Hyperbaric oxygen combined with drug therapy in the treatment of acute cerebral infarction clinical analysis

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ARTICLE INFO

Article history:
Received
Received in revised form
Accepted
Available online

Keywords:
Hyperbaric oxygen
Drugs
Acute cerebral infarction
Clinical effects

ABSTRACT

Objective: To explore the effects of hyperbaric oxygen combined with edaravone, salviae miltiorrhizae and ligustrazine and sodium ozagrel in the treatment of acute cerebral infarction clinical analysis. Methods: A total of 200 cases of acute cerebral infarction patients were randomly divided into observation group and control group. The control group was treated with edaravone, salvia miltiorrhizae and ligustrazine and sodium ozagrel; on the basis of treatment in control group, the observation group was combined with hyperbaric oxygen therapy. The neurological deficit scores were observed before and after treatment in patients of two groups, meanwhile the activities of daily living (ADL) and clinical effects were compared. Results: The total effective rate in observation group (92%) was significantly higher than control group (79%), the differences were statistically significant; the score of ADL in observation group after treatment was obviously higher than control group [(79.91±5.16) vs (61.62±5.60)], and the differences were statistically significant. The neurological deficit scores after treatment were obviously lower than the control group [(9.55±4.13) vs (15.46±4.92)], the differences were statistically significant. Conclusion: Hyperbaric oxygen combined with edaravone, salvia miltiorrhizae and ligustrazine and sodium ozagrel in the treatment of acute cerebral infarction can improve the symptoms of microcirculation and neurologic impairment, and improve the patient’s quality of life.

1. Introduction

Acute cerebral infarction is a kind of common disease with high morbidity, high disability rate and high mortality in clinical practice. Some studies reported that its death rate was about 10% and the disability rate was over 50\%\(^1\). A series of severe complications, such as hemiplegia, aphasia and other clinical symptoms may occur if the patients are not treated in time. The latest clinical findings showed that hyperbaric oxygen in the treatment of acute cerebral infarction patients can increase the oxygen content and enhance the power of scavenging free radical in brain tissue\(^2\). This study aimed to combine hyperbaric oxygen with edaravone, salvia miltiorrhizae and ligustrazine and sodium ozagrel in the treatment of acute cerebral infarction, which has been made more satisfactory clinical results.

2. Objects and methods

2.1 Objects

A total of 200 cases of acute cerebral infarction patients were selected in the Affiliated Hospital of Hainan Medical College from January 2013 and October 2014, among which 116 were males and 84 were females, with age range from 39-86 years and mean age (56.2±4.9) years. All patients met the diagnostic criteria of the Fourth National Cerebrovascular Disease Academic Meeting in 1995 and were diagnosed as cerebral infarction through CT or magnetic resonance imaging examinations in clinic. Patients were randomly divided into observation group and control group with 100 patients in each group. The patients of two groups had no statistical differences in gender, sex, past medical history, neurologic
impairment, ADL, time of occurrence, time of onset to draw first blood and other aspects ($P<0.05$) and were comparable.

### 2.2 Treatment methods

All patients were given comprehensive therapy of conventional dehydration, diuresis, depressurization and brain cell protection.

#### 2.2.1 Control group

Control group was treated with edaravone, salvia miltiorrhizae and ligustrazine and sodium ozagrel. Edaravone 30 mg/d, salvia miltiorrhizae and ligustrazine 10 mL/d, and sodium ozagrel 120 mg/d, i.e., 10 d for a course of treatment, totally 3 courses.

#### 2.2.2 Observation group

Base on the treatment in control group, the observation group was combined with hyperbaric oxygen therapy. Hyperbaric oxygen therapy includes 3 procedures: 1) high-pressure oxygen cabin for single was used, pressure value = 0.2 MPa; 2) firstly, boost pressure for 20 min, then steady pressure for 60 min, 10 min for interval, and finally reduce pressure for 20 min; 3) one time per d, 10 d for a course of treatment, totally 30 d for 3 courses.

