | 1.22 | 0.2 |

Table (3) shows that the duration of MV was higher in VALI group in comparison to their peers and this was statically significant.

**Table (3):** Association between duration of mechanical ventilation of the studied cases and VALI

| | VALI (n=15) | Non VALI (n=105) | t-test | p |
|---|---|---|---|---|
| **Duration of MV (days)**<br>Minimum- maximum<br>Mean± SD | <br>2-94<br>18.73±24.73 | <br>2-45<br>10.05±8.6 | <br>t<br>2.67 | 0.008* |

Table (4) shows correlation between Tidal Volume, respiratory rate, FiO2, PEEP and peak pressure and development of VALI with high statically significance between plateau pressure and development of VALI.

**Table (4):** Parameters of mechanical ventilation of the studied cases and development of VALI

| | VALI (n=15) | Non VALI (n=105) | t-test | P |
|---|---|---|---|---|
| **TV (ml)**<br>Minimum- maximum<br>Mean± SD | (n=15)<br>300-500<br>447.8±64.8 | (n=76)<br>260-500<br>416.33±51.02 | <br>t<br>2.08 | 0.03* |
| **RR (breath/min) (n=110)**<br>Minimum- maximum<br>Mean± SD | (n=15)<br>12-18<br>13.46±1.76 | (n=95)<br>10-24<br>15.64±2.49 | <br>t<br>3.25 | 0.001* |
| **Fi O2 (%) (n=120)**<br>Minimum- maximum<br>Mean± SD | (n=15)<br>40-100<br>74±23.54 | (n=105)<br>30-100<br>52±19.77 | 3.9 | 0.0001* |
| **PEEP**<br>Minimum- maximum<br>Mean± SD | (n=15)<br>5-25<br>8.38±6.25 | (n=99)<br>0-12<br>6.6±2.38 | 2.04 | 0.04* |
| **Peak Pr (mmHg) (n=117)**<br>Minimum- maximum<br>Mean± SD | (n=15)<br>25-50<br>38±7.36 | (n=102)<br>12-45<br>34.09±6.92 | 2.02 | 0.04* |

ISSN: 0975-3583,0976-2833 VOL11, ISSUE 03, 2020

| | | | | |
|---|---|---|---|---|
| **Plateau pressure (mmHg) (n=117)** | (n=15) | (n=102) | 3.6 | 0.0005* |
| Minimum- maximum | 15-45 | 15-35 | | |
| Mean± SD | 28.53±7.91 | 22.08±6.25 | | |

# DISCUSSION

The most crucial kind of supportive care for patients with ARDS is mechanical ventilation, but this treatment can also cause or exacerbate lung damage, which is known as ventilator-induced lung injury (VILI) [(8)].

Mechanical ventilation for more than 48 hours puts the patient at higher risk to develop complications from MV. The age of the studied group ranged from 18 to 73 years, with the mean age 56 years. There was no significant correlation between age and development of VALI in the current study; however **Chelluri and Rotondi** [(9)] concluded that older age, in addition to functional status and comorbidities, was associated with increased complications and mortality. The weight of the studied cases ranged from 40 to 140 Kg, with the mean weight was 90.37. There was a strong correlation between weight and development of VALI (P value = 0.0006). The mean weight of the VALI group was of 93.28±24.85, in comparison to 70±15.11 in the non VALI group. In addition to affecting immunity, obesity is a chronically elevated source of pro-inflammatory cytokines and, like acute lung damage, reflects a pro-inflammatory state. Additionally, there is a reduction in lung and chest wall compliance, which affects pulmonary mechanics [(10)].

The duration of MV of the studied cases ranged from 2 to 94 days. The duration was higher in VALI group (mean 18.73 days) in comparison to their peers (mean 10.07) and this was statically significant (p value = 0.008). Our data revealed a statically significant correlation between bronchiectasis (P value=0.02) as well as lung abscess (P value=0.0002) and development of VALI. This can be attributed to the underlying restrictive and obstructive lung disease and the high plateau and peak pressures and decreased compliance in those patients at the onset of MV [(11)].

