CLINICAL FEATURES AND RISK FACTORS ANALYSIS OF DELAYED ENCEPHALOPATHY AFTER ACUTE CARBON MONOXIDE POISONING

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ABSTRACT

Objective: To explore clinical features and risk factors of delayed encephalopathy after acute carbon monoxide poisoning (DEACMP).

Methods: A total of 184 ACMP patients admitted in the Department of Neurology, The First Hospital of Lanzhou University from 2008 to 2016 were recruited as study subjects. They were randomly divided into the study group (DEACMP) and the control group (non-DEACMP). Twelve (12) indices (age, sex, coma time and JSC score, COHb, WBC, CK, CK-MB, LDH, abnormal skull CT, hospitalization time, and hyperbaric oxygen therapy) of patients in the two groups were analyzed.

Results: ACMP patients in two groups, there were 114 cases with coma time of patients in the control group was 12h or less 12h, 19 patients with 13h to 24h, 11 cases with 25h to 48h, 4 cases with over 48h. 36 delayed encephalopathy cases in 184 ACMP patients were the study group. The incidence rate was 19.56%. There were 13 cases with coma time of patients in the study group was 12h or less 12h, 11 patients with 13h to 24h, 5 cases with 25h to 48h, 7 cases with over 48h. The coma time in the study group was longer. Patients in the study group compared with the control group in ratio of male and female, there were no differences in mean age and laboratory indexes (COHb concentration and WBC), data had no statistical significance (P > 0.05). According to Japan coma scale (JSC) scoring, it was found that consciousness dysfunction when admitted into hospital in the study group was worse compared with the control group. At the same time, Cranial CT in the study group showed that patients with hypoxic hypoxia brain change, which compared with the control group, the percentage was more high, differences had statistical significance (P < 0.01). According to indexes in experiment, it showed that CK, CK-MB and LDH level significantly increased in the study group compared with the control group. Compared with the control group, hospitalization time in the study group was longer and hyperbaric oxygen treatment had been prolonged. Differences between two groups had statistical significance (P < 0.01).

Conclusion: DEACMP patients should particularly pay attention to strengthen clinical monitor to decrease incidence of DEACMP.

Keywords: Delayed Encephalopathy; Acute Carbon Monoxide poisoning.

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Introduction

Delayed encephalopathy after acute carbon monoxide poisoning (DEACMP) refers to ACMP patients after salvage treatment, the clinical symptoms disappeared. 10% to 30% patients after 2 to 60d false recovery will have a series of neuropsychiatric symptoms, mainly acute stupid(1). Incidence rate of DEACMP patients in our country is from 20% to 30%(2).

After that, international incidence rate of DEACMP patients are 30% to 40%(3). Doctors need to treat and DEACMP patients immediately and evaluate prognosis. There are no effective methods to prevent and treat incidence of DEACMP. This study selects carbon monoxide poisoning patients in recent years in order to explore and analyze clinical features and influences of risk factors of DEACMP. Conditions are as the following.
Data and methods

General data

184 ACMP patients in our hospital from 2008 to 2016 were recruited as study subjects. They were randomly divided into the study group and the control group. The ratio between male and female of patients in two groups were 106 cases and 78 cases. Ages were from 25 to 82 years. Mean age was 46.7±12.8 years.

Diagnostic criteria

Acute carbon monoxide poisoning patients had explicit coma history of acute carbon monoxide poisoning, which met diagnostic criteria of acute carbon monoxide poisoning (GB8781-88)\(^4\). Clinical symptoms of DEACMP patients were psychological and consciousness dysfunction with state of dementia, catalepsy, delirium or decortical state; conic system nerve injury including hemiplegia, positive pathological reflection or incontinence of urine and defecation; manifestations of paralysis agitans syndromes: dysfunction of cerebral cortex focal including aphasia, blindness or epilepsy. Excluded criteria concerned there were no coma history, no false recovery period, incomplete cases, death of ACMP patients, loss of following-up etc. The control group and the study group all met diagnosis criteria and excluded criteria; all were hospitalization patients with routine internal treatment; the following-up was 90 days or over.

Observation indexes

Observation and analysis on CMP patients in two groups: 12 indexes including sex, age, coma time, JSC score\(^5\), COHb, WBC, CK, CK-MB, LDH, abnormal cranial CT, hospitalization and hyperbaric oxygen therapy.

Statistical methods

SPSS 19.0 software used to do data analysis. Measurement data were represented as mean ± SD. Measurement data were done with ANOVA analysis, \(\chi^2\) test, t test and Mann-whitney U test. Statistical significance was assumed at P < 0.05.

Results

DEACMP comparison of ACMP patients in different coma time points

ACMP patients in two groups, there were 114 cases with coma time of patients in the control group was 12h or less 12h, 19 patients with 13h to 24h, 11 cases with 25h to 48h, 4 cases with over 48h. 36 delayed encephalopathy cases in 184 ACMP patients were the study group. The incidence rate was 19.56%. There were 13 cases with coma time of patients in the study group was 12h or less 12h, 11 patients with 13h to 24h, 5 cases with 25h to 48h, 7 cases with over 48h. The coma time in the study group was longer (Table 1).

<table>
<thead>
<tr>
<th>Come time</th>
<th>The control group</th>
<th>The study group</th>
<th>Total</th>
<th>DEACMP incidence rate (%)</th>
<th>(\chi^2) value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤12h</td>
<td>114</td>
<td>13</td>
<td>127</td>
<td>10.24</td>
<td>96.74</td>
<td>0.001</td>
</tr>
<tr>
<td>13h-24h</td>
<td>19</td>
<td>11</td>
<td>30</td>
<td>36.67</td>
<td>31.25</td>
<td></td>
</tr>
<tr>
<td>25h-48h</td>
<td>11</td>
<td>5</td>
<td>16</td>
<td>63.63</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 48h</td>
<td>4</td>
<td>7</td>
<td>11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>148</td>
<td>36</td>
<td>184</td>
<td>19.56</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 1: DEACMP comparison of ACMP patients in different coma time points.

