Effect of adjuvant noninvasive positive pressure ventilation on blood gas parameters, cardiac function and inflammatory state in patients with COPD and type II respiratory failure

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ABSTRACT

Objective: To analyze the effect of adjuvant noninvasive positive pressure ventilation on blood gas parameters, cardiac function and inflammatory state in patients with chronic obstructive pulmonary disease (COPD) and type II respiratory failure. Methods: 90 patients with COPD and type II respiratory failure were randomly divided into observation group and control group (n=45). Control group received conventional therapy, observation group received conventional therapy + adjuvant noninvasive positive pressure ventilation, and differences in blood gas parameters, cardiac function, inflammatory state, etc., were compared between two groups of patients 2 weeks after treatment. Results: Arterial blood gas parameters pH and alveolar-arterial partial pressure of oxygen [P(A-a)O₂] levels of observation group were higher than those of control group while, potassium ion (K⁺), chloride ion (Cl⁻) and carbon dioxide combining power (CO₂CP) levels were lower than those of control group 2 weeks after treatment; echocardiography parameters Doppler-derived tricuspid lateral annular systolic velocity (DTIS) and pulmonary arterial velocity (PAV) levels were lower than those of control group (P<0.05) while pulmonary artery accelerating time (PAACT), left ventricular end-diastolic dimension (LVDd) and right atrioventricular tricuspid annular plane systolic excursion (TAPSE) levels were higher than those of control group (P<0.05); serum cardiac function indexes adiponectin (APN), Copeptin, N-terminal pro-B-type natriuretic peptide (NT-proBNP), cystatin C (CysC), growth differentiation factor-15 (GDF-15) and heart type fatty acid binding protein (H-FABP) content were lower than those of control group (P<0.05); serum inflammatory factors hypersensitive C-reactive protein (hs-CRP), tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), IL-8, IL-10, and transforming growth factor-β1 (TGF-β1) content were lower than those of control group (P<0.05). Conclusions: Adjuvant noninvasive positive pressure ventilation can optimize the blood gas parameters, cardiac function and inflammatory state in patients with COPD and type II respiratory failure, and it is of positive significance in improving the overall treatment outcome.

1. Introduction

Patients with chronic obstructive pulmonary disease (COPD) may develop type II respiratory failure after repeated acute infection attack, and these patients are extremely hard to treat and with poor prognosis. Patients with COPD and type II respiratory failure have further ventilation and exchange dysfunction as well as lung ventilation/blood flow ratio imbalance, and how to early reverse the patients’ respiratory function and increase the body’s oxygen supply is the key to the treatment[1,2]. Conventional low-flow oxygen uptake via nasal catheter is suitable for the patients with stable COPD, the body’s ability to actively take in oxygen and discharge carbon dioxide is weakened dramatically when patients are combined with type II respiratory failure, and noninvasive positive pressure ventilation is considered as a more reasonable adjuvant
means for ventilation. Noninvasive positive pressure ventilation provides appropriate pressure in the entire breathing process to adjust the airway oxygen concentration and alleviate the pressure of the respiratory system operation, and it can effectively protect the pulmonary alveoli and improve respiratory failure[3,4]. In the study, adjuvant noninvasive positive pressure ventilation was used for the treatment of patients with COPD and type II respiratory failure in our hospital, and the effect of the treatment on blood gas parameters, cardiac function and inflammatory state in patients was mainly elaborated.

2. Materials and methods

2.1. General information

90 patients with COPD and type II respiratory failure treated in our hospital between January 2014 and January 2016 were included. Inclusion criteria: (1) conforming to the diagnostic criteria for COPD established by the world health organization (WHO); (2) in accordance with the diagnostic criteria for type II respiratory failure; (3) with cardiac function II to III levels; (4) signing informed consent. Exclusion criteria: (1) with pulmonary encephalopathy; (2) complicated with acute myocardial infarction; (3) with malignant tumor diseases; (4) associated with severe liver and kidney dysfunction. 90 patients conformed to the above criteria and were randomly divided into observation group and control group (n=45). Control group included 25 male cases and 20 female cases that were 51–76 years old, and with the course of COPD 8–20 years and (14.38±3.53) years in average; observation group included 24 male cases and 21 female cases that were 50–74 years old, and with the course of COPD 8–19 years and (14.67±4.21) years in average. The two groups of patients were not statistically different in general information (P>0.05).

