Non Invasive Predictors for Decision Making in Acute Exacerbation of Chronic Obstructive Pulmonary Disease (AECOPD)

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Abstract

Introduction: Accurate prediction for prognosis is important for hospitalized patients with acute exacerbation of chronic obstructive pulmonary disease (AECOPD) requiring mechanical ventilation (MV) and for proper assessment of decision making regarding plane of management and ongoing hospital morbidity and mortality.

Aim of the Present Study: The present study was designed to determine the predictability of non invasive parameters including APACHE II score, arterial blood gases (ABGs) and bedside Echocardiography in management of critically ill patients with COPD exacerbation either invasively or conservatively.

Patients and Methods: The study was conducted on 60 adult patients (50 male & 10 female) with AECOPD, with mean age 60.1 ± 6.2 were admitted to intensive care unite (ICU). All patients were subjected to arterial blood gases (ABGs), APACHE II score and bedside Echocardiography study.

Results: Patients were divided into two groups according to ventilatory requirement invasive or non invasive into group I (34 patients) with need of MV and group II (26 patients) with success of conservative treatment. We found that APACHE II score, had a high predictive value for MV necessity, it was 19.17 ± 3.4 in the group I Vs 11.46 ± 4.4 in the group II also Doppler evidence of pulmonary hypertension had high predictive value for MV necessity.

The mean value of PASP was 48.95 ± 12.44mmHg in group I, Vs 30.71 ± 6.5mmHg in group II with significant p value. The mean value of PAPm was 42.08 ± 6.89mmHg in group I Vs 31.50 ± 7.11mmHg in group II with significant p value. Increases in the APACHE II score, the mean pulmonary artery pressure (PAPm) and pulmonary artery systolic pressure (PASP) were significantly more in patients who died in comparison to survivors in the group I.

Conclusion: APACHE II score, bedside Echocardiography and routine arterial blood gases could be used as a marker to identify patients at the time of admission who are likely to have a poor prognosis, so that such patients can be managed aggressively, either medical conservative treatment or mechanical ventilation (NIPPV or Invasive MV).

Key Words: COPD – APACHE II score – Mortality.

Introduction

**MORTALITY** associated with hospitalization for chronic obstructive pulmonary disease (COPD) exacerbation can be considerable. In-hospital mortality is more than 20% among patients who require admission to intensive care unit (ICU) [1,2]. Hospital acquired infections, weaning failure and prolonged mechanical ventilation leading to increase the mortality in these patients. Prospective randomized controlled studies have shown that addition of noninvasive mechanical ventilation (NIMV) to standard treatment reduces the need for endotracheal intubation, lowers hospital mortality and shortens the length of stay in selected patients with COPD [3,4,5]. There have been many studies that investigate the factors associated with ICU mortality in patients with COPD such as older age, higher Acute Physiology Assessment and Chronic Health Evaluation scores (APACHE II), comorbidities, ventilator associated pneumonia (VAP), severity of airflow limitation and prolonged mechanical ventilation (MV) have been reported as risk factors [6].

In acute exacerbations of COPD, pH is the best marker of severity and reflects an acute deterioration in alveolar hypventilation compared with the chronic stable state [7]. Regardless of the chronic level of arterial carbon dioxide tension (PaCO₂), an acute rise in PaCO₂ due to worsening alveolar hypoventilation is associated with a fall in pH which was included in APACHE II score [8].
The COPD is characterized by slowly progressive airflow obstruction, resulting in dyspnoea and exercise limitation and pulmonary hypertension which is a major cardiovascular complication.

Although the true prevalence of pulmonary hypertension is unknown, an elevation of pulmonary pressure is reported to occur in 5-40% of patients in series of selected individuals with severe COPD undergoing right heart catheterization [9]. The magnitude of pulmonary hypertension measured invasively correlates with indices of airflow obstruction and gas exchange, but the relationship is weak [10]. But dramatic elevations of pulmonary arterial pressure are observed on exercise, during nocturnal desaturations and during acute exacerbations [11,12]. Pulmonary pressure is an important prognostic indicator in COPD, which has been reported by many studies demonstrating that Pulmonary hypertension predicts the number of exacerbations and length of hospital stay in COPD patients [13]. Multiple studies have confirmed that elevated pulmonary arterial pressure is a predictor of mortality independent of airflow limitation in COPD [14,15].

