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The value of coagulant molecular marker in diagnosis of cerebral infarction after intracerebral hemorrhage.

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Abstract

Objective: To discover and analyze the value of coagulant molecular marker in diagnosis of cerebral infarction after intracerebral hemorrhage.

Methods: In this study, we selected a total of 42 patients with cerebral infarction after intracerebral hemorrhage as the experiment group; additionally, 42 patients with intracerebral hemorrhage were enrolled as the hemorrhage control group; besides, 42 healthy people that were confirmed through relevant examinations were selected as the healthy control group. For patients with intracerebral hemorrhage, before the onset of cerebral infarction, we detected a series of indicators, including plasma Fibrinogen (FIB), Hematocrit (HCT), and plasma Endothelin (ET-1), P-Selectin (P-S), Protein C (PC) and D-Dimer (D-D).

Results: In comparison of FIB and P-S, the values in the experiment group were significantly higher than those in the hemorrhage control group and the healthy control group, and the differences had statistical significance ($p<0.05$). While the values of D-D and ET-1 in the experiment group were significantly higher than those in the healthy control group ($p<0.05$), comparison of HCT showed that there was no statistically significant difference among three groups ($p>0.05$). However, the comparison of PC showed that the value in the experiment group was significantly lower than those in other two groups ($p<0.05$).

Conclusion: Detecting the levels of coagulant molecular markers in patients with intracerebral hemorrhage greatly contributes to the early diagnosis of secondary cerebral infarction and improvement in prognosis, which is worthy being promoted in clinical practice.

Keywords: Cerebral infarction after intracerebral hemorrhage, Coagulant molecular markers, Detection, Value.

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Introduction

Considering the serious threatens of cerebral infarction after intracerebral hemorrhage to the health and safety of patients, developing new indicators is of great significance to the effective prediction and diagnosis of secondary cerebral infarction. To discover the value of coagulant molecular marker in detecting the cerebral infarction after intracerebral hemorrhage, we enrolled 42 patients with cerebral infarction after intracerebral hemorrhage as the experiment group, 42 patient's only intracerebral hemorrhage as the hemorrhage control group, and 42 healthy people as the healthy control group. For those patients, we detected the levels of a series of indicators, including plasma Fibrinogen (FIB), Hematocrit (HCT), and plasma Endothelin (ET-1), P-Selectin (P-S), Protein C (PC) and D-Dimer (D-D). Detailed information of this study is reported as follows.

Materials and Methods

General material

We collected the cases of patients with acute traumatic intracerebral hemorrhage, spontaneous intracerebral hemorrhage or spontaneous subarachnoid hemorrhage who received the treatment in this hospital between January 2015 and May 2017, and all patients underwent the corresponding examinations (head CT or MRI examination) [1,2]. During the treatment in hospitalization, re-examinations through CT or MRI were carried out according to the specific condition of patients. In the experiment group, 42 patients were diagnosed as cerebral infarction after intracerebral hemorrhage in this hospital, in which there were 18 males and 24 females aged between 80 y old and 20 y old, and the average age of patients was 49.6 ± 18.6 y old. In these patients, there were 25 with traumatic intracerebral hemorrhage, 10 with spontaneous intracerebral hemorrhage, and 7 with spontaneous subarachnoid hemorrhage. In the hemorrhage control group, 42

patients were diagnosed as intracerebral hemorrhage through clinical examination in this hospital, in which there were 25 males and 17 females aged between 82 y old and 23 y old, and the average age of patients was 51.5 ± 12.4 y old. In these patients, there were 12 with traumatic intracerebral hemorrhage, and 30 with spontaneous intracerebral hemorrhage. In the healthy control group, 42 healthy people received the examinations in this hospital during the same period, in which there were 20 males and 22 females aged between 60 y old and 20 y old with an average age of 41.2 ± 10.6 y old. In this study, all subjects had been informed of the content of study.

