Blood Gas Analysis and Plasma D-dimer levels relationship in Chronic Obstructive Pulmonary Disease Patients

Lijun Miao[1],Ruixia Zhang[1], Zhentao Sun[2], Jing Wang[1],Zengyan Gao[1], Shifu Huang[1],Xi Wang[1]

[1]Department of Respiratory medicine, The First Affiliated Hospital of Zhengzhou University, Zhengzhou, 450052,Henan,China
[2] Department of anesthesia, The First Affiliated Hospital of Zhengzhou University,Zhengzhou,450052,Henan,China

Abstract: Objective To explore the relationship by determination of D-dimer (DD) and blood gas analysis value in chronic obstructive pulmonary disease (COPD). **Methods** D-dimer and blood gas analysis of 70 patients with COPD acute exacerbation of patients in remission were retrospectively analyzed. Results: D-dimer levels in chronic obstructive pulmonary disease exacerbation was significantly higher compared with the control group. D-dimer significantly decreased after treatment; D-dimer, and arterial oxygen partial pressure (PaO_2) was negatively correlated while arterial carbon dioxide partial pressure ($PaCO_2$) was positively correlated. **Conclusion** D-dimer can be used as a non invasive measure of outcome in patients with COPD. It can be used as thrombosis detection and secondary fibrinolysis . COPD patients with elevated D-dimer maybe used in vivo hypoxia and hypercapnia in the clinical treatment of conventional anti-inflammatory, antispasmodic, asthma and improve ventilation based on the appropriate use of anti-D-dimer levels. Anticoagulation therapy to reduce blood viscosity, conducive to slow down the progression, prevent deterioration and promote the recovery of the disease by using appropriate level of anti-D-dimer.

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Key words: chronic obstructive pulmonary disease; D-dimer; blood gas analysis; value

Recent studies found that chronic hypoxia, acidosis can increase blood coagulation status was high or prothrombotic state, can accelerate the progress of chronic obstructive pulmonary disease (chronic obstructive pulmonary disease, COPD). Hypercoagulable state as the body and fibrinolysis one of molecular markers, D-dimer (D-dimer, DD) cross-linked fibrin by plasmin degradation of a specific level of degradation products, the increase reflects the secondary fibrinolysis activity increased, so the detection the plasma DD COPD will be of great significance. Observed 70 patients with COPD, its acute exacerbation and remission of plasma D-dimer and blood gas analysis study, and 40 healthy controls were compared to investigate the D-dimer in patients with CODP the correlation of blood gas analysis.

1 Materials and Methods

1.1 General information on 70 cases of patients with COPD Respiratory Medicine admitted in May 2011 to May 2012, were characterized by enhanced chest CT diagnosis confirmed, in line with the Chinese Society of Respiratory Diseases, diagnosis and treatment of chronic obstructive pulmonary disease, "Guide "[1]. And the exclusion of heart disease, cerebrovascular disease, diabetes and blood diseases, pulmonary infarction. There were 42 males and 28 females, aged 57 to 76 years, mean (63.3 ± 7.2) years. Patients in remission 3O cases, 40 cases of acute exacerbation of. Select another control group of 40 patients were healthy. Between the two groups the difference was not statistically significant (P> 0.05), comparable.

1.2 Detection of the control group in the early morning fasting blood the COPD group admitted to hospital the next day fasting and after complete remission blood early in the morning; detection method: DD with a built-in 109mmol / L sodium citrate anticoagulant vacuum vessel extraction elbow blood 4ml (anticoagulants and blood volume ratio of 1/9) at 4 ° C, 3000 rev / min centrifugation for 15 min, draw plasma stored at -20 ° C to be tested, with Japan Sysmex CA-7000 automatic coagulation analyzer determination of the the Sysmex production of immune nephelometry DD reagents. Blood gas analysis of arterial blood was pumping 1M1 (heparin), United States NOVA phox blood gas analyzer measurement, but should stop blood and oxygen for 30 minutes.

1.3 Statistical Methods SPSS13.0 statistical software for statistical processing. Measurement data to x ± s that the groups were compared using t test, linear correlation analysis of the relationship between blood gas analysis and D-dimer. P <0.05 was considered statistically significant.

2 Results

2.1 COPD patients and healthy subjects D. D

levels in patients with acute exacerbation of COPD DD levels were significantly higher than that of COPD in remission and healthy subjects. Table 1.

Table 1 COPD patients compared with healthy subjects DD levels (x ± s)

Groups	n	D-D(mg/L) * *
COPD acute exacerbation	40	1.57±0.75*
COPDremission	30	0.47±0.22
Control group	40	0.37±0.15

Note: Compared with control group, * P <0.05; ** the D-D reference value: <0.5 mg / L.

2.2 COPD acute exacerbations of patients before and after DD PaO2 and PaCO2 comparison, after treatment after DD decreased PaO2 increased, PaCO2 decreased significantly. Illustrated in Table 2.

