Effect of mild hypothermia-assisted minimally invasive surgery on the intracranial hematoma absorption and the degree of nerve injury in patients with hypertensive cerebral hemorrhage

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ABSTRACT

Objective: To study the effect of mild hypothermia-assisted minimally invasive surgery on the intracranial hematoma absorption and the degree of nerve injury in patients with hypertensive cerebral hemorrhage. Methods: 90 patients with hypertensive cerebral hemorrhage who were treated in our hospital between April 2013 and February 2016 were collected and divided into control group (n=47) who received minimally invasive surgery and observation group (n=43) who received mild hypothermia-assisted minimally invasive surgery after the therapy that patients received and the clinical findings were reviewed. Immediately after admission and 1 week after treatment, head CT was used to record the intracranial hematoma volume of both groups, and the transcranial Doppler (TCD) was used to detect the middle cerebral arterial hemodynamic parameters; automatic biochemical analyzer was used to detect serum contents of nerve injury indexes, thiobarbituric acid method was used to detect the contents of oxidation/anti-oxidation factors, and the enzyme-linked immunosorbent assay (ELISA) was used to determine the contents of inflammatory cytokines. Results: Before treatment, the differences in intracranial hematoma volume and affected-side middle cerebral arterial hemodynamic parameters as well as serum contents of nerve injury indexes, oxidation/anti-oxidation factors and inflammatory cytokines were not statistically significant between two groups of patients (P>0.05). 1 week after treatment, the intracranial hematoma volume of observation group was less than that of control group, the affected-side middle cerebral arterial hemodynamic parameters peak systolic velocity (Vs), mean flow velocity (Vm) and relative cerebral blood flow (rCBF) levels were higher than those of control group while the time to peak (TTP) level was lower than that of control group, and serum insulin-like growth factor (IGF-1), stromal cell-derived factor-1 (SDF-1), total antioxidant capacity (T-AOC) and superoxide dismutase (SOD) contents were higher than those of control group while serum human cartilage glycoprotein 39 (YKL-40), malondialdehyde (MDA), C-reactive protein (CRP), interleukin-1 (IL-1), IL-8 and tumor necrosis factor-α (TNF-α) contents were lower than those of control group (P<0.05). Conclusion: Mild hypothermia-assisted minimally invasive surgery can promote the intracranial hematoma absorption and reduce the degree of nerve injury in patients with hypertensive cerebral hemorrhage.

1. Introduction

Hypertensive cerebral hemorrhage is a severe complication of hypertension that trends to occur in middle-aged and elderly men, and may attack in the case of rage and excessive mental labor. Minimally invasive drainage of intracranial hematoma is the preferred way for hypertensive cerebral hemorrhage treatment, and reducing the hematoma compression in time can reduce the nerve injury and decrease the incidence of risk of secondary hemorrhage[1,2]. Minimally invasive surgery can drain most of the intracranial hemorrhage, but postoperative residual hemorrhage can still cause some damage to the lesion area and the surrounding neurons, and the therapy cannot reverse the existing neural function

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damage, so the overall treatment effect is limited[3]. The cerebral protective effect of mild hypothermia-assisted treatment has been recognized by many researches[4], but the effect of the auxiliary treatment on the serum indexes of patients with hypertensive cerebral hemorrhage is unclear. In the following study, the effect of mild hypothermia-assisted minimally invasive surgery on the intracranial hematoma absorption and the degree of nerve injury in patients with hypertensive cerebral hemorrhage was analyzed.

2. Materials and methods

2.1. Inclusion and exclusion criteria

Inclusion criteria: (1) diagnosed with cerebral hemorrhage by head CT; (2) with history of essential hypertension; (3) without previous history of cerebral hemorrhage; (4) with normal bleeding and clotting function before admission; (5) the time interval between onset and admission ≤6 h. Exclusion criteria: (1) with congenital cerebrovascular malformation; (2) with the history of head injury; (3) combined with basic limb dysfunction; (4) with severe heart, liver and kidney dysfunction; (5) with local cerebral or systemic infectious diseases; (6) associated with Alzheimer’s disease or dementia and other cognitive disorders.

