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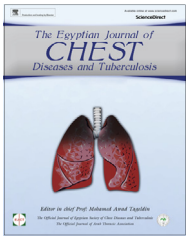

ORIGINAL ARTICLE

Prognostic role of serum uric acid in acute respiratory distress syndrome patients: A preliminary study

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KEYWORDS

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Abstract *Background:* ARDS mortality is still high, many biomarkers had been used to predict the mortality but they may have many drawbacks because of its validity, complications and cost.

Study design: Observational study was planned to evaluate the predictive role of serum uric acid level in ARDS outcome. Mortality was the primary end-point while secondary endpoints included total ICU stay, duration of mechanical ventilation and the presence or absence of complications.

Aim: The aim of this work is to study the role of serum uric acid level as an outcome predictor in ARDS patients.

Patients and methods: Thirty three ARDS patients were enrolled in this study according to Berlin 2012 definition. Patients with diabetes mellitus, chronic renal failure, cardiovascular disorders, decompensated liver disease and known malignancies were excluded from the study. On admission to ICU serum uric acid level was investigated.

Results: Sensitivity and specificity of uric acid as an outcome predictor at a cut off of 8.4 mg/dl were 89% and 80% respectively; the area under curve was 0.88 with p value <0.001, mortality in a high uric acid group that reported to be 86.7% was statistically significant higher than the normal uric acid group 38.9%.

Conclusion: Serum uric acid level at 8.4 mg/dl cut off point predicts mortality in ARDS patients with 89% sensitivity and 80% specificity.

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Introduction

The acute respiratory distress syndrome (ARDS) was defined in 1994 by the American–European Consensus Conference (AECC) [1]. Since then, issues regarding the reliability and validity of this definition have emerged. Using a consensus process, a panel of experts convened in 2011 (an initiative of the European Society of Intensive Care Medicine endorsed by the American Thoracic Society and the Society of Critical Care Medicine) developed Berlin definition, focusing on feasibility, reliability and validity [2]. Recent consensus group made a number of changes to the previous American–European Consensus Conference definition of ARDS which includes: timing within one week of a known clinical insult or new or worsening respiratory symptoms, chest imaging of bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodules, the origin of edema and respiratory failure are not fully explained by cardiac failure or fluid overload which needs objective assessment (e.g., echocardiography) to exclude hydrostatic edema if no risk factor is present; and lastly oxygenation: mild PaO_2/FIO_2 $\leqslant$300–200 mmHg with PEEP or CPAP $\geqslant 5$ cmH_2O; moderate: $\leqslant$200–100 mmHg with PEEP $\geqslant 5$ cmH_2O and severe: PaO_2/FIO_2 $\leqslant$100 mmHg with PEEP $\geqslant 5$ cmH_2O [3].

ARDS is a life threatening respiratory condition characterized by hypoxemia and stiff lungs, without mechanical ventilation most patients would die [4]. A complex network of cytokines and other pro-inflammatory compounds initiate and amplify the inflammatory response in acute respiratory distress syndrome. New evidence indicates that it is not only the production of pro-inflammatory cytokines that is important, but also the balance between pro-inflammatory and anti-inflammatory mediators [4]. Since its first description in 1967, there have been a large number of studies addressing various clinical aspects of the syndrome (risk factors, epidemiology and treatment) as well as studies addressing its pathogenesis (underlying mechanisms, biomarkers and genetic predisposition). The lack of therapeutic modalities is certainly related to the complex pathogenesis of this syndrome with multiple signaling pathways activated depending on the type of lung injury. In addition, the lack of sensitive and specific diagnostic criteria to diagnose ARDS has hampered progress [3]. Serum uric acid is the final product of purine degradation [5], which increases significantly during hypoxia [6]. Increased level of uric acid in respiratory disorders, including obstructive sleep apnea, pulmonary hypertension and COPD was reported in several studies [7,8].

Impaired pulmonary function reduces oxygen intake resulting in tissue hypoxia which is more prominent during acute exacerbation in COPD, this may lead to increased circulating uric acid levels originating from both lung and peripheral tissue damage [9]. Elevated uric acid levels have been associated with the presence of systemic inflammation [10] and increased cardiovascular risk [11]. Elevated uric acid levels have been associated with increased levels of inflammatory markers (e.g. CRP and interleukin-6) [12].

ARDS related mortality is still high, many biomarkers had been used to predict mortality but they may have many drawbacks because of its validity, complications and cost. To date, as far as we know, no studies have evaluated the role of serum uric as an outcome predictor in ARDS patients. Therefore, this cohort observational study was conducted to evaluate the possible role of serum uric acid as a biomarker for outcome prediction in ARDS patients. Mortality was the primary endpoint while secondary endpoints included total ICU stay, duration of mechanical ventilation and presence or absence of complications.

Patients and methods

After approval from the departmental ethics committee, this cohort observational study was conducted in the pulmonary critical care unit, Mansoura University Hospitals during the period from July 2013 to August 2014 including 11 females and 22 males with ages ranging from 18 to 50 years suffering from ARDS according to the Berlin 2012 definition, which is based on history, X-ray opacities, exclusion of cardiac causes of pulmonary edema and PaO_2/FIO_2 ratio [1]. Patients with diabetes mellitus, chronic renal failure, cardiovascular disorders, decompensated liver disease and known malignancies were excluded from the study. Plain chest X-ray, trans-thoracic echocardiography was done to exclude patients with cardiovascular disorders.

Admission APACHEII score was calculated. Blood samples were collected from each patient on admission and prior to initiation of any treatment for basic serum uric acid and standard laboratory measurements (complete blood count, serum creatinine and blood gases). Uric acid was measured using enzymatic colourimetric method. Uric acid values above or equal to 6.4 mg/dl were considered the high uric acid group and below it were the normal uric acid group. Mortality rate was reported. Total ICU stay, duration of mechanical ventilation and presence or absence of complications were also reported, consent was taken from first-degree patient relative.

Statistical analysis

Statistical analysis was conducted by using SPSS (version 17, Chicago, IL). For continuous variables, data were tested for normal distribution using the Kolmogorov–Smirnov test. The description of the data was done in the form of mean ± SD for quantitative data and frequency and proportion for qualitative data. The analysis of the data was done to test statistically significant difference between groups. For quantitative date, unpaired Student's *t*-test was used to compare between two groups. Chi square test was used for qualitative data. Receiver operating characteristics (ROC) curve analysis was performed for the evaluation of the sensitivity and specificity of serum uric acid level and PaO_2/FIO_2 in predicting ARDS mortality. Correlations were performed with Pearson's rank correlation coefficient. For all tests, statistical significance was considered when $p < 0.05$.

Results

Comparing data in high and normal uric acid groups, there was no statistically significant difference as regards age in both high ($\geqslant$6.4 mg/dl) and normal uric acid ($<$6.4 mg/dl) groups 32.6 ± 8 and 32.7 ± 9 respectively, p value of 0.97 (Table 1). Statistically significant lower APACHEII was reported in high uric acid group 27.1 ± 3.8 in comparison with normal uric