Method

Firstly, samples were collected from the patients. Within 3 d after the onset, 5 mL fasting venous blood was drawn from patients, and the plasma was also collected simultaneously. Then the samples were frozen and preserved at -30°C in an ultra-low temperature freezer for later detection [3,4]. Thereafter, detections were carried out in following procedure, i.e. the double-antibody sandwich ELISA method, in which we detected the indicators of FIB, HCT, ET-1, P-S, PC and D-D [5,6]. Kits used in this study were provided by Westang (Shanghai) BioScience Co., Ltd. and the procedures were in strict accordance with the instructions of kit.

Statistical methods

Statistical Product and Service Solutions 12.0 (SPSS 12.0) was used to analyze and process the data. Count data were presented as (n, %), and chi-square test was carried out for the comparison of count data. The measurement data were presented as ($\bar{x}$ ± s), and t-test was performed for the comparison of measurement data. $p<0.05$ suggested that the difference had statistical significance.

Table 1. Comparison of the general data of patients between the experiment group and the hemorrhage control group.

Groups	Cases	Age (y)	Sex (male/female)	Hemorrhage cases	Glassgow score
Observed group	42	49.6 ± 18.6	18/24	25	6.78 ± 3.42
Hemorrhage control group	42	51.5 ± 12.4	25/17	12	10.69 ± 3.55
P		>0.05	<0.05	<0.05	<0.05

Table 2. Comparison of the relevant indicators between the experiment group and the hemorrhage control group ($\bar{x}$ ± s).

Groups	FIB (g/L)	HCT (g/L)	ET-1 (pg/mL)	P-S (mg/L)	PC (mg/L)	D-D (mg/L)
Observed group (n=42)	3.98 ± 1.52	0.39 ± 0.05	21.25 ± 2.73	22.50 ± 4.06	3.26 ± 1.23	1.51 ± 0.25
Hemorrhage control group (n=42)	2.54 ± 0.65	0.43 ± 0.02	20.36 ± 2.56	17.24 ± 3.68	5.88 ± 1.53	1.62 ± 0.23

Table 3. Comparison of relevant indicators of patients between the experiment group and the healthy control group ($\bar{x}$ ± s).

Groups	FIB (g/L)	HCT (g/L)	ET-1 (pg/mL)	P-S (mg/L)	PC (mg/L)	D-D (mg/L)
Observed group (n=42)	3.98 ± 1.52	0.39 ± 0.05	21.25 ± 2.73	22.50 ± 4.06	3.26 ± 1.23	1.51 ± 0.25
Hemorrhage control group (n=42)	2.43 ± 0.42	0.37 ± 0.11	15.35 ± 2.71	14.95 ± 3.12	6.89 ± 1.67	1.02 ± 0.33

Results

Statistics on general data of patients

As shown in the Table 1, the comparison of age of patients showed that there was no statistically significant difference between the experiment group and the hemorrhage control group ($p>0.05$). As for the gender, a great difference was identified between the experiment group and the hemorrhage control group, and the male patients in the former group were significantly fewer than those in the latter group ($p<0.05$). In comparison of the type of hemorrhage, we found that the incidence rate of traumatic intracerebral hemorrhage in the experiment group was significantly higher than that in the hemorrhage control group ($p<0.05$). In comparison of the severity of condition of patients, i.e. the Glassgow score, we found that the score in the experiment group was significantly lower than that in the hemorrhage control group ($p<0.05$).

Results of experiment in three groups

Statistics of the results are shown as Table 2. In this study, we compared the levels of FIB, HCT, ET-1, P-S, PC and D-D in patients between the experiment group and the hemorrhage control group, and found that the levels of FIB and P-S in the experiment group were significantly higher than those in the hemorrhage control group ($p<0.05$). While there were no statistically significant differences in comparison of HCT, ET-1 and D-D of patients between the two groups ($p>0.05$), as for PC, the level in the experiment group was much lower ($p<0.05$).

The results are listed in Table 3. Compared with the healthy control group, the levels of FIB, ET-1, P-S and D-D in the patients of the experiment group were much higher, and the difference had statistical significance ($p<0.05$). In comparison of HCT, there was no statistically significant difference between the two groups ($p>0.05$), whereas the level of PC in the experiment group was significantly lower than that in the healthy control group ($p<0.05$).

