Oxiracetam effects on tumor necrosis factor-α of patients with severe craniocerebral injury

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Objective: To study the oxiracetam effects on patients with severe brain injury of tumor necrosis factor-α. Method: 92 cases patients of severe traumatic brain injury treated in our hospital were randomly divided into control group and observation group, 46 cases in each. The control group: accept conventional treatment of severe traumatic brain injury. The observation group: accept the conventional treatment of severe head injury while applying oxiracetam. Results: The total efficiency of the observation group 95.65% more than 80.44% that of control group, the difference was statistically significant (P<0.05). After treatment, ICP levels of patients in the observation group were higher than that of control group. The difference was statistically significant (P<0.01). After treatment, GCS, GOS score and Barthel Index of patients in the observation group was higher than that of control group, the difference was statistically significant (P<0.01). After treatment, Inflammatory markers IL1β, TNF-α levels of patients in the observation group was lower than that of control group, the difference was statistically significant (P<0.01). Conclusion: Oxiracetam treated severe brain injury have a significant curative effect, can effectively reduce the patient's intracranial pressure, relieve the patient's inflammatory response, promote the rehabilitation of the prognosis of patients, improve the quality of life of patients.

Keywords:
Severe traumatic brain injury
Oxiracetam
Tumor necrosis factor
Cerebral contusion
Oxygen

1. Introduction

Severe craniocerebral injury is a common clinical severe disease, the brain injury mainly caused by external forces[1]. Clinical study[2] has found that oxiracetam can treat severe craniocerebral injury patients with obvious therapeutic effect, but its mechanism report is still rare. Therefore, we adopted oxiracetam to treat severe craniocerebral injury and its influence on tumor necrosis factor (TNF-α) was observed. Now report as follows.

2. Materials and methods

2.1. Clinical information

92 patients who are aged from 37 to 78 years old with severe craniocerebral injury were collected since January 2012 to August 2014 in our hospital. 92 patients were divided into control group and observation group by random number table method; there were 46 patients in each group. There were 25 male patients and 21 female patients in the control group whose average age was (48.4±7.3) years old. There were 60.87% (28/46) of patients suffered from cerebral contusion and laceration, 23.91% (11/46) of patients suffered from subdural hematoma and 15.22% (7/46) of patients suffered from epidural hematoma. There were 24 male patients and 22 female patients in the control group whose average age was (48.9±7.7) years old. There were 58.70% (27/46) of patients suffered from cerebral contusion and laceration, 21.74% (10/46) of patients suffered from epidural hematoma. There were 24 male patients and 22 female patients in the control group whose average age was (48.9±7.7) years old. There were 58.70% (27/46) of patients suffered from cerebral contusion and laceration, 21.74% (10/46) of patients suffered from epidural hematoma. There was no statistically significant difference between two groups from the aspects of age, sex and craniocerebral injury types (P>0.05), it is comparable.
2.2. Treatment method

The control group was treated by routine treatment of severe craniocebral injury which including oxygen uptake anti-infection, hemostatic and dehydration to release encephalomalacia, as well as nutritional support. The observation group was treated by oxiracetam (Produced by sanlian Harbin pharmaceutical co., LTD., approval number: H20060070) besides the routine treatment of severe craniocebral injury. The patients were intravenous dripped by 4 g of oxiracetam injection and 250 mL of dextrose injection once a day for 2 weeks. The patients must be treated by oxiracetam one day after surgery if the patients need surgery.

2.3. Observing indexes

(1) Intracranial pressure (ICP)[3]: the changes of ICP level of both group before and after treatment were observed which was monitored under the dura mater. (2) Grade of Glasgow Coma Scale (GCS) and Glasgow Prognosis Score (GOS) and Barthel index[4]; the changes of GCS and GOS grade were observed , as well as the Barthel index. (3) Inflammation index[5]; the inflammation indexes including interleukins-1β (IL-1β), tumor necrosis factor (TNF-α) was observed and tested by radioimmunoassay

2.4. Evaluation of therapeutic efficiency

The curative standard proposed by references[6]. If the symptoms disappeared and patients gain self-care ability, it will be considered as cure. If the symptoms were obviously improved and the patient can take care himself basically, it will consider as excellent. If the symptoms improved and self-care ability improved also, it will be considered as effective. If there were no obvious changes of symptom and self-care ability, it will be considered as invalid.

2.5. Statistical analysis

SPSS13.0 software was adopted to analyze the data. Measurement data was symbolled by mean±SD and tested by t test. Enumeration data was tested by χ² test. There was statistical significance when P<0.05.

3. Results

3.1. Comparison of curative efficiency

The total effective rate of the observation group was 95.65%; the cure rate of the observation group was 54.35%; the excellent rate of the observation group was 23.91%; the effective rate of the observation group was 17.39%; the invalid rate of the observation group was 4.35%. The total effective rate of the control group was 80.44%; the cure rate of the control group was 36.96%; the excellent rate of the control group was 26.09%; the effective rate of the control group was 17.39%, the invalid rate of the control group was 19.56%. The differences were statistically significant (χ² = 5.560, P<0.05).