2.2. Clinical information

90 patients with hypertensive cerebral hemorrhage who were treated in our hospital between April 2013 and February 2016 were included, and patients' families signed informed consent. The therapies and clinical findings of the included patients were reviewed, and then they were divided into control group (n=47) who received minimally invasive surgery and observation group (n=43) who received mild hypothermia-assisted minimally invasive surgery. Control group included 25 male cases and 22 female cases, they were 49–78 years old, the time between onset and admission was 1–5 h and (2.37±0.59) h in average, and the body weight was 49–83 kg and (71.59±8.63) kg in average; observation group included 23 male cases and 20 female cases, they were 47–76 years old, the time between onset and admission was 1–6 h and (2.45±0.63) h in average, and body weight was 48–82 kg and (70.64±8.95) kg in average. Differences in gender, age, admission time and weight distribution were not statistically significant between two groups of patients (P>0.05), and the hospital ethics committee approved the study.

2.3. Treatment methods

Control group received minimally invasive puncture and drainage, specifically as follows: the CT scan results were referred to clarify the final and needle-inserting level of hematoma, 2 cm was opened near frontal midline for puncture, the inserting depth was long axis of hematoma + 1–2 cm, the hematocele of 20% of total hematoma was extracted at first, and then urokinase was used once every 8 h to dissolve and liquefy the hematoma, and then the hematocele was extracted out of the body via drainage tube. The drainage tube was pulled out after CT confirmed that most of the hematoma (> 80%) was removed.

Observation group of patients received mild hypothermia-assisted minimally invasive surgery, specifically as follows: the medical low-temperature treatment instrument (Zhuhai Hokai Medical Instruments Co., Ltd., HGT-200II) was used, patients were de-pillowed and lay on the back, the head was placed in the ice cap and towards the affected side, soft pillow was placed between the ice cap and the inner cap, the ears were padded with antifreeze pad, thin sponge was used to fill the ice cap space, and the temperature was set at -4 ℃ to 4 ℃. Ice bag was packed on neck artery. Infrared ear thermometer (Beijing Jetong Kangnuo Pharmaceutical Technology Co., LTD., model ICT-100) was used to measure temperature every 30min, the temperature of the ice cap was controlled, and the tympanic temperature was controlled at 33 ℃ to 35.5 ℃ 2 d. The ice packs and head temperature-decreasing instrument were removed in turn for rewarming, the rewarming was done naturally at room temperature, and the rate of temperature increase was below 1 ℃/4 h. In the process of cooling and rewarming, patients received ECG monitoring.

2.4. Intracranial hematoma volume and craniocerebral hemodynamic parameters

Immediately after admission and after 1 week treatment, two groups of patients received head CT to get the intracranial hematoma volume. Transcranial Doppler (TCD) was used to detect the hemodynamic parameters of middle cerebral artery, including peak systolic velocity (Vs), time to peak (TTP), mean flow velocity (Vm) and relative cerebral blood flow (rCBF).

2.5. Nerve injury indexes

Immediately after admission and after 1 week treatment, 1.5 mL of fasting cubital venous blood was extracted from two groups of patients, anti-coagulated with sodium citrate (Guangxi Ecan Pharmaceutical Co., LTD., approved by H45020137), let stand for 30 min at room temperature and centrifuged at low speed (2 500 r/min) for 15 min, and the supernatant was collected and frozen at -20 ℃ for test. Specific detection indexes were as follows: (1) nerve injury indexes: automatic biochemistry analyzer (Hitachi High-tech International Trade Co., LTD., model 7600-200) was used to detect
insulin-like growth factor (IGF-1), human cartilage glycoprotein 39 (YKL-40) and stromal cell-derived factor-1 (SDF-1) contents; (2) oxidation/anti-oxidation factors: thiobarbituric acid method was used to detect total antioxidant capacity (T-AOC), malondialdehyde (MDA) and superoxide dismutase (SOD) contents; (3) inflammation factors: the ELISA method was used to determine C-reactive protein (CRP), interleukin-1 (IL-1), interleukin-8 (IL-8) and tumor necrosis factor-α(TNF-α) contents.