**Aim of the present study:**

The present study was designed to determine the predictability of non invasive parameters including APACHE II score, arterial blood gases (ABGs) and bedside echocardiography in management of critically ill patients with COPD exacerbation either invasively or conservatively.

**Patients and Methods**

The study was conducted on 60 adult patients (50 males & 10 females) with acute respiratory failure on top of chronic obstructive pulmonary disease. Patients were admitted to critical care medical department in Cairo and Beni-Swief Universities over the period from November 2004 to March 2006.

Patients were classified into two groups according to the outcome of the first aid managements in ICU.

**Group I:** Included patients who required mechanical ventilation (invasive or non-invasive) due to failure of conservative treatment. Endotracheal intubation was done due to deterioration of the level of consciousness, severe respiratory acidosis, unstable hemodynamics, or failure of NIMV.

**Group II:** Included patients with good response to conservative treatment and did not required mechanical ventilation.

Patients with Major systemic diseases e.g. hepatic dysfunction, renal impairment, neurological disorder or other significant illness and in whom the primary cause of respiratory failure was pulmonary edema or pulmonary embolism were excluded from the study.

**All patients were subjected to:**

Full clinical evaluation, routine lab investigation, chest X-ray, Arterial blood gases (ABG), Bedside Doppler Echocardiography to assess (RVEDD, RVESD, RV EF and mean PAP) and APACHE-II score [16].

**Results**

Sixty adult patients suffering from acute exacerbation of chronic obstructive lung disease were including in the study, the patients were classified into two groups according to the outcome of the first aid managements in ICU. The clinical and demographic data of the studied patients illustrated in Table (1).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (N=34)</th>
<th>Group II (N=26)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, (years)</td>
<td>63±6.84</td>
<td>61.3±5.95</td>
<td>NS</td>
</tr>
<tr>
<td>Sex:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>28 (82.4%)</td>
<td>22 (84.6%)</td>
<td>NS</td>
</tr>
<tr>
<td>F</td>
<td>6 (17.6%)</td>
<td>4 (15.4%)</td>
<td></td>
</tr>
<tr>
<td>Heart rate (beat/min)</td>
<td>101.29±17.75</td>
<td>106.15±17.38 NS</td>
<td></td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>76.15±10.03</td>
<td>88.88±14.02 NS</td>
<td></td>
</tr>
<tr>
<td>Respiratory rate, (breath/min)</td>
<td>37.05±4.25</td>
<td>28.15±5.33 0.0001</td>
<td></td>
</tr>
<tr>
<td>APACHE-II</td>
<td>19.17±3.43</td>
<td>11.46±4.42 0.0001</td>
<td></td>
</tr>
<tr>
<td>Clinical RSHF</td>
<td>24 (70.6%)</td>
<td>10 (38.5%) 0.01</td>
<td></td>
</tr>
</tbody>
</table>

MAP: Mean arterial blood pressure.
RSHF: Right sided heart failure.

There was significant increase in mean respiratory rate 37.05±4.25 in group I Vs 28.15±5.33 in group II (p=0.0001), APACHE-II score 19.17±3.43 in group I, Vs 11.46±4.42 in group II (p=0.0001), and the clinical sings of RSHF was found in 24 (70.6%) patients in group I and in 10 (38.5%) patients in group II (p=0.01).

**Blood gases at admission in both groups:**

There were a highly significant reduction in PaO2/FIO2 ratio, pH, with an increase in PaCO2 and HCO3 in group I when compared with the group II.

The mean value of PaO2/FIO2 ratio was 214.29±29.16 in group I Vs 230.46±21.18 in group II, the mean value of PaCO2 was 87.51±13.40 in group
I: 58.85±7.78 in group II and the mean value of pH was 7.22±0.073 in group I V 7.33±0.06 in group II, while mean value of HCO₃⁻1mmol/L was 41.94±4.27 in group I Vs 36.01±6.11 in group II. Table (2).