Table 2 COPD patients before and after treatment of acute exacerbation of DD and arterial blood gas analysis (x ± s)

Time	n	D-D(mg/L)	PaO_2(mmHg)	$PaCO_2$(mmHg)
Before Treating	40	1.57±0.75	57.2±15.9* *	66.8±16.9* *
After Treating	40	0.78±0.87*	78.2±9.1*	44.6±8.1* *

Note: before and after treatment, * P <0. O1; ** acute exacerbation of arterial PaO2 and DD levels were negatively correlated (F = -0.82, P <0.01); *** acute exacerbation of arterial PaCO2 and DD levels were positively correlated (r = 0.61 , P <0.05), 1 mmHg = 0.133 kPa.

3.Discussion

COPD is a kind of the airway, chronic inflammation of the lung parenchyma, and pulmonary vascular disease characterized by involving a variety of inflammatory cells and inflammatory mediators. The hypoxemia causes secondary polycythemia, thereby increasing the viscosity of the blood. In addition, oxidative stress, hypercapnia can damage endothelial cell function, and damage to endothelial cells but also promote blood clotting [2]. Known COPD patients blood viscosity, high coagulation state, even in the presence of thrombosis or blood clots state, and is more common in acute exacerbation of their causes with the following factors [3]: (1) long-term chronic hypoxia can cause on behalf of liquidating an increase in red blood cells, red blood cell membrane energy metabolism disorder, compliance, red blood cell aggregation ability, increased blood viscosity; (2) long-term tissue hypoxia promote white blood cells, macrophages release a variety of inflammatory mediators interferon -2 (IFN -2), interleukin -6 (IL-6), resulting in alveolar epithelial cells and vascular endothelial cell damage and stimulate platelet adhesion and aggregation; ③ patients with COPD with acute exacerbation of prevalence of antithrombin - III (AF-III) activity was significantly decreased, blood anticoagulant dysfunction; the ④ acidosis directly vascular endothelial cells, causing activation of the endogenous coagulation system. These causes COPD patients in exacerbation of the presence of blood high viscosity, high coagulation state, especially in patients with pulmonary heart disease even in the presence of a thrombus or blood clots state, and thus cause or aggravate pulmonary hypertension [4].

Fibrinolytic system is the most important human anticoagulation system. In the process of dissolution, thrombin, fibrin hydrolysis, the release of soluble fibrin monomer III A role of factor X to form a stable crosslinked fibrin. Cross-linked fibrin the plasmin degradation process, biodegradable smallest fragment D-dimer [5]. D-dimer is a small molecule generated by plasmin action to crosslinked fibrin dimer, is sensitive and specific indicators reflecting vivo plasminogen activation and fibrin formation and elevated D-dimer which means that the body following the onset increased fibrinolytic activity. Studies have shown that the body changes in patients with acute exacerbation of COPD may be associated with alveolar capillary congestion, damage to the endothelial cells, thereby activating the coagulation and fibrinolysis system, the DD increase. The findings confirm the higher DD DD level of the higher mortality rate ≥ 2.0 mg / L group mortality was significantly higher. DD ≥ 1.0 mg / L as the cut-off point to predict the death of a sensitivity of 80.6% and a specificity of 46.5%, negative predictive value of 88.5% [6]. Reported in the literature, COPD patients with heparin in the treatment of patients with DD, coagulation of fibrinogen (Fib) were significantly

decreased, accompanied by reduced arterial PaCO2 and PaO2 increased, a further indication of the coagulation system changes and blood gas changes correlated [7]. This study shows that COPD exacerbation of before treatment, after the D-dimer, arterial blood gas analysis and comparison of the differences were significant statistically significant (P <0.05), and arterial partial pressure of oxygen and D-dimer was significantly negatively related to arterial carbon dioxide partial pressure and D-dimer was significantly positively correlated, suggesting that hypoxemia, hypercapnia, and patients with elevated levels of D-dimer has a close relationship. Another study confirmed: COPD patients with intravenous infusion of heparin treatment, plasma fibrinogen, D-dimer and other coagulation parameters were significantly decreased, accompanied by arterial partial pressure of carbon dioxide reduction and increased blood oxygen partial pressure, suggesting that changes in coagulation and blood gas change is closely related to [8].

In summary, the determination of D-dimer levels to identify lung disease, has the following advantages [9]: (1) The sampling and testing simple. D-dimer to identify chronic obstructive pulmonary disease, pulmonary heart disease, the operation is very simple, and detected by enzyme-linked immunosorbent assay to detect, the operating sampling is very simple; (2) without a handling patients. COPD patients with chronic hypoxia, hypercapnia and chronic inflammation, the secondary red blood cells compensatory increase easily lead to the body in a hypercoagulable state, small arterial thrombosis, D-dimer can be used as non-traumatic patients with COPD thrombosis in vivo detection and secondary fibrinolysis OUTCOME MEASURES. Clinical treatment to improve hypoxia, ventilation and appropriate the blood anticoagulant therapy to control the disease progresses important value.

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