2.2. Treatment analysis

Control group received conventional treatment, which was specifically as follows: continuous low-flow oxygen uptake, anti-infection, eliminating phlegm and relieving asthma. Based on conventional treatment, observation group received the adjuvant noninvasive positive pressure ventilation, which was specifically as follows: ventilator (Draeger Company in Germany, DRAGER Savina) mode was set to S/T, and IPAP increased from 8 cm H2O and was slowly adjusted to appropriate levels (mostly ≤20 cm H2O) within 20 min. EPAP was set to 4–8 cm H2O, respiratory frequency to 12–18 times/min, inspiratory/expiratory ratio to 1.0:1.5 and fraction of inspired oxygen to 40%. The above ventilator parameters were reasonably and prudently adjusted based on patients’ condition, patients’ tolerance was the clinical standard, and they were treated for 2 weeks in a row. The conventional treatment was the same as that of the control group.

2.3. Blood gas parameters

2 weeks after treatment, 1 mL of left radial artery blood was collected from two groups of patients, and bedside blood monitoring system (Abbott Company in the United States, i-STAT model) was used to detect blood gas values, including pH value (pH), differential alveolar-arterial partial pressure of oxygen [P(A-a)O2], potassium ion (K+), chloride ion (Cl-) and carbon dioxide combining power (CO2CP).

2.4. Cardiac function parameters

2 weeks after treatment, echocardiography was used to detect the right atrioventricular tissue Doppler-derived tricuspid lateral annular systolic velocity (DTIS), pulmonary arterial velocity (PAV), pulmonary artery accelerating time (PAACT), left ventricular end-diastolic dimension (LVDd) and right atrioventricular tricuspid annular plane systolic excursion (TAPSE) of two groups of patients. Meantime, fasting peripheral venous blood was collected and centrifuged to get serum and detect the content of cardiac function-related indicators in it, including adiponectin (APN), Copeptin, N-terminal pro-B-type natriuretic peptide (NT-proBNP), cystatin C (CysC), growth differentiation factor-15 (GDF-15) and heart type fatty acid binding protein (H-FABP).

2.5. Inflammatory factors

2 weeks after treatment, fasting peripheral venous blood was collected from two groups of patients and centrifuged to get serum, and ELISA method was used to determine inflammatory factors hypersensitive C-reactive protein (hs-CRP), tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), IL-8, IL-10 and transforming growth factor-β1 (TGF-β1) content, and part of the projects were tested by Kindstar Medical Examination Center.

2.6. Statistical analysis

Data in the study was input in SPSS23.0 software, measurement data comparison between two groups was by t test and P<0.05 indicated statistical significance in the obtained results.
3. Results

3.1. Blood gas parameters

2 weeks after treatment, comparison of radial arterial blood gas parameters pH, P(A-a)O₂, K⁺, Cl⁻ and CO₂CP levels between two groups of patients is as follows: arterial blood PH and P(A-a)O₂ levels of observation group were significantly higher than those of control group (P<0.05), while K⁺, Cl⁻ and CO₂CP levels were significantly lower than those of control group (P<0.05) (Table 1).

3.2. Echocardiography parameters

2 weeks after treatment, comparison of echocardiography parameters DTIS, PAV, PAACT, LVDd and TAPSE between two groups of patients is as follows: DTIS and PAV levels of observation group were significantly lower than those of control group (P<0.05) while PAACT, LVDd and TAPSE levels were significantly higher than those of control group (P<0.05) (Table 2).

3.3. Cardiac function indexes

2 weeks after treatment, comparison of serum cardiac function indexes APN, Copeptin, NT-proBNP, CysC, GDF-15 and H-FABP content of observation group were significantly lower than those of control group (P<0.05) (Table 3).

3.4. Inflammatory factors

2 weeks after treatment, comparison of serum inflammatory factors hs-CRP, TNF-α, IL-1β, IL-8, IL-10 and TGF-β1 content between two groups of patients is as follows: serum hs-CRP, TNF-α, IL-1β, IL-8, IL-10 and TGF-β1 content of observation group were significantly lower than those of control group (P<0.05) (Table 4).

### Table 1
Comparison of arterial blood gas parameters between two groups of patients after treatment (n=45, x̄±s).

<table>
<thead>
<tr>
<th>Groups</th>
<th>pH (x̄±s)</th>
<th>P(A-a)O₂ (mmHg)</th>
<th>K⁺ (mmol/L)</th>
<th>Cl⁻ (mmol/L)</th>
<th>CO₂CP (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation group</td>
<td>7.38±0.14</td>
<td>40.63±4.38</td>
<td>3.82±0.41</td>
<td>103.27±11.32</td>
<td>23.47±2.91</td>
</tr>
<tr>
<td>Control group</td>
<td>7.24±0.25</td>
<td>26.84±3.84</td>
<td>4.51±0.52</td>
<td>112.19±14.63</td>
<td>30.26±3.82</td>
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<td>t</td>
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### Table 2
Comparison of echocardiography parameter levels between two groups of patients after treatment (n=45, x̄±s).