2.6. Statistical analysis

The data in the study was input in the statistical software SPSS18.0 for related processing, measurement data was in terms of mean ± standard deviation (\(\bar{X} \pm s\)), comparison before and after treatment was by paired \(t\) test, separate comparison before and after treatment was by grouping \(t\) test, and \(P<0.05\) indicated statistical significance in differences.

3. Results

3.1. Intracranial hematoma volume

Before and after treatment, comparison of intracranial hematoma volume between two groups of patients was as follows: before treatment, the intracranial hematoma volume of observation group was (33.28±5.17) mL, intracranial hematoma volume of control group was (32.41±5.38) mL, and differences between groups were not statistically significant (\(P>0.05\)). After treatment, intracranial hematoma volume of observation group was (5.14±0.67) mL, intracranial hematoma volume of control group was (9.64±0.92) mL, the intracranial hematoma volume of observation group was less than that of control group, and differences between groups were statistically significant (\(P<0.05\)).

3.2. Craniocerebral hemodynamic parameters

Before and after treatment, the comparison of craniocerebral hemodynamic parameters \(\text{Vs}, \text{TTP}, \text{Vm}\) and \(\text{rCBF}\) between two groups of patients was as follows: before treatment, differences in craniocerebral hemodynamic parameters \(\text{Vs}, \text{TTP}, \text{Vm}\) and \(\text{rCBF}\) levels were not statistically significant between two groups of patients (\(P>0.05\)); after treatment, craniocerebral hemodynamic parameters \(\text{Vs}, \text{Vm}\) and \(\text{rCBF}\) levels of both groups were higher than those before treatment while the \(\text{TTP}\) levels were lower than those before treatment, and differences within group were statistically significant before and after treatment (\(P<0.05\)). After treatment, craniocerebral hemodynamic parameters \(\text{Vs}, \text{Vm}\) and \(\text{rCBF}\) levels of observation group were higher than those of control group while the \(\text{TTP}\) level was lower than that of control group, and differences between groups were statistically significant after treatment (\(P<0.05\)), shown in Table 1.

3.3. Nerve injury indexes

Before and after treatment, the comparison of serum nerve injury indexes IGF-1, YKL-40 and SDF-1 contents between two groups of patients was as follows: before treatment, differences in serum nerve injury indexes IGF-1, YKL-40 and SDF-1 contents were not statistically significant between two groups of patients (\(P>0.05\)); after treatment, serum nerve injury indexes IGF-1 and SDF-1 contents of both groups were higher than those before treatment while YKL-40 contents were lower than those before treatment, and differences within group were statistically significant before and after treatment (\(P<0.05\)). After treatment, serum nerve injury indexes IGF-1 and SDF-1 contents of observation group were higher than those of control group while YKL-40 content was lower than that of control group, and differences between groups were statistically significant after treatment (\(P<0.05\)), shown in Table 2.

### Table 1

Comparison of craniocerebral hemodynamic parameter levels between two groups of patients before and after treatment (\(\bar{X} \pm s\)).

<table>
<thead>
<tr>
<th>Groups</th>
<th>(n)</th>
<th>(\text{Vs (mL/min)}) Before treatment</th>
<th>(\text{After treatment})</th>
<th>(\text{TTP (s)}) Before treatment</th>
<th>(\text{After treatment})</th>
<th>(\text{Vm (cm/s)}) Before treatment</th>
<th>(\text{After treatment})</th>
<th>(\text{rCBF [mL/(100 g·min)]}) Before treatment</th>
<th>(\text{After treatment})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>43</td>
<td>56.82±7.19</td>
<td>78.53±8.42</td>
<td>14.83±1.79</td>
<td>8.41±0.95</td>
<td>42.18±5.09</td>
<td>57.42±6.09</td>
<td>9.62±0.98</td>
<td>21.47±2.38</td>
</tr>
<tr>
<td>Control</td>
<td>47</td>
<td>56.74±6.83</td>
<td>69.36±7.21</td>
<td>14.52±1.68</td>
<td>11.76±1.84</td>
<td>41.34±4.87</td>
<td>49.63±5.21</td>
<td>9.53±0.95</td>
<td>15.61±1.87</td>
</tr>
<tr>
<td>(t)</td>
<td></td>
<td>0.183</td>
<td>0.154</td>
<td>7.273</td>
<td>0.189</td>
<td>9.182</td>
<td>0.153</td>
<td>7.251</td>
<td></td>
</tr>
<tr>
<td>(P)</td>
<td></td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 2

Comparison of serum nerve injury index contents between two groups of patients before and after treatment (\(\bar{X} \pm s\)).

