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Clinical value of detection on serum monocyte chemotactant protein-1 and vascular endothelial cadherin levels in patients with acute cerebral infarction

Xia Zhou[1], Cheng Zhang[2*]

[1]*Department of Neurology, The Third People's Hospital, Zigong 643020, Sichuan Province, China*

[2]*Department of Neurosurgery, The Third People's Hospital, Zigong 643020, Sichuan Province, China*

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ABSTRACT

Objective: To study the correlation of serum monocyte chemotactant protein-1 (MCP-1) and vascular endothelia cadherin (VE-cadherin) levels in patients with acute cerebral infarction, and nerve injury molecules, interleukins and matrix metalloproteinases.

Methods: A total of 86 patients with acute cerebral infarction treated in our hospital from April 2012 to October 2015 were selected as the observation group and 50 healthy subjects in the same period treated in our hospital were selected as the control group. The serums were collected and the contents of MCP-1, VE-cadherin, heart-type fatty acid binding protein (H-FABP), S100 calcium binding protein B (S100B), neuron-specific enolase (NSE), interleukin-1β (IL-1β), IL-6, IL-17, IL-18, matrix metalloproteinase-2 (MMP2), MMP3 and MMP9 were measured.

Results: The serum contents of MCP-1, VE-cadherin, H-FABP, S100B, NSE, IL-1β, IL-6, IL-17, IL-18, MMP2, MMP3 and MMP9 in observation group were significantly higher than those of control group. Carotid artery plaque formation and unstable plaque properties will increase the serum contents of MCP-1, VE-cadherin, H-FABP, S100B, NSE, IL-1β, IL-6, IL-17, IL-18, MMP2, MMP3 and MMP9 in patients with cerebral infarction. The serum levels of MCP-1, VE-cadherin and the contents of H-FABP, S100B, NSE, IL-1β, IL-6, IL-17, IL-18, MMP2, MMP3 and MMP9 were positively correlated.

Conclusions: The serum levels of VE-cadherin and MCP-1 were significantly increased in patients with acute cerebral infarction. MCP-1 and VE-cadherin can increase the secretion of interleukins and matrix metalloproteinases, which can result in the carotid artery plaque formation, unstable plaque properties and the injury of nerve function.

1. Introduction

Acute cerebral infarction is one of the cardiovascular and cerebrovascular diseases seriously threatening to human health with high disabling rate and increased morbidity year by year and the morbidity crowd has a trend of rejuvenation. Cerebral infarction can cause hypoxic-ischemia necrosis of brain tissue and leave the varying degrees of dysfunction, which will have a serious impact on patients' daily life and work[1–3]. Thus, how to screen high-risk groups, early diagnose disease and determine the severity of the disease has been a hot spot in clinical research. Carotid atherosclerosis is an independent risk factor to cause acute cerebral infarction and inflammatory response is an important pathological change through carotid atherosclerosis in all aspects. After the vascular endothelium is injured, the inflammatory cells will get into the vascular wall and mediate inflammatory response, which is considered to be the initial step of atherosclerosis[4–6]. Interleukins (ILs) and matrix metalloproteinases (MMPs) are considered to be the important mediators of inflammation response in the process of mediating carotid atherosclerosis. The formations of atheromatous plaques and the changes of plaque characteristics are related to the local infiltration of the inflammatory mediators above. Local ischemia and hypoxia caused by

*Corresponding author: Cheng Zhang, Department of Neurosurgery, The Third People's Hospital, Zigong 643020, Sichuan Province, China.
Tel: +86 13990010657
E-mail: zhangchenghx@163.com

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cerebral infarction and the ischemia reperfusion by the treatment of intervention or thrombolysis will cause local excessive inflammatory response of the brain tissue and mediate the injury of neural function by ILs and MMPs and other mediators[7–10].

Monocyte chemotactant protein-1 (MCP-1) and vascular endothelial cadherin (VE-cadherin) are the important molecules to modulate inflammatory cell chemotaxis and adhesion to the lesion. In the course of the pathology of injury of neural function after carotid atherosclerosis and cerebral infarction, MCP-1 and VE-cadherin may be the upstream factor to regulate inflammation response and promote the infiltration of inflammatory cells and the release of inflammatory mediators[11–14]. However, the present specific change of MCP-1 and VE-cadherin in patients with acute cerebral infarction is not clear. Besides, the correlation between MCP-1, VE-cadherin and the release of inflammatory mediators and the injury of neural function has not been reported. In the present study, the serum levels of MCP-1 and VE-cadherin in patients with acute cerebral infarction and the correlation with ILs, MMPs and other inflammatory mediators were analyzed.