Tidal volumes were set around 5-8 mL/kg, respiratory rate around 12-15 breaths/min and PEEP around 5 $cmH_2O$. Protective ventilation strategy was used in all the studied cases. Thirty-four patients (28.3%) in the current study developed VAP. Clinical Pulmonary Infection Score (CPIS) was a useful tool in VAP diagnosis. CPIS in the studied cases ranged from 2 -8. In patients with CPIS > 6 VAP was likely. In this study, forty-one patients had positive sputum culture but not all of them had CPIS >6.

The most common organisms found in patients with VAP in our study were Acintobactar (19.5 %), methicillin resistant Staphylococcus aureus (MRSA) (19.5%), pseudomonas (12.2%) and Klebsilla (12.2 %). Also, a statistically significant correlation were found between pseudomonas (P value=0.02) as well as staph (P value=0.02) and development of VALI, as staph and pseudomonas infections are associated with lung cavitations that lower lung compliance and increase the risk of VALI [(12)]. The diagnosis of biotrauma was done by exclusion of other causes that lead to systemic inflammatory response; this may be inaccurate way for diagnosis of biotrauma because the definite diagnosis needs postmortem pathology which was not available.

Univariate analysis revealed high peak inspiratory pressure, increasing positive end-expiratory pressure, and TV to be significant risk factors. Major non ventilator risk factors for ARDS included sepsis, low pH, elevated lactate, low albumin, and transfusion of packed RBCs, transfusion of plasma, high net fluid balance, and low respiratory compliance [(12)].

# CONCLUSION

Protective ventilation strategy is used routinely in Ain Shams University Pulmonary ICUs, making the incidence of VALI low despite the underlying lung pathology in the MV patients; high plateau Pressure and long duration of MV are important risk factors for development of VALI.

ISSN: 0975-3583,0976-2833 VOL11, ISSUE 03, 2020

**Supporting and sponsoring financially:** Nil.

**Competing interests:** Nil.

## REFERENCES

1. Parker JC, Hernandez LA, Peevy KJ (2010). Mechanisms of ventilator-induced lung injury. Crit Care Med 21(1):131-43.
2. Albaiceta GM, Blanch L (2011). Beyond volutrauma in ARDS: the critical role of lung tissue deformation. Crit Care 15 (2): 304. doi:10.1186/cc10052. PMC 3219320.PMID 21489320.
3. Doelken P, Sahn SA. (2008). Pleural disease in the critically ill patient. In: Intensive Care Medicine, 6th ed, Irwin RS, Rippe JM (Eds), Lippincott, Williams, and Wilkins, Philadelphia. p.636.
4. Halbertsma FJ, Vaneker M, Scheffer GJ, van der Hoeven JG (2015). Cytokines and biotrauma in ventilator-induced lung injury: a critical review of the literature. Neth J Med 63 (10): 382–92. PMID 16301759.
5. Dreyfuss D, Saumon G. (1998). Ventilator-induced lung injury: lessons from experimental studies. Am J Respir Crit Care Med ; 157: 294–323
6. Mutlu GM, Mutlu EA, Factor P. (2011). GI complications in patients receiving mechanical ventilation. Chest. 119(4):1222-41.
7. Chastre J, Fagon J-Y. Ventilator-associated pneumonia. American journal of respiratory and critical care medicine. 2012;165(7):867-903.
8. Slutsky AS, Ranieri VM. Ventilator-induced lung injury. New England Journal of Medicine. 2013;369(22):2126-36.
9. Chelluri L, Rotondi AJ, Sirio CA, Donahoe MP. 2-month mortality and functional status of critically ill adult patients receiving prolonged mechanical ventilation. Chest. 2012;121 (2):549.
10. Gong MN, Bajwa EK, Thompson BT, Christiani DC. Body mass index is associated with the development of acute respiratory distress syndrome. Thorax. (2010) 65:44–50.
11. Gajic O, Dara SI, Mendez JL, Adesanya AO, Festic E, Caples SM, et al. Ventilator-associated lung injury in patients without acute lung injury at the onset of mechanical ventilation. CRITICAL CARE MEDICINE-BALTIMORE- 2014;32(9):1817-24
12. Jia X, Malhotra A, Saeed M, Mark RG, Talmor D. Risk factors for ARDS in patients receiving mechanical ventilation for> 48 h. CHEST Journal. 2018; 133(4):853-61.