The changes and significance of serum inflammatory factors and hemodynamics in patients with acute cerebral infarction

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ABSTRACT

Objective: To investigate the changes of serum inflammatory factors and hemodynamics in patients with acute cerebral infarction and its clinical significance. Methods: A total of 55 cases of acute cerebral infarction (ACI) patients as observation group, and cases of healthy physical examination were selected as the observation group, and 55 healthy persons as control group. ELISA method was used to detect inflammatory cytokines interleukin-6 (IL-6), interleukin-8 (IL-8), C-reactive protein (CRP) and tumor necrosis factor (TNF-α) level, WA-880 heart and brain integrated digital hemodynamic monitor to detect bilateral carotid artery blood flow velocity, blood flow and peripheral resistance. Results: The serum levels of IL-8, CRP, IL-6 and TNF-α were higher in the observation group than in the control group, the difference was statistically significant (P<0.05). The blood flow velocity and blood flow velocity in the observation group were significantly lower than those in the control group. The difference was statistically significant (IL-8). With the increase of infarct size, serum IL-6, CRP, P<0.05 and TNF-α increased significantly (P<0.05). Conclusions: The changes of serum inflammatory factors and hemodynamic indexes can be used to judge the early cerebral infarction and the size of the infarct size of the index, the clinical dynamic monitoring of its changes in patients with acute cerebral infarction and the severity of the prognosis and the prognosis of the important significance of the judgment.

1. Introduction

Acute cerebral infarction (ACI) refers to the local blood circulation disorder, resulting in cerebral ischemia, hypoxia, and the occurrence of softening necrosis, the corresponding neurologic function defect[1]. ACI is a common cerebrovascular disease, its incidence, morbidity, mortality is high, the quality of life for patients, family, social impact, so early to take active and reasonable prevention and treatment is particularly important[2]. The author analyzes the inflammatory cytokines and hemodynamics in patients with ACI, and discusses its significance in the pathogenesis of ACI.

2. Clinical data

2.1. General information

Area selected 2014 June–2015 June in our hospital from 55 patients with ACI patients as the observation group, of which 31 male patients and 14 female patients, ranging in age from 46 to 78 years old, average (62.7±5.2) years old. According to the stroke patients neurological function defect score standard[3] is divided into three groups, large area cerebral infarction (large area group) (n = 15), cerebral infarction (Group) 21 cases of small area cerebral infarction (small area group) (n = 19). All the patients were first onset at 72 h after treatment by CT or MRI, and meet the ACI diagnosis standard[4], to the exclusion of recurrent cerebral infarction and is associated with severe disturbance of consciousness, serious heart, liver and kidney disease, autoimmune disease, severe infection and so on. Another 55 healthy persons were selected as control group, including 30 males and 15 females,
aged from 78 to 45 years, with an average of years (62.3±5.7). The two groups of subjects were excluded from the factors that affect the inflammatory factors and cerebral blood flow dynamics, and there was no significant difference in age and sex.

2.2. Method

The two groups of subjects were taken from the early morning fasting blood 5 mL, 3000 to 15 min, and the supernatant was set at -20 °C. ELISA was used to detect inflammatory cytokines-6 (IL-6), interleukin-8 (IL-8), C-reactive protein (CRP) and tumor necrosis factor (TNF-α) level, the kit was Shanghai, and strictly follow the instructions. The blood flow velocity, blood flow, and peripheral resistance of the bilateral carotid artery were detected by using the WA-880 digital blood flow dynamics monitor.

2.3. Statistical processing

Measurement data to (±s), the group using t test, using SPSS 16 statistical software for analysis. P<0.05 said there was a statistically significant.

3. Results

3.1. Levels of serum inflammatory cytokines in the two groups

ELISA method was used to detect the serum levels of IL-6, IL-8, CRP and TNF-α in the observation group were significantly higher than those in the control group, the difference was statistically significant (P<0.05) (Table 1).

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>IL-6 (pg/mL)</th>
<th>IL-8 (ng/mL)</th>
<th>CRP (mg/L)</th>
<th>TNF-α (pg/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>55</td>
<td>83.65±6.71</td>
<td>46.81±3.15</td>
<td>15.90±2.61</td>
<td>176.52±21.17</td>
</tr>
<tr>
<td>Control</td>
<td>55</td>
<td>37.21±5.71</td>
<td>21.70±1.55</td>
<td>4.58±2.12</td>
<td>41.15±7.58</td>
</tr>
</tbody>
</table>

Compared with the control group, P<0.05

3.2. Comparison of hemodynamic parameters in two groups

The blood flow velocity and blood flow of the observation group were less than those of the control group, and the peripheral resistance was significantly higher than that in the control group, and the difference was statistically significant (P<0.05) (Table 2).

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Blood flow velocity</th>
<th>Blood flow</th>
<th>Peripheral resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>55</td>
<td>13.55±3.27</td>
<td>8.55±2.67</td>
<td>1885.15±213.58</td>
</tr>
<tr>
<td>Control</td>
<td>55</td>
<td>18.17±1.67</td>
<td>10.03±2.11</td>
<td>372.71±221.07</td>
</tr>
</tbody>
</table>

Compared with the control group, P<0.05

3.3. Levels of serum inflammatory cytokines in different areas of the infarct size were observed

With the increase of infarct size, serum IL-6, IL-8, CRP and TNF-α increased significantly, and the large area and small area, the difference was statistically significant (P<0.05), the difference was statistically significant (P<0.05) (Table 3).

