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Correlation Analysis of Resistin, Interleukin-6 and Interleukin-1 with Carotid Atherosclerotic Plaques in Elderly Patients with Acute Progressive Cerebral Infarction

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Abstract: Objective: To investigate the relationship between carotid atherosclerotic plaques and resistin, interleukin 6 (il-6), and interleukin 1 (il-1) in elderly patients with acute progressive cerebral infarction. Methods : It was selected that 113 elderly patients with acute progressive cerebral infarction who were hospitalized from June 2016 to December 2018.First of all, the patients were divided into the non-plaque group (n = 30) and the plaque group (n = 83) according to the carotid artery ultrasound results, then according to the characteristics of plaques, the patients were further divided into stable plaques group (n = 32) and unstable plaques group (n = 51), Serum resistin, IL-6, and IL-1βwere compared between the each groups. Result: it was higher than in the non-plaque group that the levels of resistin, IL-6 and IL-1β in the plaque group, and it was statistically significant of the differences between the two groups P <0.05); (2) it was significantly higher that the levels of resistin, IL-6 and IL-1β in the unstable plaque group than that in the stable plaque group (P <0.05), and it was statistically significant of the differences between the two groups P <0.05). Conclusion: It is associated with that carotid artery plaque instability and the progression of cerebral infarction in elderly patients, And It closely related to that carotid plaque instability and the levels of resistance, IL-6 and IL-1β.

Keywords: Resistance; IL-6; IL-1β; Progressive cerebral infarction; Carotid; Atherosclerotic plaques

1. Introduction

It is an important risk factor that Carotid atherosclerotic plaque for ischemic cerebrovascular disease (ICVD). interleukin-6 (IL -6) is closely related to the instability of atherosclerotic plaques as an inflammatory response factor. IL-1β is widely involved in various pathological processes such as tissue destruction and edema formation that is an inflammatory cytokine. Resistin is a peptide hormone specifically secreted by adipose tissue. Recent studies [1] have shown that it is closely related to other independent risk factors of cerebral infarction, such as hypertension, obesity, blood glucose, dyslipidemia, and insulin resistance (IR), so it is speculated that resistin is correlated with the incidence of cerebral infarction.

It was examined that the carotid artery of elderly patients with acute progressive cerebral infarction by color Doppler ultrasonography, and it was detected that plasma levels of interleukin-6 (IL-6), IL-1β and resistin, so as to explore the correlation between the disease progression and the characteristics of carotid atherosclerotic plaques and the levels of IL-6, IL-1β and resistin in serum in this study.

2. Objects and Methods

2.1. Datum of cases

All the selected patients were admitted to the department of neurology of our hospital for acute cerebral infarction from June 2016 to December 2018, meeting the diagnostic criteria revised by the fourth national conference on cerebrovascular diseases in 1995 [2]. All were found disease that infarcts consistent with functional deficits by CT or MRI examination of the brain within 72 hours. The age of onset is over 60 and time within a week. The severity of the disease continued to worsen after clinical treatment, and the NHISS score decreased by 4 points or more. It was excluded that Patients with previous history of cerebrovascular disease, severe liver and kidney disease, thyroid disease, blood disease and autoimmune disease.

2.2. Groups

All cases were divided into 3 subgroups according to the nature of carotid artery ultrasound plaques: cerebral infarction group (113 cases), age (67.3±9.8) years old, male 68 cases, female 65 cases; No plaque group (20 cases), stable plaque group (42 cases), unstable plaque group (51); The control group (40 cases) included 68 males and 65 females, Age (66.9±9.6). There was no significant difference in age and gender between the above groups (P>0.05).

2.3. Detection indicators and methods

Venous blood was collected in the morning of the next day, and blood lipid and liver function tests were conducted on the same day after all the subjects were routinely fasted for 12h. patient's elbow vein blood 4ml with an empty stomach on the morning of the day after admission. After placing 1h, the blood was treated with 3, 000 RPM and l5min. Take that in supernatant fluid - 70℃ refrigerator under test. The content of IL-6、IL-1β and restin in serum were determined by double antibody sandwich ELISA. The kit was provided by Beijing Qi Sheng biology Co, Ltd. and the enzyme labeling instrument was Finland (Finland) 35200.