<table>
<thead>
<tr>
<th>Groups</th>
<th>DTIS (cm/s)</th>
<th>PAV (m/s)</th>
<th>PAACT (ms)</th>
<th>LVDd (mm)</th>
<th>TAPSE (mm)</th>
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<tr>
<td>Observation group</td>
<td>13.28±1.71</td>
<td>0.81±0.09</td>
<td>114.38±13.29</td>
<td>44.75±4.93</td>
<td>20.74±2.83</td>
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<td>Control group</td>
<td>15.32±1.83</td>
<td>0.94±0.11</td>
<td>89.64±9.12</td>
<td>41.08±4.83</td>
<td>18.17±2.19</td>
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### Table 3
Comparison of serum cardiac function index content between two groups of patients after treatment (n=45, x̄±s).

<table>
<thead>
<tr>
<th>Groups</th>
<th>APN (ng/L)</th>
<th>Copeptin (ng/L)</th>
<th>NT-proBNP (μg/L)</th>
<th>CysC (ng/L)</th>
<th>GDF-15 (ng/L)</th>
<th>H-FABP (ng/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation group</td>
<td>11.83±1.95</td>
<td>1 321.84±150.83</td>
<td>3.18±0.34</td>
<td>1.28±0.17</td>
<td>759.36±85.12</td>
<td>57.48±6.12</td>
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<tr>
<td>Control group</td>
<td>14.27±1.85</td>
<td>1 932.71±242.84</td>
<td>4.79±0.53</td>
<td>1.71±0.19</td>
<td>1 983.62±241.55</td>
<td>96.34±10.15</td>
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### Table 4
Comparison of serum inflammatory factor content between two groups of patients after treatment (n=45, x̄±s).

<table>
<thead>
<tr>
<th>Groups</th>
<th>hs-CRP (ng/L)</th>
<th>TNF-α (μg/L)</th>
<th>IL-1β (ng/L)</th>
<th>IL-8 (ng/L)</th>
<th>IL-10 (ng/L)</th>
<th>TGF-β1 (ng/L)</th>
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<tbody>
<tr>
<td>Observation group</td>
<td>11.28±1.76</td>
<td>0.12±0.01</td>
<td>35.84±4.12</td>
<td>53.28±6.11</td>
<td>8.49±0.91</td>
<td>101.73±13.28</td>
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<tr>
<td>Control group</td>
<td>30.69±3.85</td>
<td>0.28±0.03</td>
<td>79.63±8.52</td>
<td>70.95±8.23</td>
<td>17.53±2.01</td>
<td>120.69±14.37</td>
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4. Discussion

COPD progression leads to ventilation and exchange dysfunction, and will cause type II respiratory failure characterized by hypoxia and carbon dioxide accumulation. The difficulty in treatment of patients with COPD and type II respiratory failure increases, and early targeted intervention measures were needed to improve the patients' respiratory function. Continuous low-flow oxygen uptake is the conventional way to increase oxygen supply for patients with COPD, but the upper airway resistance further increases in COPD patients combined with respiratory failure, and conventional way of oxygen uptake is difficult to achieve the role in improving red blood cell oxygenation. Noninvasive positive pressure ventilation can increase average airway pressure, increase the alveolar filling, improve ventilation/blood flow ratio in COPD patients, increase the lung compliance, and finally exert the effect on optimizing patients' respiratory function[5]. Many studies have shown that noninvasive positive pressure ventilation technology has become a reliable way to improve the illness of patients with end-stage COPD, and in the study, the ventilation way was used in patients with COPD and type II respiratory failure in our hospital and specifically studied from three aspects: blood gas parameters, cardiac function and inflammatory factors.

COPD patients are mainly characterized by decreased oxygen partial pressure, increased CO₂ partial pressure, increased blood acidity, etc., and in the case of COPD aggravation and type II respiratory failure, the above arterial blood gas changes further increase and will lead to the changes in the levels of related ions in circulating blood[6,7]. In the study, radial artery blood gas of two groups of patients were tested 2 weeks after treatment, and it was found that pH and P(A-a)O₂ levels of observation group were higher (P<0.05) while K⁺, Cl⁻ and CO₂CP levels were lower (P<0.05). It indicates that both oxygen uptake and carbon dioxide removal increase after noninvasive positive pressure ventilation intervention, and the blood meta-acid state are improved.