Table (2): Comparison between both groups regarding the blood gases analysis on admission.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (N=34)</th>
<th>Group II (N=26)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂/FIO₂ ratio</td>
<td>214.29±29.16</td>
<td>230.46±21.18</td>
<td>0.02</td>
</tr>
<tr>
<td>PaCO₂, mmHg</td>
<td>87.51±13.40</td>
<td>58.85±7.78</td>
<td>0.0001</td>
</tr>
<tr>
<td>pH</td>
<td>7.22±0.073</td>
<td>7.33±0.06</td>
<td>0.0001</td>
</tr>
<tr>
<td>HCO₃⁻1 mmol/L</td>
<td>41.94±4.27</td>
<td>36.01±6.11</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Doppler echocardiographic parameters at admission in both groups:

There were highly significant increases in RVEDD, RVESD, pulmonary artery systolic pressure (PASP) and mean pulmonary artery pressure (PAPm) in group I.

The mean value of RVEDD was 36.29±10.44mm in group I, Vs 25.60±9.67mm in group II. The mean value of RVESD was 29.52±8.11mm in group I, Vs 20.45±8.37mm in group II. The mean value of PASP was 48.95±12.44mmHg in group I, Vs 30.71±6.5mmHg in group II. The mean value of PAPm was 42.08±6.89mmHg in group I, Vs 31.50±7.1mmHg in group II (Table 3).

Table (3): Comparison of bedside Echocardiographic parameters in both groups at admission.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group I (N=34)</th>
<th>Group II (N=26)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVEDD, (mm)</td>
<td>36.29±10.44</td>
<td>25.60±9.67</td>
<td>0.0001</td>
</tr>
<tr>
<td>RVESD, (mm)</td>
<td>29.52±8.11</td>
<td>20.45±8.37</td>
<td>0.0001</td>
</tr>
<tr>
<td>EF.RV%</td>
<td>43.22±5.58</td>
<td>45.44±5.71</td>
<td>NS</td>
</tr>
<tr>
<td>PASP, (mmHg)</td>
<td>48.95±12.44</td>
<td>30.71±6.5</td>
<td>0.0001</td>
</tr>
<tr>
<td>PAPm, (mmHg)</td>
<td>42.08±6.89</td>
<td>31.50±7.1</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Data were shown as mean±SD. Unpaired t-test was used.

NS: Nonsignificant.

RVEDD: Right ventricle end-diastolic diameters.

RVESD: Right ventricle end systolic diameters.

EF.RV: Ejection fraction of right ventricle.

PASP: Pulmonary artery systolic pressure.

PAPm: Mean pulmonary artery pressure.

Potential predictors and the need for MV:

There was significant positive correlation between the need of MV and APACHE-II (r=0.72, p=0.0001) and PASP (r=0.73, p=0.0001) Table (4).

Table (4): Correlation of the potential predictors and the need for MV.

<table>
<thead>
<tr>
<th>Variable</th>
<th>r-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE-II</td>
<td>0.72</td>
<td>0.0001</td>
</tr>
<tr>
<td>PASP, (mmHg)</td>
<td>0.73</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

PASP: Pulmonary artery systolic pressure.

ROC curve (Figs. 1,2) showed that all these variables have good predictive capability with area under the ROC curve (AUC) of 0.84 (p=0.0001) for APACHE II score and 0.869 (p=0.0001) for PASP (Table 5).

Table (5): Area under ROC curve (AUC) of different predictors to predict the need for MV.

<table>
<thead>
<tr>
<th>Variable</th>
<th>AUC</th>
<th>Significant</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE-II</td>
<td>0.84</td>
<td>0.0001</td>
</tr>
<tr>
<td>PASP, (mmHg)</td>
<td>0.869</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Accurate diagnostic predictive value if area under ROC curve <0.60. Highly accurate diagnostic predictive value if area under ROC curve <0.75.

PASP: Pulmonary artery systolic pressure.