2.4. Research methods

Inspection of carotid plaque by color ultrasound, all examined by the professional who is engaged in color ultrasonic examination adopted American SEQUOLA512 color super display instrument. The patient is placed in the supine position or supine with the pillow, with the neck extended and the head slightly tilted to the opposite side. The probe positioned the carotid artery prior to the supravicular fossa, and observed the vascular trend, lumen patency, and wall thickness using two-dimensional methods. The intravascular -- media thickness (IMT) was mainly observed, and it was scanned successively the common carotid artery (CCA) trunk, BIF and ICA. Observe there are plaques in the inner wall of arterial vessels and the intima is smooth whether or not. It observe in detail there are ulcers whether or not, calcifications and thrombosis If there are plaques. All atherosclerotic plaques should be confirmed by longitudinal and transverse two-dimensional methods. Carotid atherosclerosis plaque type ultrasonic credit [3], intimal thickening type or rough type (Ⅰ): 1.0 mm≤IMT≤1.2 mm. Tube cavity atherosclerosis plaque formation type (Ⅱ type): It was determined to be plaque formation include 0.5 mm thicker than the adjacent areas, lack of patube cavity filled with blood flow in area of 10mm or IMT＞1.2 mm the plaques can be divided into soft plaques (the echo in the plaques is lower than or equal to the echo in the vessel wall), hard plaques (stronger than the echo in the lumen, often accompanied by echo shadow in the rear, mostly calcification and fibrosis), and mixed plaques (the echo is not uniform, and there are both strong echo and low echo in the interior of the plaques) according to the echo characteristics of arteriosclerotic plaques. Carotid plaque score (CAS) [4] It was added without calculating its length that the maximum thickness (in mm) of each isolated carotid atherosclerotic plaque on one side, and it was obtained that the score of carotid atherosclerotic plaque on that side. It was the sum of the scores of bilateral plaques that total score of carotid atherosclerotic plaque in patients.

2.5. Statistical processing

SPSS19.0 statistical software was used to process the data. The measurement data are represented by (±S); T test was used in the two groups. In the three groups, one-way analysis of variance was used. P < 0.05 was considered statistically significant.

3. Consequence

Table 1. Comparison of general conditions of each group

General factors	Unstableplaques (n=51)	Stable plaque (n=42)	No plaque (n=20)	Control group (n=40)
Tg(mmol/L)	2.17±0.84	1.92±0.45	1.47±0.28	1.26±0.36
Tc(mmol/L)	4.81±1.23	4.65±0.69	4.29±0.65	3.85±0.49
Ldl(mmol/L)	1.15±0.26	1.05±0.13	1.01±0.16	0.85±0.11
Glucose/(mmol/L)	5.23±1.94	5.20±1.91	5.09±1.89	4.62±1.11
Nhiss/分	24.26±3.15	23.58±3.11	23.71±3.12	

There were significant differences in serum IL-6 IL-1β and resistance levels in the subgroups. It was found that the cerebral infarction group of unstable plaque were significantly higher than the stable plaque cerebral infarction group. The cerebral infarction group of stable plaques was significantly higher than that of the free cerebral infarction group (P<0.01).

Table 1. Comparison of general conditions of each group

Group	Cases	IL-6（pg/mL）	IL-1β （pg/mL）	Resistance（pg/mL）
Unstableplaques	51	331.51+51.18*	200.14±21.35*	16.72±1.64*

Stable plaques	42	225.21+40.12*	184.35±18.84*	14.18±1.26*
No plaques	20	170.48+21.58*	95.62 ±10.65*	13.65±1.02*
Control group	40	87.21+11.36	50.24 ±8.16	10.72±0.31

4. Disscuion

Progressive cerebral infarction is a common type of acute cerebral infarction, in which the symptoms of neurological function loss are mild after the occurrence of cerebral infarction, but progressive aggravation is presented, and the disease continues to progress until the occurrence of more serious neurological function defects. The main changes in atherosclerosis are the focal deposition of lipids, complex carbohydrates and blood components in the endometrium, hyperplasia of smooth muscle cells and collagen fibers, which lead to the formation of lipid striae, which are manifested as endometrium thickening, endometrium damage to a certain extent, and plaque formation. It is a hot topic of scholars that how to intervene the progression of progressive cerebral infarction. We analyzed the main clinical risk factors and discussed the relationship between them and progressive cerebral infarction.