<table>
<thead>
<tr>
<th>Groups</th>
<th>(n)</th>
<th>IGF-1 (nmol/L) Before treatment</th>
<th>After treatment</th>
<th>YKL-40 (μg/L) Before treatment</th>
<th>After treatment</th>
<th>SDF-1 (ng/mL) Before treatment</th>
<th>After treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>43</td>
<td>24.38±2.96</td>
<td>53.79±6.15</td>
<td>215.82±24.76</td>
<td>54.27±6.09</td>
<td>13.28±1.76</td>
<td>27.54±3.09</td>
</tr>
<tr>
<td>Control</td>
<td>47</td>
<td>24.45±2.84</td>
<td>39.64±4.72</td>
<td>209.95±23.57</td>
<td>89.61±9.75</td>
<td>13.09±1.58</td>
<td>21.63±2.58</td>
</tr>
<tr>
<td>(t)</td>
<td></td>
<td>0.194</td>
<td>8.394</td>
<td>0.116</td>
<td>11.284</td>
<td>0.163</td>
<td>7.384</td>
</tr>
<tr>
<td>(P)</td>
<td></td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>
3.4. Oxidation/anti-oxidation factors

Before and after treatment, the comparison of serum oxidation/anti-oxidation factors T-AOC, MDA and SOD contents between two groups of patients was as follows: before treatment, the differences in serum oxidation/anti-oxidation factors T-AOC, MDA and SOD contents were not statistically significant between two groups of patients \((P>0.05)\); after treatment, serum oxidation factor MDA contents of both groups were lower than those before treatment, anti-oxidation factors T-AOC and SOD contents were higher than those before treatment, and differences within group were statistically significant before and after treatment \((P<0.05)\). After treatment, serum oxidation factor MDA content of observation group was lower than that of control group, anti-oxidation factors T-AOC and SOD contents were higher than those of control group, and differences between groups were statistically significant after treatment \((P<0.05)\), shown in Table 3.

3.5. Inflammatory cytokines

Before and after treatment, the comparison of serum inflammatory cytokines CRP, IL-1, IL-8 and TNF-α contents between two groups of patients was as follows: before treatment, the differences in serum inflammatory cytokines CRP, IL-1, IL-8 and TNF-α contents were not statistically significant between two groups of patients \((P>0.05)\); after treatment, the serum inflammatory cytokines CRP, IL-1, IL-8 and TNF-α contents of both groups were lower than those before treatment, and differences within group were statistically significant before and after treatment \((P<0.05)\). After treatment, serum inflammatory cytokines CRP, IL-1, IL-8 and TNF-α contents of observation group were lower than those of control group, and differences between groups were statistically significant after treatment \((P<0.05)\), shown in Table 4.

### Table 3
Comparison of serum oxidation/anti-oxidation factor contents between two groups of patients before and after treatment \((t<0.05)\).

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>T-AOC (U/mL) Before treatment</th>
<th>After treatment</th>
<th>MDA (mmol/mL) Before treatment</th>
<th>After treatment</th>
<th>SOD (U/mL) Before treatment</th>
<th>After treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>43</td>
<td>11.28±1.76</td>
<td>19.75±2.48</td>
<td>3.48±0.45</td>
<td>1.24±0.16</td>
<td>45.28±5.09</td>
<td>83.76±9.55</td>
</tr>
<tr>
<td>Control</td>
<td>47</td>
<td>11.34±1.68</td>
<td>15.18±1.76</td>
<td>3.51±0.47</td>
<td>2.18±0.25</td>
<td>45.17±5.26</td>
<td>72.91±8.34</td>
</tr>
<tr>
<td>t</td>
<td></td>
<td>0.173</td>
<td>6.495</td>
<td>0.109</td>
<td>6.283</td>
<td>0.174</td>
<td>9.283</td>
</tr>
<tr>
<td>P</td>
<td></td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

### Table 4
Comparison of serum inflammatory cytokine contents between two groups of patients before and after treatment \((t<0.05)\).