A cut off at 16 on ROC curve of APACHE-II had sensitivity (true positive prediction) of 94% and specificity (true negative prediction) of 85% with positive predictive value (positive prediction in all positive values) of 88.8%, negative predictive value (negative prediction in all negative values) of 84.6% and accuracy (true positive and negative prediction) of 90%.
A cut off at 36.6 on ROC curve of PASP mmHg had sensitivity (true positive prediction) of 82% and specificity (true negative prediction) of 70% with positive predictive value (positive prediction in all positive values) of 80.3%, negative predictive value (negative prediction in all negative values) of 79.3% and accuracy (true positive and negative prediction) of 85%.

Best cut-off, taken in Table (6) as the value on the ROC curve at the point where the curve sharply angulated.

Table (6): Accuracy of different cut off points of different predictors.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Cut off</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE-II</td>
<td>16</td>
<td>94%</td>
<td>85%</td>
<td>88.8%</td>
<td>84.6%</td>
<td>90%</td>
</tr>
<tr>
<td>PASP</td>
<td>36.6</td>
<td>82%</td>
<td>70%</td>
<td>80.3%</td>
<td>79.3%</td>
<td>85%</td>
</tr>
</tbody>
</table>

PPV: Positive predictive value. NPV: Negative predictive value.

Data of non invasive versus invasive ventilation in group I:

There was a significant increases in the total duration of mechanical ventilation, in APACHE-II score and decrease in pH value in invasively ventilated patients when compared to non invasively ventilated patients at the time of starting MV. There were no significant difference in PaCO₂ and PaO₂/FIO₂ ratio, while at the time of starting MV, APACHE-II score was 21.1 ± 3.14 Vs 26.2 ± 2.8 (p=0.0001), PH was 7.23 ± 0.07 Vs 7.12 ± 0.79 (p=0.001), PaCO₂ was 91.5 ± 6.8mmHg Vs 96 ± 6.4 mmHg and PaO₂/FIO₂ ratio was 167 ± 30.16 Vs 155 ± 16.9 for noninvasive and invasive MV, respectively. The total duration of the two ventilation modalities was 5.04 ± 3.77 days Vs 9.55 ± 3.7 days for noninvasive and invasive MV, respectively (p=0.003).

The mean time of start of MV was 11.82 ± 2.95 range as (1-48) hours. Ten patients (29%) required invasive ventilation and twenty four patients (71%) required noninvasive ventilation in the group I.

Table (7): Comparison between non invasive and invasive ventilation regarding duration of MV, blood gases and APACHE-II.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Noninvasive ventilation (N=24)</th>
<th>Invasive ventilation (N=10)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE-II</td>
<td>21.16±3.14</td>
<td>26.22±2.85</td>
<td>0.0001</td>
</tr>
<tr>
<td>PH</td>
<td>7.23±0.072</td>
<td>7.12±0.79</td>
<td>0.001</td>
</tr>
<tr>
<td>PaCO₂ (mmHg)</td>
<td>91.58±6.28</td>
<td>96±6.49</td>
<td>NS</td>
</tr>
<tr>
<td>PaO₂/FIO₂ ratio</td>
<td>167±30.16</td>
<td>155±16.9</td>
<td>NS</td>
</tr>
<tr>
<td>Duration of MV (days)</td>
<td>5.04±3.77</td>
<td>9.55±3.7</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Chi square test was used.

Pre- diction of mortality in group I:

There was significant positive correlation between the mortality in group I and APACHE-II score (r=0.38, p<0.02) & PASP (r=0.73, p<0.0001).

Table (8): Mortality rate and survival rate in non invasive versus invasive ventilation in the failure group.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Noninvasive ventilation (N=24)</th>
<th>Invasive ventilation (N=10)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality rate</td>
<td>4 (16.7%)</td>
<td>6 (60%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Survival rate</td>
<td>20 (83.3%)</td>
<td>4 (40%)</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Discussion:

Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality and is an important health economic problem. Since 1960, there has been an increase in mortality associated with COPD, especially in men. Recently, mortality from COPD, especially among women, has increased further [17].

Assessment of therapeutic response and prediction of prognosis are two of the major goals of bedside respiratory monitoring, determining the factors predicting need for mechanical ventilation and mortality which is interested to intensivists, because early identification of patients at high risk is essential, as well as tailoring monitoring, therapy and counseling families [18].