Discussions

Intracerebral hemorrhage, especially in some critical cases, can activate the coagulant system and coagulant system, thereby inducing the intensive contraction of vessels and aggravating the damages to nerve tissues, which usually result in the attack of cerebral infarction [7,8]. The occurrence of cerebral infarction after intracerebral hemorrhage will further exacerbate the condition of patients, and increase the morbidity rate and mortality rate. Clinically, prethrombotic state refers to the imbalance between the pro-coagulant mechanism and natural coagulant mechanism, and the variations in vascular endothelial cells, coagulant system, platelet system and fibrinolytic system, which can contribute to the pathologic condition conducive to the thrombosis [9-11]. Before thrombosis, variations in varying degrees occur in many factors. As for the factors inducing the cerebral infarction after intracerebral hemorrhage, scholars in China and other countries have carried out many studies, and discovered that these factors are mainly correlated with the following aspects [12-15]: (a) Hematoma induced by intracerebral hemorrhage poses direct compression on the vessels; (b) Hematoma gives rise to the edema in peripheral brain tissues and intracerebral hypertension, and leads to the shift of midline structure, resulting in the traction, compression or torsion of contralateral or ipsilateral vessels, which usually causes disorders in local blood circulation frequently seen in infantile cerebral infarction and basal nucleus infarction; (c) Damages to red blood cells also contribute to the disorders in metabolism of degradation product, vasospasm and, finally, cerebral infarction; (d) Direct injuries to the vascular endothelium interrupts the activity of prothrombin, leading to the thrombosis; (e) since the reactions of radicals are initiated and catalyzed in this process, the peroxidation of lipid is damaged, in addition to the imbalanced intracellular environment, induces the formation of microthrombi; (f) Factors like fluid loss, fasting treatment and administration of dehydrator, can increase the blood viscosity and induce the thrombosis.

As one of the vasoconstrictive factors, ET-1 is characterized with the most promising effectiveness in vasoconstriction so far. Research has shown that in the acute phase of intracerebral hemorrhage and cerebral infarction, ET level in the plasma is significantly elevated, and, especially, the secretion of ET is augmented with the increase in bleeding amount. Thus, ET might be involved in the pathological process of intracerebral hemorrhage [16]. In this study, the results showed that the difference on the levels of ET-1 between the experiment group and the healthy control group had statistical significance, but there was no statistically significant difference in comparison of ET-1 level between the experiment group and the hemorrhage control group; P-S, as the specific indicator of platelet activation and the adhesive factor in granulocyte and endothelial cells in ischemic reperfusion, exerts its effect during the activation of inflammatory responses. After the onset of intracerebral hemorrhage, the level of P-S is significantly increased with the augmentation in positive expression, indicating the activation status of platelet; meanwhile, research shows that the level of P-S is significantly elevated in the cerebral infarction patients [17,18]. In this study, the results showed that compared with other two groups, the level of P-S in the experiment group was significantly different. PC, as a kind of key coagulant protein, can directly affect the balance in coagulant mechanism; however, the lack of PC will promote the coagulant activity, and decrease the fibrinolytic activity. In this study, the results suggested that there were statistically significant differences in comparison of PC level in the experiment group with other two groups; D-D is a kind of degradation product of cross-linked fibrin, and the increase of D-D level suggests the secondary augmentation of fibrinolytic activity. FIB, coagulation factor I, can increase the content of fibrinogen during the onset of intracellular hemorrhage, and after the release of tissue coagulation factors in brain tissues, it will also increase the coagulant activity [19,20]. In this study, the results revealed that the FIB level in the experiment group was significantly different from those in other two groups; we also found that there was a statistically significant difference in comparison of D-D level between the experiment group and the healthy control group, but no statistical significance was identified in difference between the experiment group and the hemorrhage control group.

Conclusion

In conclusion, cerebral infarction after intracerebral hemorrhage severely threatens the health and life of patients. Detecting the coagulant molecular marker in patients with intracerebral hemorrhage can better predict the onset of secondary cerebral infarction, and also is conducive to the prophylaxis of secondary cerebral infarction. Thus, it is of great application value and worthy of being promoted in clinical practice.

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