Analysis and comparison of clinical relevant indexes of CMP patients in two groups

Patients in the study group compared with the control group in ratio of male and female, mean age and laboratory indexes (COGb concentration and WBC) had no differences, data had no statistical significance (\(P > 0.05\)). According to JSC scoring, it was found that consciousness dysfunction when admitted into hospital in the study group was worse compared with the control group. At the same time, Cranial CT in the study group showed that patients with hypoxia brain change, which compared with the control group, the percentage was more high, differences had statistical significance (\(P < 0.01\)). According to indexes in experiment, it showed that CK, CK-MB and LDH level significantly increased in the study group compared with the control group, differences had statistical significance (\(P < 0.01\)). Compared with the control group, hospitalization time in the study group was longer and hyperbaric oxygen treatment had been prolonged. Differences between two groups had statistical significance (\(P < 0.01\)). It is shown in the Table 2.

Discussion

The onset mechanism of DEACMP is still unclear. Many scholars believe that it has relations with direct toxin and allergic reaction of capillaries injury in brain, wide small embolism, cerebral edema and CO on cerebral cells and brain\(^6-7\).
Clinical features and risk factors analysis of delayed encephalopathy after acute carbon monoxide poisoning

There are documents show that incidence of DEACMP is linked with age, occupation, coma time, stopping treatment early of CO toxin, particularly hyperbaric oxygen treatment or error diagnosis, thus delaying treatment etc. There are reports show that senility will accelerate incidence of DEACMP. Ages of patients without DEACMP less than 30 years. Therefore, older people are more easy to have DEACMP. ACMP patients is necessary. Therefore, patients should pay attention to strengthen clinical monitor to decrease incidence of DEACMP.

Results of this study show that patients in the study group compared with the control group in ratio of male and female, mean age and laboratory indexes (COHb concentration and WBC) have no differences, data have no statistical significance (P>0.05). According to JCS scoring, it is found that consciousness dysfunction when admitted into hospital in the study group is worse compared with the control group. At the same time, Cranial CT in the study group show that patients with hypoxic hypoxia brain change, which compared with the control group, the percentage is more high, differences have statistical significance (P<0.01). According to indexes in experiment, it shows that CK, CK-MB and LDH level significantly increase in the study group compared with the control group, differences have statistical significance (P<0.01). Compared with the control group, hospitalization time in the study group is longer and hyperbaric oxygen treatment had been prolonged. Differences between two groups have statistical significance (P<0.01).

In conclusion, patients regard CO toxin as suicide methods and risk factors of DEACMP. It includes persistent time of coma, hypoxic cerebral changes, high-level CK, CK-MB and LDH. Early learning of DEACMP, positively communicating with families to give hyperbaric oxygen treatment. During this period, follow-up given for high-risk DEACMP patients is necessary. Therefore, DEACMP patients should particularly pay attention to strengthen clinical monitor to decrease incidence of DEACMP.

Table 2: Analysis and comparison of clinical relevant indexes of CMP patients in two groups.


<table>
<thead>
<tr>
<th>Indexes</th>
<th>The study group (n=7)</th>
<th>The control group (n=184)</th>
<th>Statistical check value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (male/female)</td>
<td>19/17</td>
<td>79/69</td>
<td>χ²=0.152</td>
<td>1</td>
</tr>
<tr>
<td>Age (years, x̄±s)</td>
<td>47.2±13.5</td>
<td>46.2±11.8</td>
<td>t=1.382</td>
<td>1</td>
</tr>
<tr>
<td>JCS scoring (points, x̄±s)</td>
<td>215.3±102.58</td>
<td>96.6±62.47</td>
<td>U=9.522</td>
<td>0.001</td>
</tr>
<tr>
<td>COHb-concentration (%x̄±s)</td>
<td>19.8±10.53</td>
<td>24.6±11.65</td>
<td>F=2.128</td>
<td>2.204</td>
</tr>
<tr>
<td>WBC (&gt;109/L, x̄±s)</td>
<td>16.2±11.56</td>
<td>14.05±10.27</td>
<td>F=1.527</td>
<td>0.878</td>
</tr>
<tr>
<td>CK (IU/L, x̄±s)</td>
<td>5887.3±2415.73</td>
<td>2356.45±646.68</td>
<td>F=6.425</td>
<td>0.001</td>
</tr>
<tr>
<td>CK-MB (IU/L, x̄±s)</td>
<td>52.6±32.15</td>
<td>22.8±14.74</td>
<td>F=4.532</td>
<td>0.001</td>
</tr>
<tr>
<td>LDH (IU/L, x̄±s)</td>
<td>398.6±264.38</td>
<td>218.6±98.78</td>
<td>F=1.122</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal cranial CT [Cases (%)]</td>
<td>28 (77.78%)</td>
<td>10 (6.76%)</td>
<td>χ²=0.526</td>
<td>0.001</td>
</tr>
<tr>
<td>Hospitalization time (d, x̄±s)</td>
<td>86.48±24.82</td>
<td>38.12±37.55</td>
<td>F=2.316</td>
<td>0</td>
</tr>
<tr>
<td>Hyperbaric oxygen courses (d, x̄±s)</td>
<td>52.8±14.58</td>
<td>8.21±5.76</td>
<td>F=6.218</td>
<td>0.001</td>
</tr>
</tbody>
</table>
References