The aim of our study is to detect the possible predictor factors for the need for MV in COPD patients with acute exacerbation, as well as the predictor of the outcome and also to assess which patient can be managed by noninvasive ventilation versus invasive ventilation.

Prediction for mechanical ventilation in AECOPD:

We found that APACHE II score, had a high predictive value for MV necessity, it was 19.17 ± 3.4 in the group I and 11.46 ± 4.4 in the group II. The
area under the ROC curve (AUC) of APACHE II was 0.84 ($p=0.0001$), the accuracy of a cut off APACHE-II was 90% and it can predict the need for MV in 94% of the patients in the group I.

Vitacca [19] et al., 1996 confirmed our results, they found that the APACHE II was significant in predicting the need for MV. In a prospective study by Irfan [18] et al., 2006, over 656 admissions with AECOPD, they found that the high APACHE II could predict the need for MV. They used data obtained over the first 24h of respiratory intensive care unit admission. The higher APACHE-II score was determined as factor associated with intubation.

On the other hand, our results do not agree with the study by Breen [20] et al., 2002 in which, the only independent predictor for the requirement of mechanical ventilation was premorbid FEV1, while PaO2, PaCO2, pH, FiO2 and APACHE II scores were not predictive. This apparent paradox may suggest that, in patients with acute respiratory failure and COPD, the need for ventilation relates more to the underlying respiratory reserve than the magnitude of measurable physiological disturbance on presentation to the ICU.

In our study different physiological parameters estimated at the time of presentation were analyzed to find suitable predictors of the need for MV. Most of the parameters included in the APACHE II score were found to be independent predictors as PaO2, pH, Serum sodium, respiratory rate, leucocytic count, hematocrit and mean blood pressure. In acute exacerbations of COPD, pH is the best marker of severity and reflects an acute deterioration in alveolar hypoventilation compared with the chronic stable state [7].

Regardless of the chronic level of arterial carbon dioxide tension (PaCO2), an acute rise in PaCO2 due to worsening alveolar hypoventilation is associated with a fall in pH which was included in APACHE II score [8]. Our results demonstrated that pH value was 7.22±0.07 in the group I compared with 7.33±0.06 in the group II at the time of admission. This result is in agreement with the result of the study by Vitacca [19] et al., 1996, who found that the pH in the failure group was less than in the success group at the time of admission. Also a study by Khilnani and Amit [21] 2004 demonstrated that the pH <7.26 was an independent predictor of need for MV in patients with COPD-ARF and $p<0.05$. Liu [22] et al., 2007 had a cohort of 138 patients with COPD requiring IMV ≥12 hours for acute respiratory failure in COPD patients requiring IMV, the post intubation pH value can not only reflect patients response to treatment, but also serve as an independent determinant of hospital mortality apart from other risk factors such as a higher preintubation APACHE II score and development of MODS. A close correlation between the response to IMV and prognosis was proved in these patients.

Echocardiographic parameters:

In our study, the following echocardiographic parameters had a strong diagnostic predictive value for the need of mechanical ventilation, the increase in RVEDD, RVESD, PASP, PAPm.

In our results, when COPD is so severe enough to produce pulmonary hypertension and cor-pulmonale, patients are more likely to need mechanical ventilation during their acute exacerbations as evidenced by the ROC curve analysis for the PASP as at a cut off 36.6mmHg, the accuracy was 85% and can predict the need for MV in 82% of patients in the group I.

Our echocardiographic parameters are in agreement with a study done by Nandakumaran [23] et al., 2004. Also Higham [24] et al., 2001, concluded that pulmonary arterial hypertension is common in severe COPD. In view of the adverse effects of Pulmonary hypertension on morbidity and mortality routine echocardiography in patients with severe COPD may be warranted.

Kessler and his colleagues [13] in 1999 demonstrated that pulmonary hypertension and chronic hypercapnic respiratory insufficiency are predictive factors of hospitalization for acute exacerbation in COPD patients. Also the presence of pulmonary hypertension in COPD is also associated with a poorer clinical evolution and more frequent use of healthcare resources [28].