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>CRP (mg/L) Before treatment</th>
<th>After treatment</th>
<th>IL-1 (pg/mL) Before treatment</th>
<th>After treatment</th>
<th>IL-8 (ng/L) Before treatment</th>
<th>After treatment</th>
<th>TNF-α (ng/L) Before treatment</th>
<th>After treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>43</td>
<td>43.28±5.01</td>
<td>11.07±1.64</td>
<td>74.18±8.53</td>
<td>35.99±4.14</td>
<td>65.28±7.16</td>
<td>21.64±2.85</td>
<td>93.27±9.84</td>
<td>15.38±1.76</td>
</tr>
<tr>
<td>Control</td>
<td>47</td>
<td>42.76±5.32</td>
<td>23.64±2.95</td>
<td>73.26±8.09</td>
<td>51.24±5.76</td>
<td>65.39±7.05</td>
<td>35.92±4.56</td>
<td>92.16±9.53</td>
<td>31.72±3.85</td>
</tr>
<tr>
<td>t</td>
<td></td>
<td>0.183</td>
<td>8.298</td>
<td>0.216</td>
<td>7.261</td>
<td>0.176</td>
<td>8.492</td>
<td>0.153</td>
<td>8.192</td>
</tr>
<tr>
<td>P</td>
<td></td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

4. Discussion

Local hematoma oppresses the nerve tissue after hypertensive cerebral hemorrhage, it leads to ischemic hypoxic neuron damage, and the edema in lesion edge may further affect the surrounding normal tissue function\^[5,6\]. Minimally invasive drainage and local compression relief can effectively prevent the further neuron injury in lesion area, but the residual hematoma can stimulate the neurons to secrete cytotoxic amino acids, and therefore, the overall efficacy of minimally invasive hematoma drainage is limited\^[7\]. Hypothermia is the acknowledged auxiliary cerebral protective means, and its mechanisms of action include: (1) inhibiting leukotriene, nitric oxide and nitric oxide synthase formation after brain injury, and maintaining the stability of cerebrovascular systolic and diastolic function; (2) protecting blood brain barrier; (3) reducing brain cell metabolism rate and cerebral oxygen consumption; (4) inhibiting neuronal ischemic calcium overload; (5) inhibiting endogenous nerve injury factor formation; (6) reducing endogenous antioxidant material consumption, and inhibiting oxygen free radical generation; (7) reducing the diffuse axonal injury and inflammation\^[8,9\].

In order to further enlarge the neuroprotective effect on patients with hypertensive cerebral hemorrhage, many scholars suggest adding mild hypothermia therapy at the same time of minimally invasive surgery, but the previous studies pay more attention to the overall curative effect, and less cover the serum index changes of nerve injury.

In the study, the observation group of patients received mild hypothermia combined with minimally invasive surgery, the hematoma residue was compared between the two groups of patients 1 week after treatment, and it was found that compared with control group, the observation group were with smaller residual hematoma volume after treatment, indicating that the auxiliary mild hypothermia therapy is helpful for the further residual hematoma absorption after minimally invasive surgery. Hypothermia \((30 ^\circ\mathrm{C}–35 ^\circ\mathrm{C})\) is an effective auxiliary cerebral protective means.
was the first to be used in patients with cerebral ischemia and cerebral hypoxia at abroad, and research has confirmed that the mild hypothermia therapy can reduce the focal cerebral edema, which conforms to the results in the study. Both brain hematoma and secondary local edema are affecting the normal blood supply of neurons and can induce the normal surrounding hematoma systolic and diastolic dysfunction, and ultimately affect the cerebral hemodynamics[10]. Middle cerebral artery is a direct continuation of internal carotid artery, it supplies above 80% of blood flow to bilateral hemispheres, and detecting its blood flow state can macroscopically reflect blood flow to the brain hemisphere[11,12]. In the study, comparison of affected-side middle cerebral artery TCD examination results between two groups of patients showed that compared with control group, the observation group were with higher affected-side middle cerebral artery Vs, Vm and rCBF levels, and lower TTP level after treatment ($P<0.05$), confirming that the auxiliary mild hypothermia therapy can increase the blood supply to affected-side hemisphere and reduce the flow resistance. This is mainly associated with the effects of mild hypothermia therapy on promoting local hematoma and edema absorption, reducing intracranial pressure, speeding up the blood flow to affected side, and so on.

