Effect of heparin in combined with magnesium sulfate on the blood gas and blood viscosity in patients with pulmonary heart failure

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1. Introduction

Chronic pulmonary heart disease (CPHD), i.e. pulmonary heart disease, is a commonly severe and acute respiratory disease in China, most of which are originated from COPD, with an increasing morbidity rate. Respiratory failure is a common complication of pulmonary heart disease at the acute and severe stage. The hospital death rate of pulmonary heart disease in China can reach 13.3%-44.1%[1]. Currently, routine treatments of anti-inflammation, spasmylosis, asthma relieving, cough suppressing, and phlegm dispersing are involved in the treatment of CPHD; moreover, anticoagulation drug, such as heparin is adopted to reduce the blood viscosity, improve the myocardial metabolism, and strengthen the cardio-pulmonary function[2]. The study is aimed to observe the effect of heparin in combined with magnesium sulfate on the blood gas indicators and blood viscosity in patients with pulmonary heart disease merged with respiratory failure.

2. Materials and methods

2.1. General materials

A total of 80 patients with CPHD who were admitted in our hospital from January, 2012 to December, 2015 were included in the
study. All the patients were in accordance with the diagnostic criteria of CPHD in 1977[3], accompanied by heart failure, with NYHA of I - IV. The patients were randomized into the observation group \((n=40)\) and the control group \((n=40)\). In the observation group, 28 were male, and 12 were female; aged from 56 to 73 years old, with an average age of \(62.32±5.19\) years old; 9 at grade II, 18 at grade III, and 13 at grade IV. In the control group, 27 were male, and 13 were female; aged from 58 to 70 years old, with an average age of \(63.14±5.23\) years old; 10 at grade II, 17 at grade III, and 13 at grade IV. The comparison of gender, age, and cardiac function grading between the two groups was not statistically significant, and it was comparable \((P>0.05)\). Informed consents were obtained from the patients. Exclusion criteria: (1) those who had taken anti-coagulation or anti-thrombus drugs recently; (2) those who had trauma history or operation history recently; (3) those who had severe pulmonary, liver, and renal dysfunction; (4) those who were merged with diabetes and hypertension; (5) those who had active digestive ulcer.

2.2. Methods

The patients in the control group were given routine symptomatic treatments, i.e. continuous low-flow oxygen intaking, antibiotics to prevent the pulmonary infection, cough and asthma relieving, respiratory stimulant, phlegm dispersing, acid-base and electrolyte balance regulating, and nutrition support.

On the above basis, the patients in the observation group were given heparin (50 mg) + 25% magnesium sulfate (10 mL) + 5% glucose injection (250 mL), ivdrip, 30-40 droplets/min, 1 time/d, continuously for 14 d. The clinical symptoms and signs were closely observed during the treatment period.

2.3. Observation indicators and efficacy criteria

The blood gas indicators, hemorheology indicators, PAPs, SBP, DBP, and clinical efficacy in the two groups were detected and compared. The coagulation function, liver and renal function, blood routine examination, urine routine examination, and blood sugar were detected.

The blood gas analyzer was used to detect the blood gas indicators, including \(\text{PaO}_2\), \(\text{PaCO}_2\), and pH. WBV, Hct, BRV, EAI, PV, and Fg were involved in the detection of hemorheology. Sonoace 8000 color Doppler ultrasound was used to detect \(V_{\text{max}}\). Bernoulli[4] was used to calculate PAPs. \(\text{PAPs} (\text{mmHg}) = 4V_{\text{max}}^2 + 10 \text{ mmHg}\).

2.4. Statistical analysis

SPSS 19.0 software was used for the statistical analysis. The measurement data were expressed as mean ± SD. The paired \(t\) test was used for the intra-group comparison, and the independent \(t\) test was used for the comparison between the two groups. The enumeration data were expressed as percentage, and chi-square test was used. \(P<0.05\) was regarded as statistically significant.

