Effects of budesonide on serum inflammatory factors and immune function in patients with bronchial asthma

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ABSTRACT

Objective: To explore the effects of budesonide on serum inflammatory factors and immune function in patients with bronchial asthma. Methods: A total of 98 cases of bronchial asthma patients were randomly divided into the observation group and the control group according to the order of treatment, each of 49 cases. Patients in control group were treated with conventional treatment. On the basis of this, patients in the observation group were treated with budesonide inhalation. The clinical symptoms and signs, serum level of inflammatory factors and immune function of two groups were observed before and after treatment. Results: After treatment, the serum IL-6, IL-8, IL-17, TNF-alpha levels of observation group were significantly lower than the control group, with significantly difference (P<0.05). After treatment, IgA, IgG, IgM, CD4+, CD8+, and CD4+/CD8+ levels were significantly higher than the control group, with significantly difference (P<0.05). Conclusion: The effect of budesonide on bronchial asthma is good, can effectively improve the symptoms and signs, reduce serum inflammatory factor levels, enhance immune function in patients. It is worthy of promotion and application.

1. Introduction

Bronchial asthma, a chronic respiratory tract inflammatory disease, is very common in clinic. Clinical symptoms include recurrent wheezing, chest tightness, shortness of breath, dyspnea, cough or expectoration. If not being treated timely and effectively, continuous asthma may lead to respiratory failure or even endanger patients’ life[1,2]. Glucocorticoid inhalation has gradually become the most commonly used method in clinical treatment of bronchial asthma, but its effects on immune function and cytokines are rarely reported[3]. We aim to investigate effects of budesonide inhalation on patients’ serum inflammatory factor and immune function.

2. Materials and methods

2.1. General information

A total of 98 patients with bronchial asthma treated in our hospital from Nov. 2012 to Nov. 2013 were enrolled. Bronchial asthma was diagnosed according to the bronchial asthma prevention and treatment guide[4,5]. There were 52 male and 46 female 46 cases. Patients’ age ranged from 21 to 68 years old, with a mean age of (42.37±6.25). Disease course ranged from 1 to 12 years with an average course of (4.26±0.78) years. Patients with heart, liver, kidney or other important organs dysfunction, or accompanied by acute and chronic infectious diseases, or other lung diseases, endocrine system or autoimmune diseases were excluded. Those who took corticosteroids, bronchodilators or immuno-suppressive drugs during the recent 1 month, lactating women or pregnancy were also excluded. Patients had allergic reactions to budesonide and other contraindications were excluded too. Our study conforms to the requirements of Hospital Ethical Committee, all
patients were informed of the research and signed the protocol of treatment. Patients were divided into two groups according to the order of treatment. There was no significant difference in gender composition, age, severity of disease and other clinical conditions ($P>0.05$) between the two groups.

2.2. Treatments

After admitted to hospital, all the patients were given oxygen, relieving cough and asthma, expectorant (ambroxol hydrochloride), anti infection, spasmyloyis, maintaining acid-base and water electrolyte balance and other conventional treatment. Patients with bacterial infection were given moderate antibiotics treatment. The treatment of the disease should reduce the inflammatory cells and the immune and other aspects participated the pathogenesis of brochial asthma. A variety of cytokines and immune factors plays important roles in the pathogenesis and development of bronchial asthma.

2.3. Observation indexes

Venous blood were collected in the morning when fasting before and after treatment, and were centrifuged to get serum which were reserved under -80 °C. Serum inflammatory factors including interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-17 (IL-17) and tumor necrosis factor alpha (TNF-α) levels were detected by the enzyme linked immunosorbent assay (ELISA); serum immunoglobulin IgA, IgG, IgM levels were detected by Immune Turbidimetry; The expression of T lymphocyte subsets CD4$^+$, CD8$^+$ and CD4$^+$/CD8$^+$ measured by Flow Cytometry. Patients’ clinical symptoms such as coughing, shortness of breath, breath hold and pulmonary wheeze were observed in the two groups before and after treatment.

2.4. Statistical analysis

Dates were analyzed by statistical software SPSS15.0, measurement data between two groups and the date before and after treatment. Dates were analyzed by statistical software SPSS15.0, measurement data between two groups and the date before and after treatment. Patients were divided into two groups according to the composition, age, severity of disease and other clinical conditions.

### 3. Results

#### 3.1. Comparison of serum inflammatory factor levels before and after treatment

Before treatment, the serum IL-6, IL-8, IL-17 and TNF-α level showed no significant difference between two groups ($P>0.05$). After the treatment, the above indexes levels were significantly lower than the same group before treatment ($P<0.05$), and those in the observation group were significantly decreased than in the control group after treatment ($P<0.05$) (Table 1).

#### 3.2. The level of immunoglobulin and T lymphocyte testing results of the two groups before and after treatment

Before treatment, The level of immunoglobulin IgA, IgG, IgM and T lymphocyte CD4$^+$, CD8$^+$ and CD4$^+$/CD8$^+$ were not significant different between two groups ($P>0.05$). The level of those indexes showed no significant difference between the control group before and after treatment ($P>0.05$).After treatment, The IgA, IgG, IgM, CD4$^+$, CD8$^+$, and CD4$^+$/CD8$^+$ levels of the observation group were significantly higher than that before treatment, they were also significantly higher than the control group after treatment, ($P$ both<0.05) (Table 2).