The airway blockage and carbon dioxide accumulation in patients with severe COPD can lead to pulmonary vasoconstriction and vessel wall reconstruction, the right cardiac load further increases after type II respiratory failure occurs, and the occurrence of right ventricular hypertrophy, in particular, will directly lead to right heart failure[8]. Related study shows that long-term heart failure rate is higher than 50% in patients with COPD and type II respiratory failure. The roles of noninvasive positive pressure ventilation technology in maintaining patients' pulmonary functional residual capacity, reducing the pulmonary shunt, etc., can dilate pulmonary vessels and reduce pulmonary artery pressure and cardiac preload, and in other words, the change of the cardiac function in COPD patients can also indirectly show the curative effect of noninvasive positive pressure ventilation[9]. In the study, ultrasonic electrocardiogram of two groups of patients were collected after treatment, differences in the levels of related parameters were compared, and it was found that right atrioventricular tissue DTIS and PAV levels of observation group were lower (P<0.05) while PAACT, LVdD and right atrioventricular TAPSE levels were higher after treatment (P<0.05)[10,11]. DTIS and PAV are directly correlated with the pulmonary artery pressure, and studies have shown that as pulmonary artery diameter decreases and pulmonary artery pressure increases, and DTIS and PAV levels rise. PAACT, LVdD, DTIS and TAPSE are associated with right heart contraction capacity, and the above results indicate that the enhanced right ventricular contractility of observation group after treatment is the direct effect of lung function improvement.

Cardiac and pulmonary functions are vitally interrelated, and with the change of lung function and overall illness in patients with COPD, there will be corresponding changes in serum cardiac function-related indicator content. APN, Copeptin, NT-proBNP, CysC, GDF-15 and H-FABP are the indexes closely related to the cardiac function. APN has the recognized anti-inflammatory and anti-atherosclerosis effect, but a new study has found that APN level increases with the increase of patients' cardiac function grade, and it is speculated to be a sign of high consumption state in patients with heart failure[12]. Copeptin is an ideal indicator to evaluate the prognosis of patients with heart failure, its trend is consistent with that of NT-proBNP, and the content of Copeptin and NT-proBNP increase as the cardiac function declines. CysC is an independent risk factor for patients with heart failure, and GDF-15 levels increase with the increase of cardiac function grade[13]. H-FABP is a new type of myocardial injury marker, it is released into the bloodstream early after myocardial injury, and its content increases with the disease aggravation. In the study, the contents of the cardiac function indexes of two groups of patients were detected after treatment, and it was found that serum APN, Copeptin, NT-proBNP, CysC, GDF-15 and H-FABP content of observation group were lower after treatment (P<0.05), indicating that the adjuvant noninvasive positive pressure ventilation can significantly improve cardiac function in patients with COPD and type II respiratory failure, and further illustrating that the ventilation greatly optimizes the overall condition of patients with COPD.

Infection is the key factor that aggravates the illness in COPD patients, and during the treatment of patients with COPD, the change of serum inflammatory factor content is directly correlated to the trend of disease. hs-CRP, TNF-α, IL-1β, IL-8, IL-10 and TGF-β1 are the inflammatory factors closely associated with the inflammatory reaction in patients with COPD infections, and they are involved in the activation and cascade amplification of the inflammatory response. hs-CRP, TNF-α, IL-1β and IL-8 are the cytokines with pro-inflammatory effect, their levels can rise in the...
early inflammatory response, and with the aggravation of infection, their secretion further increases[14,15]. IL-10 and TGF-β1 are the cytokines with anti-inflammatory effect, they are with compensatory rise during the activation of inflammatory reaction, and they can inhibit excessive inflammation damage to the tissue[16,17]. In the study, analysis of the content of these inflammatory cytokines showed that serum hs-CRP, TNF-α, IL-1β, IL-8, IL-10 and TGF-β1 content of observation group were significantly lower than those of control group ($P<0.05$). This means that the adjuvant noninvasive positive pressure ventilation can reduce the systemic inflammatory state in patients with COPD and type II respiratory failure, and it helps to improve the overall condition. To sum up, it is concluded as follows: adjuvant noninvasive positive pressure ventilation can optimize the blood gas parameters, cardiac function and inflammatory state in patients with COPD and type II respiratory failure, and it is of positive significance in improving the overall treatment outcome.

References