Table 3

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>IL-6 (pg/mL)</th>
<th>IL-8 (ng/mL)</th>
<th>CRP (mg/L)</th>
<th>TNF-α (pg/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large area</td>
<td>15</td>
<td>90.05±3.18</td>
<td>50.12±1.58</td>
<td>22.55±2.17</td>
<td>191.11±16.41</td>
</tr>
<tr>
<td>Middle area</td>
<td>21</td>
<td>84.52±4.91</td>
<td>44.85±3.74</td>
<td>17.53±2.24</td>
<td>172.55±17.88</td>
</tr>
<tr>
<td>Small area</td>
<td>19</td>
<td>75.12±5.31</td>
<td>35.73±2.17</td>
<td>11.73±3.18</td>
<td>125.65±18.90</td>
</tr>
</tbody>
</table>

Compared with small area, small area, *P<0.05, and small area, *P<0.05

3.4. Comparison of hemodynamic parameters of different infarct size in the observation group

With the increase of infarct size, blood flow velocity and blood flow were decreased, and the peripheral resistance increased. The large area and middle area and small area group were significantly different (P<0.05). The difference was statistically significant (P<0.05) (Table 4).

Table 4

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Blood flow velocity</th>
<th>Blood flow</th>
<th>Peripheral resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large area</td>
<td>15</td>
<td>10.92±3.25</td>
<td>6.05±2.55</td>
<td>2152.71±170.11</td>
</tr>
<tr>
<td>Middle area</td>
<td>21</td>
<td>14.45±3.15</td>
<td>8.12±2.07</td>
<td>1901.61±215.37</td>
</tr>
<tr>
<td>Small area</td>
<td>19</td>
<td>17.11±3.18</td>
<td>10.05±2.11</td>
<td>1574.27±231.57</td>
</tr>
</tbody>
</table>

Compared with small area, small area, *P<0.05, and small area, *P<0.05

4. Discussion

Aci is due to cerebral artery blood self-curing formed embolus or exotic emboli into the cerebral artery lumen of blood vessel blockage, so that the clogging artery blood supply to the region of the brain tissue in acute myocardial ischemia, hypoxia, resulting in ischemic brain injury disease, the main pathological basis for atherosclerosis, and atherosclerosis is a chronic inflammatory lesions, many inflammatory cytokines involved the occurrence and development of atherosclerosis, induces platelet aggregation and adhesion, and the formation of unstable plaque and thrombus formation[5,6].

Studies show that serum IL-8, CRP, IL-6 and TNF-α are closely related to Aci[7]. IL-6 is mainly derived from T, B cells and astrocytes, which have immune and inflammatory regulation. By activating macrophages, T lymphocytes and vascular endothelial cells, it is an independent predictor of cerebral infarction. The IL-6
level in brain, plasma and cerebrospinal fluid after central nervous system ischemia[8,9]. IL-8 is a pro-inflammatory factor, main source in macrophages, induction of T and B lymphocytes and natural killer cells secretion of interferon, chemotaxis neutrophil aggregation at the site of inflammation, direct damage to cells and tissues, promoting the generation and development of the inflammation, currently found in the strongest chemotactic factor[10,11]. IL-8, and IL-8 were significantly higher in patients with acute cerebral infarction than in patients with small area infarction and neurological impairment. CRP is produced by activated macrophages, which stimulate the liver cells, which is an acute phase reactive protein. It is closely related to the occurrence and development of atherosclerosis, which can predict the occurrence of cardiovascular events[12]. TNF- is an important inflammatory immune regulating factor. The brain tissue and astrocytes can produce TNF-α. The activated complex of thrombin is formed on the surface of vascular endothelial cells, which can activate the complex of thrombin to induce the deposition of fibrinogen on the vessel wall[13]. Serum inflammatory factors is a kind of cytokine which can promote the inflammatory response in cells. It has a certain effect on the stress state of the body. It may promote the inflammatory reaction and the formation of plaque. The level of serum inflammatory cytokines can reflect the severity of the disease[14]. ACI patients serum IL-8, CRP, IL-6 and TNF-α were higher than the normal group, and with the increase of infarct size, the level of inflammatory factors increased significantly, which was statistically significant (P<0.05), which indicated that high levels of inflammatory cytokines play a role in the pathogenesis of acute cerebral infarction.

With the occurrence of cerebral vascular diseases cerebral vascular function corresponding to a series of changes, hemodynamic abnormalities is one of the important pathogenesis of ischemic cerebrovascular disease, and the occurrence of atherosclerosis, more depends on the protection of endothelial cells and the risk factors of arterial blood, and blood vessel endothelial cell active material on the flow dynamics of the reaction is different, is the formation of atherosclerosis in the cell and molecular basis[15]. The hemodynamic factors of patients with cerebral infarction, and the change of blood pressure level is one of the important factors which affect the blood flow and blood supply of brain tissue, so that the occurrence of cerebral infarction[16]. The study showed that the blood flow velocity and blood flow in patients with acute cerebral infarction were less than those of the control group, and the peripheral resistance was greater than that of the normal control group (P<0.05). With the increase of infarct size, blood flow velocity and blood flow were decreased, and the peripheral resistance increased, the difference was statistically significant (P<0.05). The abnormal fluctuation of blood flow dynamics in patients with cerebral infarction, which suggests that the blood supply of the brain showed a poor state, and it also has a certain significance to understand the size of the infarction.

To sum up, the changes of serum inflammatory factors and hemodynamic indexes can be used to judge the early cerebral infarction and infarct size of ACI, and to monitor the changes of the patients with acute cerebral infarction.

References