### 4. Discussion

Bronchial asthma is a chronic airway inflammatory disease, and its etiology and pathogenesis of bronchial asthma is complex and still not completely clear in clinical research at present. Generally, researchers agree that multiple environmental, genetic and/or immune and other aspects participated the pathogenesis of bronchial asthma. A variety of cytokines and immune factors plays important roles in the pathogenesis and development of bronchial asthma. Therefore, modern clinical research thinks, the key lies in the treatment of the disease should reduce the inflammatory cells and the

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Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Time</th>
<th>IL-6 (μg/L)</th>
<th>IL-8 (μg/L)</th>
<th>IL-17 (μg/L)</th>
<th>TNF-α (ng/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td>Before treatment</td>
<td>125.89±12.47</td>
<td>0.68±0.12</td>
<td>21.73±3.65</td>
<td>164.96±37.28</td>
</tr>
<tr>
<td></td>
<td>after treatment</td>
<td>102.3±10.62$^b$</td>
<td>0.54±0.10$^b$</td>
<td>18.41±2.82$^b$</td>
<td>121.52±32.49$^b$</td>
</tr>
<tr>
<td>Observation group</td>
<td>Before treatment</td>
<td>126.15±12.76</td>
<td>0.71±0.13</td>
<td>21.69±3.76</td>
<td>165.18±38.62</td>
</tr>
<tr>
<td></td>
<td>after treatment</td>
<td>84.21±9.38$^b$</td>
<td>0.22±0.06$^b$</td>
<td>14.23±1.94$^b$</td>
<td>87.26±27.14$^b$</td>
</tr>
</tbody>
</table>

$^aP<0.05$: compared with the control group; $^bP<0.05$: compared with before treatment.

Table 2

<table>
<thead>
<tr>
<th>Group</th>
<th>Time</th>
<th>IgA (g/L)</th>
<th>IgM (g/L)</th>
<th>IgG (g/L)</th>
<th>CD4$^+$ (%)</th>
<th>CD8$^+$ (%)</th>
<th>CD4$^+$/CD8$^+$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td>Before treatment</td>
<td>4.36±1.22</td>
<td>3.48±1.56</td>
<td>8.72±2.34</td>
<td>38.21±4.61</td>
<td>25.16±3.83</td>
<td>1.28±0.27</td>
</tr>
<tr>
<td></td>
<td>after treatment</td>
<td>4.39±1.36</td>
<td>3.52±1.24</td>
<td>9.14±2.62</td>
<td>39.36±5.43</td>
<td>26.42±3.65</td>
<td>1.32±0.46</td>
</tr>
<tr>
<td>Observation group</td>
<td>Before treatment</td>
<td>4.35±1.24</td>
<td>3.46±1.59</td>
<td>8.69±2.21</td>
<td>37.89±4.38</td>
<td>26.87±3.76</td>
<td>1.26±0.28</td>
</tr>
<tr>
<td></td>
<td>after treatment</td>
<td>4.98±1.57$^ab$</td>
<td>4.37±1.38$^a$</td>
<td>11.58±2.76$^a$</td>
<td>44.15±6.72$^ab$</td>
<td>32.54±3.43$^a$</td>
<td>1.75±0.54$^ab$</td>
</tr>
</tbody>
</table>

$^aP<0.05$: Compared with the control group, $^bP<0.05$: compared with before treatment.
release of inflammatory factors, suppression of airway inflammation, and improve the immune function of body [6-10].

Glucocorticoids are the most effective drugs for the treatment of bronchial asthma currently in clinical medicine, it can direct role in airway epithelial cells, relieve airway inflammatory reaction by inhibiting the inflammatory cell exudation and epithelial cell proliferation [11,12], its therapeutic effect has been clinical certain. Budesonide, a commonly used inhaled glucocorticoid, inhibit the respiratory tract immune reaction through multiple links to play an anti-inflammatory effect. In addition, it inhibit the synthesis and release of histamine to reduce its biological activity, it also inhibit the synthesis and release of bronchial constriction substances to reduce airway resistance and secretion, so as to improve ventilation function [13,14]. The results of our study show that, after treatment with budesonide inhalation, the efficiency of observation group was significantly higher than control group received only conventional treatment, serum inflammatory factors IL-6, IL-8, IL-17, TNF-α level obviously improved, this indicated that budesonide can reduce the secretion of airway mucus, relieve bronchial spasm and improve clinical symptoms. Budesonide specificity inhibit eosinophils proliferation and differentiation, after inhalation, it forms high concentration and quick acting on target organs, restrain the generation, activation and migration of inflammatory cells, reduce the release of cell factors such as IL-6, IL-8, IL-17, TNF-α and inflammatory mediators like cell adhesion factor, so as to play a local anti-inflammatory effect [15,16]. Local inhalation drug directly on the bronchial to reduce airway responsiveness, improve the clinical symptoms and ventilatory function and repair airway epithelium inflammatory injury. After treatment, patients’ immune function indexes inducing IgA, IgG, IgM, CD4+, CD8+, and CD4+/CD8+ levels were significantly improved than control group, this indicated that budesonide can inhibit the occurrence of immune response through multiple links, improved the imbalance ratio of T lymphocyte subsets and regulate patients immune function. The local inhalation drug directly on the bronchial and reduce airway responsiveness, fast play the clinical efficacy, improve the clinical symptoms and ventilatory function in patients, and make the inflammation of airway epithelium injury to repair; in addition, index of immune function in patients with IgA, IgG, IgM, CD4+, CD8+, and CD4+/CD8+ levels were significantly improved than control group, that of budesonide can inhibit the immune response through several links the occurrence, improved the ratio of T lymphocyte subsets imbalance, regulate the immune function.

In summary, budesonide can effectively improve the symptoms and signs, suppress airway inflammation, improve the immune function in patients, it has exact effect in treating bronchial asthma and worthy the clinical promotion and application.

References

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