Unstable plaques and progressive cerebral infarction: Carotid arteriosclerosis can reflect cerebral arteriosclerosis as it is a part of systemic arteriosclerosis. Carotid atherosclerosis is an important source of cerebral emboli in ischemic stroke and its progression and it has an effect on cerebral hemodynamics. The progression of ischemic stroke is thought to be largely dependent on the stability of carotid plaque. Arteriosclerotic plaques can be divided into two categories: one is stable plaques, including homogeneous plaques and hard plaques; the other type is unstable plaques, including heterogeneous plaques, soft spots or mixed spots, heterogeneous plaques, vulnerable plaques. The former exists for a long time but does not cause the symptoms of arterial ischemia, while the latter is prone to rupture or morphological changes, resulting in the remote vascular ischemia of plaques and the symptoms of cerebral apoplexy [5-7]. In this study, carotid atherosclerotic plaques were significantly more in the progressive group than in the control group especially unstable plaques (soft plaques and mixed plaques), and which were the main risk factors for the occurrence and progression of stroke.

The relationship between IL-6 and unstable plaques: IL-6 and its receptor are continuously expressed at low concentration in brain tissue, with multiple functions including central immune mediation, nerve repair, coordination of metabolism and neuroendocrine under physiological conditions. RothwellE [8], Cojocaruim [9] et al. pointed out that il-6 concentration was highly expressed within 24h after the occurrence of cerebral infarction, so it could be used as an early warning sign of inflammation. IL-6 can stimulate macrophages to produce mmp-9 and aggregate platelets, increase the expression of glycoprotein and make vascular smooth muscle hyperplasia, so as to accelerate arteriosclerosis and make plaques develop from stable to unstable. This study also found that the level of IL-6 in patients with progressive cerebral infarction was significantly increased compared with the normal control group, and the level of IL -6 and IL-6 in patients with progressive cerebral infarction was increased with the increase of the severity of the disease.

IL-1 β and progressive cerebral infarction: One of the mechanisms of cerebral ischemia reperfusion injury is excessive inflammatory response. IL-1 β is a pro-inflammatory factor secreted by various cells, which promotes blood coagulation, damages the blood-brain barrier, increases the release of excitatory amino acids and free radicals, etc.[10], thus participating in the development of cerebral infarction. Studies have shown that it will lead to aggravation of brain injury which direct injection of IL-1 β into cerebral ventricle or brain tissue of cerebral ischemia rats. Serum IL-1 β concentration was correlated with infarct volume. This indicates that the level of inflammatory response reflects the severity of cerebral infarction.

Resistin and progressive cerebral infarction : Resistin can directly lead to the damage of endothelial function by act on intercellular adhesion molecule 1(ICAM -1), endothelial cells and factors regulating the secretion of endothelin 1(ET -1).Vascular endothelial injury leads to thrombosis and disease progression via the activation and secretion of tissue factor (TF), which initiates the coagulation cascade amplification effect. On the one hand, it promotes platelet aggregation, and at the same time activates various coagulation factors, resulting in increased synthesis of fibrin.

The injury caused by ischemia and the secondary injury caused by reperfusion induce the acute inflammatory reaction after acute cerebral infarction. In addition, the body is in an emergency state and inflammation is an important factor for vascular endothelium damage, which makes it elevated and results in arteriosclerosis plaque rupture and thrombosis. This study found that the levels of IL-1 β, IL-6 and resistin in patients with progressive cerebral infarction were significantly increased compared with the normal control group, and the differences were statistically significant both in the levels of the other two groups and in the unstable plaque group, indicating that the unstable plaques and inflammatory responses in patients with progressive cerebral infarction. To sum up, the occurrence of progressive cerebral infarction is the result of multiple factors and mechanisms. To explore its multiple risk factors provides many intervention points for clinical treatment.

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