### 2.3 Observational index

Patients of two groups were observed with neurologic impairment, ADL and progress on treatment effect before and after treatment.

### 2.4 Criteria of curative effects

Clinical nerve deficiency scale standard and criterion of clinical treatment effect approved by the Fourth National Cerebrovascular Disease Academic Meeting in 1995 were used to evaluate efficiency. Basically cured: signs and symptoms almost returned to normal, clinical nerve deficiency scale (NDS) reduces 91%-100%; significant progress: signs and symptoms basically recover over 60%, NDS reduces 46%-90%; progress: signs and symptoms basically recover over 20%, NDS reduces 18%-45%; No changes or deterioration: signs and symptoms basically recover below 20% or even lower, NDS reduces <17%.

### 2.5 Statistical method

Data in this study were analyzed by software SPSS version 10.0. Measurement data were expressed by $(\text{Mean} \pm \text{SD})$ and tested by $t$-test. Enumeration data were tested by $\chi^2$. $P<0.05$ was considered as statistical significance.

### 3. Results

#### 3.1. Comparison of ADL score in two groups of patients after treatment

Comparison of ADL score in two groups of patients before treatment were considered no statistical significance ($P<0.05$). After 3 courses of treatment, ADL score of each course in observation group were increased obviously compared with control group, which had statistical difference ($P<0.05$), (Table 1).

#### 3.2. Comparison of NDS after treatment in two groups (Mean±SD)

#### 3.3. Comparison of Two groups of clinical curative effect \[n\%\]

### Table 1.
Comparison of ADL score by barthel index.

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>Before treatment</th>
<th>The first course of treatment (10 d)</th>
<th>The second course of treatment (20 d)</th>
<th>The third course of treatment (30 d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>100</td>
<td>38.05±5.54</td>
<td>58.46±5.27</td>
<td>65.16±5.63</td>
<td>79.91±5.16</td>
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<tr>
<td>Control</td>
<td>100</td>
<td>37.64±5.68</td>
<td>47.33±5.60</td>
<td>55.52±5.67</td>
<td>61.62±5.60</td>
</tr>
<tr>
<td>$P$ value</td>
<td></td>
<td>1.35</td>
<td>0.06</td>
<td>0.06</td>
<td>0.04</td>
</tr>
<tr>
<td>$T$ value</td>
<td></td>
<td>0.78</td>
<td>0.06</td>
<td>0.06</td>
<td>0.04</td>
</tr>
</tbody>
</table>

#### Table 2.
Comparison of NDS after treatment in two groups (Mean±SD).

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>Before treatment</th>
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<th>The second course of treatment (20 d)</th>
<th>The third course of treatment (30 d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>100</td>
<td>27.13±5.12</td>
<td>16.08±4.22</td>
<td>12.16±4.63</td>
<td>9.55±4.13</td>
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<tr>
<td>Control</td>
<td>100</td>
<td>26.06±4.87</td>
<td>20.32±4.67</td>
<td>17.09±5.55</td>
<td>15.46±4.92</td>
</tr>
<tr>
<td>$P$ value</td>
<td></td>
<td>1.11</td>
<td>3.16</td>
<td>3.41</td>
<td>3.72</td>
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<tr>
<td>$T$ value</td>
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<td>1.08</td>
<td>0.07</td>
<td>0.05</td>
<td>0.04</td>
</tr>
</tbody>
</table>

#### Table 3.
Comparison of Two groups of clinical curative effect \[n\%\].

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>The first course of treatment (10 d)</th>
<th>The second course of treatment (20 d)</th>
<th>The third course of treatment (30 d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>100</td>
<td>53 (53)</td>
<td>77 (77)</td>
<td>92 (92)</td>
</tr>
<tr>
<td>Control</td>
<td>100</td>
<td>46 (46)</td>
<td>64 (64)</td>
<td>79 (79)</td>
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<tr>
<td>$\chi^2$</td>
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<td>1.78</td>
<td>5.67</td>
<td>7.93</td>
</tr>
<tr>
<td>$P$</td>
<td></td>
<td>0.23</td>
<td>0.05</td>
<td>0.03</td>
</tr>
</tbody>
</table>
3.2 Comparison of NDS after treatment in patients of two groups

After 3 courses of treatment, ADL score of each course in observation group were reduced obviously compared with control group, which had statistical significance ($P<0.05$) (Table 2).