Poor treatment outcome for patients with hypertensive cerebral hemorrhage is mainly associated with neuron necrosis and apoptosis, and cerebral hematoma oppresses nerve tissue and causes toxic amino acid release, which directly impact on nerve cell function and result in the generation of a large number of nerve injury-related factors[13]. IGF-1 is a kind of multifunctional cell proliferation regulator, and it has been confirmed that it plays an important role in cell growth, differentiation and proliferation. IGF-1 can repair the damaged neurons and prompt their regeneration, and in the case of sustained neuron injury and apoptosis, IGF-1 content drops due to excessive consumption[14]. YKL-40 is mainly expressed in the cerebrospinal fluid, and plays an important role in secondary nerve injury of stroke. Study has shown that YKL-40 content increases in ischemia area of patients with acute cerebral infarction, and gradually declines in the recovery period[15]. SDF-1 is a member of CXC chemokine family, and its combination with receptor CXCR4 gene can regulate the proliferation and apoptosis of a variety of cells. SDF-1 expression increases in the case of tissue ischemia hypoxia injury in order to promote the wound repair, and it has protective effect on cerebral ischemia. In the study, detection of the contents of above nerve injury markers showed that compared with control group, the observation group were with higher serum IGF-1 and SDF-1 contents, and lower YKL-40 content after treatment ($P<0.05$), confirming that the auxiliary mild hypothermia therapy can prompt nerve cell function recovery.

Brain hemorrhage results in changes of local blood flow and causes generation of oxygen free radicals and inflammatory mediators, which will further damage the nerve function[16]. Oxidative stress plays an important role in cerebral hemorrhagic nerve damage, MDA is a marker oxidation product that participates in the body’s lipid peroxidation, and detecting the MDA level can accurately evaluate the body level of oxidative stress. Both T-AOC and SOD can neutralize oxygen free radicals and reduce oxidative stress, their contents are in dynamic equilibrium with the levels of oxygen free radicals under physiological state, and excessive oxygen free radicals are produced in local brain tissue after cerebral hemorrhage occurs, leading to massive consumption and low levels of T-AOC and SOD[17]. It was found in the study that compared with control group, the observation group were with lower serum MDA content, and higher T-AOC and SOD content after treatment ($P<0.05$), showing that the auxiliary mild hypothermia therapy can optimize the body state of oxidative stress and enhance antioxidant capacity.

Inflammation theory occupies an important place in the nerve injury after recovery, the present study shows that the inflammatory response after total cerebral ischemia mainly comes from the local microglia activation and cytokine release by white blood cells accumulated in blood vessel walls[18]. In vitro culture studies have shown that CRP, IL-1, IL-8, TNF-α and other inflammatory mediators released by the cells after low-temperature treatment significantly reduce[19,20]. In the study, detection of the contents of serum inflammatory mediators of two groups after treatment showed that compared with control group, the observation group were with lower serum CRP, IL-1, IL-8 and TNF-α contents after treatment ($P<0.05$), it confirms the effect of adjuvant mild hypothermia therapy on reducing local cerebral ischemia and systemic inflammatory state, and this is also one of the important mechanisms for mild hypothermia therapy to exert neuroprotective effect.

To sum up, it is concluded that mild hypothermia-assisted minimally invasive surgical treatment of hypertensive cerebral hemorrhage can prompt local hematoma absorption and protect neural function, and it is of positive clinical significance.

References


[4] Li X, Zhang CY, Wang L. Local mild hypothermia adjuvant therapy for