2. Materials and methods

2.1. Research objects

A total of 86 patients with acute cerebral infarction treated in our hospital from April 2012 to October 2015 and 50 healthy subjects treated in our hospital in the same period were selected to perform prospective study, which was approved by Hospital Ethics Committee. Patients with acute cerebral infarction were served as observation group, with the inclusion criteria as follows: 1. In line with the diagnostic criteria of acute cerebral infarction in the Fourth National Cerebrovascular Disease Conference; 2. The onset time not exceeded 72 h and duration of symptoms for more than 24 h; 3. The brain CT and magnetic resonance imaging were used to confirm the presence of the corresponding to the function of cerebral infarction. Patients with intracerebral hemorrhage, severe heart and lung diseases or hepatic and kidney function obstacle and patients with history of infection in nearly one month were excluded. The 50 healthy subjects were served as control group, excluding patients with a history of acute or chronic inflammatory disease combined with peripheral vascular diseases or thrombotic disease.

2.2. Evaluation methods of carotid plaques properties

After admission for 1 week, carotid ultrasonography was undergone with transducer frequency of 7.5 MHz. The detection range was from carotid artery distal 2.0 cm to internal carotid artery and external carotid artery proximal 1.0 cm, measuring the intima-media thickness of multiple parts. Any side of carotid artery intima-media thickness with more than 1.5 mm was judged as plaque formation, with 1.0–1.5 mm as the thickening of intima and with less than 1.0 mm as a normal intima. The parts in the carotid plaque area were scanned again. If the echo intensity was higher than that of vessel wall, it was judged as high echo plaques; if uneven, it was judged as mixed echo plaques; if lower than that of vessel wall and the plaque highlighted in uterine cavity, it was judged as low echo plaques.

2.3. Evaluation methods of cerebral infarction volume

After admission, emergency CT examination was performed. The length, width and height of the lesion were measured after obtaining the images of cerebral infarction lesion and then the volume of infarction lesion was calculated. The calculating method was as follows:

Volume of infarction lesion (V, cm^3) = length (cm) × width (cm) × height (cm) × $\pi/6$.

V < 5 cm^3 was considered as small area infarction, V = 5–10 cm^3 as middle area infarction, V > 10 cm^3 as large area infarction.

2.4. Serum specimen collections and detection methods

Patients in observation group were immediately collected 10 mL peripheral venous blood when admission to the hospital. The healthy subjects in control group were collected 10 mL peripheral venous blood when taking physical examination. After standing at room temperature for 10–15 min, they were centrifuged for 10 min at 12 000 r/min centrifugal force. Then, the upper serum was collected and the contents of MCP-1, VE-cadherin, heart-type fatty acid binding protein (H-FABP), S100 calcium binding protein B (S100B), neuron-specific enolase (NSE), IL-1β, IL-6, IL-17, IL-18, MMP2, MMP3 and MMP9 were measured by ELISA kits.

2.5. Statistical methods

The data were input and analyzed by SPSS version 19.0 software and the measurement data were performed by mean ± SD. The analyses between two groups were carried out by *t*-test and among three groups by ANOVA. The enumeration data were performed by frequency form and analyzed by *Chi*-square test. The correlations between two variables were verified by Pearson's correlation analysis. Differences were statistically significant when $P < 0.05$.

3. Results

3.1. General clinical data of two groups

In observation group, 86 patients included 51 males and 35 females, with (56.30 ± 7.80) years of age, body mass index (BMI) (23.14 ± 2.97) kg/m^2, 53 cases with high blood pressure, 24 cases with diabetes, 45 cases with smoking history. In control group, 50 patients included 29 males and 21 females, with (55.80 ± 8.60) years of age, BMI (22.48 ± 2.48) kg/m^2, 7 cases with high blood pressure, 3 cases with diabetes, 14 cases with smoking history. According to statistic analysis, there were no differences in sex, age and BMI in two groups, and cases with high blood pressure, diabetes and smoking history in observation group were significantly higher than those of in control group (Table 1).

3.2. Comparison of serum biochemical indexes in two groups

The analyses between two groups on serum biochemical indexes of MCP-1, VE-cadherin, H-FABP, S100B, NSE, IL-1β,

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