3. Results

3.1. Comparison of the blood gas indicators before and after treatment between the two groups

The comparison of blood gas indicators before treatment between the two groups was not statistically significant \((P>0.05)\). After 2-week treatment, \(\text{pH}\) value was not significantly changed, \(\text{PaO}_2\) in the observation group was significantly higher than that in the control group, while \(\text{PaCO}_2\) was significantly lower than that in the control group \((P<0.01)\) (Table 1).

3.2. Comparison of the hemorheology indicators before and after treatment between the two groups

The comparison of various indicators before treatment between the two groups was not statistically significant \((P>0.05)\). After treatment, the various indicators in the two groups were reduced to a different degree, and the reduced degree in the observation group was significantly superior to that in the control group \((P<0.01)\). Except for the whole blood reduced viscosity, the comparison of the rest five indicators was statistically significant \((P<0.01)\).

3.3. Comparison of PAPs and blood pressure before and after treatment between the two groups

The comparison of PAPs, SBP, and DBP before treatment between the two groups was not statistically significant \((P>0.05)\). PAPs after treatment in the observation group was significantly reduced when compared with the control group \((P<0.05\) or \(P<0.01)\), and the reduced degree in the observation group was significantly superior to that in the control group \((P<0.05)\). SBP and DBP after treatment in the two groups were not significantly changed \((P>0.05)\) (Table 3).

**Table 1.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>(n)</th>
<th>Time</th>
<th>(\text{PaO}_2) (kPa)</th>
<th>(\text{PaCO}_2) (kPa)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>40</td>
<td>Before treatment</td>
<td>7.33±1.28</td>
<td>8.31±1.48</td>
<td>7.23±0.18</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After treatment</td>
<td>9.43±1.34**</td>
<td>6.23±1.52**</td>
<td>7.31±0.21</td>
</tr>
<tr>
<td>Control</td>
<td>40</td>
<td>Before treatment</td>
<td>7.32±1.29</td>
<td>8.34±1.46</td>
<td>7.27±0.22</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After treatment</td>
<td>8.06±1.31*</td>
<td>7.54±1.50*</td>
<td>7.33±0.20</td>
</tr>
</tbody>
</table>

*\(P<0.05\); **\(P<0.01\), when compared with before treatment; ***\(P<0.01\), when compared with the control group.
4. Discussion

Due to the long-term chronic hypoxia and carbon dioxide retention in patients with pulmonary heart disease merged with respiratory failure, the symptoms of heart failure and respiratory failure can be aggravated[5], with a poor therapeutic effect in the clinic. Due to infection and severe hypoxia at an acute attack, a large amount of histamine is released, the platelet gathering and releasing function is strengthened, the blood is in a state of hypercoagulation, hyperviscosity, and high aggregation, thus the microcirculatory thrombus is formed[8]. It is reported by He et al[7] that the whole blood viscosity and relative whole blood viscosity in patients with pulmonary heart disease at an acute phase were significantly higher than those in the control group and at a remission stage, while those indicators at a remission stage were significantly higher than those in the control group. Some other researches demonstrate that[8] autopsy on the dead patients at an acute stage of pulmonary heart disease shows that the occurrence rate of pulmonary arteriole thrombosis is 89.8%, all of which is in situ thrombosis but not thromboembolism; therefore, reducing the blood viscosity and anti-thrombosis are of great significance in the treatment of pulmonary heart disease merged with respiratory failure.

Heparin is a kind of anticoagulant, is a polymer connected by two polysaccharides, has an anticoagulation effect, and currently exerts a preferable efficacy in the treatment of severe pulmonary heart disease; therefore, it is positively accepted. Heparin can prevent the platelet aggregation through preventing the blood coagulation factors in order to reduce the blood viscosity and inhibit its continuous elevation, thus, playing an anticoagulation effect; and meanwhile can also improve the various indicators of hemorheology[9,10]. With the development of pharmacology and clinical medicine, heparin also has effects of diuresis, anti-allergy, expanding the bronchial smooth muscle, and anti-inflammation. It is reported that[11-14] heparin in a small dosage can resist the platelet aggregation and prevent the pulmonary artery thrombosis, with a significant efficacy in the treatment of pulmonary heart disease at an acute stage. Magnesium sulfate can improve the microcirculation and alleviate the pulmonary congestion to enhance the respiratory function, and also increase the myocardial contractility to improve the myocardial metabolism. Magnesium ion can activate the adenylate cyclase to improve the permeability of cell membrane, prevent the release of histamine, and relieve the bronchial spasm[15,16].