The pulmonary arterial pressure is an important prognostic indicator in COPD, which has been underlined by a recent study demonstrating that pulmonary hypertension predicts the number of exacerbations and length of hospital stay in COPD patients [26]. So, the development of pulmonary hypertension affects both mortality and morbidity.

Outcome in the Group I:

The overall mortality rate of our patients was (16.5%) and the total mortality rate was 29% in the group I. Four patients (16.7%) and six patients (60%) for non invasively ventilated and invasively ventilated respectively died. The final causes of death were: Four with severe pneumonia, one with severe gastric bleeding and five patients with multi-organ failure (MOF).
Weiss & Hudson [27] 1994 reviewed 11 studies carried out to study outcome of patients with exacerbation of COPD and they found that the combined mortality rate to be 20.3%. Direct comparison of the mortality between studies is difficult, as the criteria for inclusion, medical therapy and moreover, indication and use of MV between countries are different.

The overall mortality rate was near to the mortality rate reported by Vitacca [19] et al., 1996 which was 15% and it is higher than that reported by Jeffrey [28] et al., 1992 which was 12%. Also our study is in agreement with Assimo [29] et al., 1998. Overall mortality rate in the study of Khilnani and Amit [24] et al., 2004 is not in agreement with our mortality rate, it was 36.6%, this was due to the high incidence of the need for MV (84.1%) in comparison to our study in which the rate of MV was (56.5%). Differences in disease severity and diversity of the patients may partly explain the variations in hospital outcome.

Our study showed that increase in the APACHE II score and increase in the PAPm and PASP were significantly more in patients who died in comparison to survivors in the Group I and there was no significant difference in PaCO2. Warren [30] et al., 1980 and Jeffrey [28] et al., 1992, reported that mortality was associated with increasing age and low pH, pH of <7.26 being associated with a particularly poor prognosis. Also Hypoxia and hypercapnia were not different between the survivors and those who died.

In our study, PaCO2 has not been shown to be helpful in predicting mortality in patients with acute respiratory failure, this was confirmed by previous studies by Costello [31] et al., 1997 and Nava [32] et al., 1998 but in the study of Breen [20] et al., 2002, analysis of PaCO2 and APACHE II score were significant.

Earlier studies have also found that APACHE II score is useful in predicting mortality in COPD patients with acute exacerbation although the timing of scoring after admission has varied in different studies [1]. In the present study, analysis of APACHE II scoring done at the time of admission to ICU revealed significant difference for patients in the Group I who died in comparison to those patients who survived. This was confirmed by the study done by Khilnani [21] et al., 2004. In which, the APACHE II score at admission to ICU was 20 in those patients who died versus 18 in those who survived (p<0.02) and this value was found to be significant for mortality in the ventilated group. Nevins and Epstein [33], 2001 found APACHE II score at 6 hrs after initiation of ventilation to be a useful predictor of mortality.

The study done by Irfan [18] et al., 2006, confirmed our results. The median APACHE II score was 26 in those patients who died versus 22 in those who survived (p<0.001) and they attributed the higher mortality rate in their study to the greater severity of the illness and higher APACHE II score.

Our echocardiographic finding are in agreement with Traver [34] et al., 1979 who showed that, after correcting for age, the presence or absence of cor pulmonale was one of the best predictors of mortality. Also with Weitzenblum [35], 2003 and Burgess [36] et al., 2002.

The severity of pulmonary hypertension correlates closely with survival in chronic lung disease. When COPD is complicated by cor-pulmonale, about 50% of patients die within 7 years, whereas patients without cor-pulmonale survive an average of 13.5 years [37].

Large studies have confirmed that elevated pulmonary arterial pressure is a predictor of mortality independent of airflow limitation in COPD Prichard [38] 1996.

Conclusion:

from our study and the previous studies discussed before, there are several indices which can be done at the time of admission for the COPD patients with acute exacerbation as a routine investigation for ICU admission which includes: APACHE II score, bedside Echocardiography and routine arterial blood gases to identify patients at the time of admission who are likely to have a poor outcome, so that such patients can be managed aggressively. Also, to predict which patient can be managed with medical treatment or MV either (NIPPV or Invasive MV).

References