3.2. Comparison of ICP level

The ICP level after treatment in both groups was lower than that before treatment. And the ICP level of the observation group was lower than the control group. The differences were statistically significant (P<0.01), see Table 1.

<table>
<thead>
<tr>
<th>Group</th>
<th>Before treatment</th>
<th>After treatment</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>314.2±12.5</td>
<td>251.8±9.8</td>
<td>26.645</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Observation</td>
<td>316.9±13.0</td>
<td>208.7±7.6</td>
<td>48.733</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

3.3. Comparison of GCS, GOS grade and Barthel index

The GCS, GOS grade and Barthel index after treatment in both groups were lower than that before treatment. The differences were statistically significant (P<0.01). And the GCS, GOS grade and Barthel index of the observation group were higher than the control group. The differences were statistically significant (P<0.01), see table2.

<table>
<thead>
<tr>
<th>Group</th>
<th>GCS score</th>
<th>GOS score</th>
<th>Barthel index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Control</td>
<td>5.60±1.28</td>
<td>9.40±2.03</td>
<td>2.38±0.32</td>
</tr>
<tr>
<td>Observation</td>
<td>5.58±1.25</td>
<td>10.96±2.31</td>
<td>2.44±0.38</td>
</tr>
<tr>
<td>t</td>
<td>0.076</td>
<td>3.440</td>
<td>0.546</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.05</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

3.4. Comparison of inflammatory indexes

The IL-1β and TNF-α level after treatment in both groups was lower than that before treatment. The differences were statistically significant (P<0.01). And IL-1β and TNF-α level of the observation group were lower than the control group. The differences were statistically significant (P<0.01), see table3.

<table>
<thead>
<tr>
<th>Group</th>
<th>IL-1β</th>
<th>TNF-α</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>Control</td>
<td>38.75±6.18</td>
<td>32.38±5.82</td>
</tr>
<tr>
<td>Observation</td>
<td>38.86±6.62</td>
<td>25.67±5.18</td>
</tr>
<tr>
<td>t</td>
<td>0.882</td>
<td>5.841</td>
</tr>
<tr>
<td>P</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

4. Discussion

Investigation shows that the number of cases of heavy craniocebral injury has increased in recent years, which is related with high incidence rate of the traffic accident and occupational injury. The prognosis of patients with severe craniocebral injury is
The symptoms of patients with severe craniocerebral injury are nausea, feeling obstacle, confusion, and limb paralysis; there will be serious complications such as pulmonary infection, gastrointestinal bleeding, and renal failure. The mortality rate and disability rate of patients with severe craniocerebral injury is high. In this study, we adopted conventional symptomatic treatment for the control group. The observation group was treated by oxiracetam besides the conventional symptomatic treatment. Results show that the total effective rate (%) of the observation group is higher than that of the control group (80.4%). The ICP level of the observation group after treatment is lower than that of the control group. The GCS, GOS grade, and Barthel indexes after treatment are higher than that of the control group, the difference is statistically significant. It improved that oxiracetam is effective to treat severe craniocerebral injury; it can reduce intracranial pressure and improve the quality of life.

Patients generally exist obvious inflammation reaction after craniocerebral injury inflammation will further aggravate the brain tissue damage. Study found that the IL-1β and TNF-α level were obviously raised in patients with severe craniocerebral injury, which may be one of the important cause of brain edema. IL-1β belongs to the proinflammatory cytokines, which not only involved in the inducing of brain edema but also the secretion of inflammatory medium such as TNF-α. High level of TNF-α may cause irreversible cognitive and neural dysfunction. Therefore, it has great significance to control the inflammatory response in time of patients with severe craniocerebral injury after arrive hospital. The IL-1β and TNF-α level of the observation group after treatment were lower than that of the control group according to the results, the difference is statistically significant. It suggests that oxiracetam can release the inflammatory response of patients with severe craniocerebral injury. The author thinks this may because the oxiracetam is derivatives of aminobutyric acid which belongs brain activator. Oxiracetam can act on cholinergic neurons to improve the cerebral metabolic rate. Oxiracetam not only can promote the formation of phosphatidyl choline and phosphoryl ethanol amine to speed up the transport rate of acetylcholine at the cerebral cortex and hippocampus region, but also can reinforce the activity of phosphatidase A1 to restrict the breakdown of cephalin and benefit the formation of DNA and RNA to promote the recovery of cerebral cells. Patients with severe craniocerebral injury tend to suffer from sequelae such as amnesia. Oxiracetam can release cerebral vasospasm to reduce vascular resistance and inhibit platelet aggregation to prevent the formation of thrombus. Moreover, oxiracetam can increase perfusion of brain tissue to benefit brain microcirculation and promote nutrition metabolism of brain cells to enhance the recovery of damaged cortex function. Studies have shown that oxiracetam can effectively inhibit lipid peroxidation and reduce local inflammation to avoid further damage to the brain tissue, stabilize the patient and promote the prognosis of patients.

In conclusion, the curative effect of oxiracetam for severe craniocerebral injury is exact. It can effectively reduce the intracranial pressure of patients with severe craniocerebral injury, relieve inflammatory reaction, promote the rehabilitation prognosis and improve the patient's life quality.

References