The results in the study showed that the blood gas indicators in the observation group were improved, the oxygen partial pressure was significantly enhanced, the carbon dioxide discharge was increased, and the hemorheology indicators before treatment in the two groups were significantly close to or exceeding the normal values, indicating that the blood hypercoagulation and hyperviscosity are prevailing in patients with pulmonary heart disease at an acute stage. The various indicators after treatment in the two groups were improved to a different degree, among which the comparison of WBV, PV, Hct, EAI, and Fg between the two groups was significantly different, indicating that the improved degree of blood hypercoagulation and hyperviscosity in the observation group was significantly superior to that in the control group. After treatment, PAPs was significantly reduced, DBP and SBP were not significantly changed, indicating that the treatment protocol can reduce the pulmonary artery pressure and has no effect on the systemic blood pressure in patients with pulmonary heart disease merged with respiratory pressure. The symptoms of short of breath, limb edema, cough, and cyanosis are involved in the patients in the study, with a poor hospitalization therapeutic effect. The treatment total effective rate after treatment in the observation group was significantly higher than that in the control group, and the clinical symptoms and signs were significantly improved, indicating that this treatment protocol can improve the blood circulation, alter the pulmonary blood volume, reduce the pulmonary hypertension, and improve the myocardial metabolism.

In conclusion, heparin in combined with magnesium sulfate in the treatment of pulmonary heart disease merged with respiratory failure can improve the blood gas indicators, reduce the blood

Table 2.
Comparison of the hemorheology indicators before and after treatment between the two groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>Before treatment</th>
<th>After treatment</th>
<th>P</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>40</td>
<td>WBV (mpa/s)</td>
<td>PV (mpa/s)</td>
<td>BRV (mpa/s)</td>
<td>Hct (%)</td>
</tr>
<tr>
<td>Before</td>
<td>6.55±0.59</td>
<td>1.62±0.55</td>
<td>7.45±2.99</td>
<td>49.56±4.43</td>
<td>1.93±0.33</td>
</tr>
<tr>
<td>After</td>
<td>4.15±0.50</td>
<td>1.15±0.43</td>
<td>6.21±3.12</td>
<td>41.05±3.76</td>
<td>1.32±0.22</td>
</tr>
<tr>
<td>Control</td>
<td>40</td>
<td>Before</td>
<td>6.87±0.54</td>
<td>1.60±0.57</td>
<td>7.37±3.14</td>
</tr>
<tr>
<td>After</td>
<td>6.17±0.61</td>
<td>1.54±0.51</td>
<td>6.23±3.21</td>
<td>45.66±3.38</td>
<td>1.69±0.23</td>
</tr>
</tbody>
</table>

*P<0.05, **P<0.01, when compared with before treatment; ### P<0.05, #### P<0.01, when compared with the control group.

Table 3.
Comparison of PAPs and blood pressure before and after treatment between the two groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>Time</th>
<th>PAPs</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>40</td>
<td>Before treatment</td>
<td>40.56±6.21</td>
<td>135.42±20.89</td>
<td>79.66±7.09</td>
</tr>
<tr>
<td>After</td>
<td>33.24±5.42</td>
<td>133.27±16.63</td>
<td>78.32±6.54</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>40</td>
<td>Before treatment</td>
<td>40.28±7.05</td>
<td>136.57±18.26</td>
<td>79.11±7.36</td>
</tr>
<tr>
<td>After</td>
<td>36.36±6.48</td>
<td>134.75±14.21</td>
<td>76.29±6.42</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*P<0.05, **P<0.01, when compared with before treatment; # P<0.05, ## P<0.01, when compared with the control group.
viscosity, decrease the pulmonary arterial systolic pressure, alleviate the cardiac load, and improve the heart failure, with a safety and no obvious side effects; therefore, it deserves to be widely recommended in the clinic.

References