3.3 Therapeutic effect

In observation group, effective rate of the first course, second course and third course of treatment was 53%, 77% and 92%, respectively. In control group, effective rate of the first course, second course and third course of treatment was 46%, 64% and 79% respectively. Observation group was better than control group, which had statistical significance (Table 3).

4. Discussions

Cerebral infarction refers to a series of changes of pathophysiology, such as tissue ischaemia, brain cells lack of oxygen, edema and necrosis. Common occurrence on the basis of hypertensive arteriosclerosis, high blood viscosity, high blood fat and rising of fibrous proteins, thus result in thrombosis. Lots of clinical studies have found that the key for the treatment of acute cerebral infarction is to ease the cerebral ischemia penumbra caused by acute cerebral infarction as well as protect cerebral nerve function. Edaravone is a kind of free radical scavenger and neuroprotective agents that mainly used for neurological improvement. ADL and functional disorder caused by acute cerebral infarction, besides, its permeation rate of blood brain barrier is up to 60%. Salviae miltiorrhizae and ligustriazine possesses the functions of anti-platelet aggregation, dilating coronary artery, reducing blood viscosity, increasing red blood cell flow and improving microcirculation; Sodium ozagrel has the functions of inhibiting platelet aggregation cerebrovascular contracture and expand blood vessel as well as improving the dyskinesia in the acute phase of cerebral infarction. Besides, it can also improve the circulatory disturbance and energy metabolism abnormality in the acute phase of cerebral ischemia[5,6]. Although the application of edaravone, salvia miltiorrhizae and ligustriazine and sodium ozagrel have certain curative effect in the treatment of acute cerebral infarction in clinical practice, the symptoms and signs recovery is slow with long term treatment, which can be shown in the data of control group.

The functions of hyperbaric oxygen in the treatment of acute cerebral infarction are to increase the dispersion ability of capillary oxygen in tissue, enhance the partial pressure and content of blood oxygen, ameliorate oxygen supply of lesion area and improve metabolism and microcirculation. Meanwhile it can also increase the blood flow volume of vertebrobasilar artery and ischemic region as well as enhance the ability of red blood cells through the narrow capillaries. The viscosity of blood and platelet aggregation can be reduced. Furthermore, it can also accelerate the dissolution of blood clots. Thus it has the advantages such as the blood supply of cerebral thrombosis lesions and the recovery of blood circulation in hypoxic brain regions[7,8].

The results from this study showed that after 3 courses (30 d) of the treatment of acute cerebral infarction by hyperbaric oxygen combined with edaravone, salvia miltiorrhizae and ligustriazine and sodium ozagrel, the total effective rate in observation group (92%) was obviously higher than control group (79%), and the therapeutic effect is obvious; the improvement of neurologic impairment and ADL in observation group was more obvious than control group, which had significant differences ($P<0.05$). Hyperbaric oxygen combined with edaravone, salvia miltiorrhizae and ligustriazine and sodium ozagrel in the treatment of acute cerebral infarction can reduce the blood viscosity of patients as well as lower hemodynamic index and improve cerebral blood circulation. The purpose of cerebral infarction treatment can be reached by reducing blood viscosity, relaxing blood vessels, declining arterial pressure, increasing blood oxygen concentration in cerebral infarction area, saving brain tissue in ischemic penumbra, and promoting the recovery of neurological function[9].

To sum up, hyperbaric oxygen combined with edaravone, salvia miltiorrhizae and ligustriazine and sodium ozagrel in the treatment of acute cerebral infarction can adjust blood viscosity of cerebral infarct patients effectively, reduce the levels of blood lipid and fibrinogen, and improve microcirculation and nerve function defect symptoms. Thus the patient’s survival, quality of life and activity of daily living can be increased.

References


