Clinical Effect of Ozagrel Sodium Combined with Eureka on Acute Cerebral Infarction

Shen Shiheng^{1, a}, Yan Shaoxiong^{1, b, *}

¹Zhaotong Health Vocational College 657000

Abstract: Cerebral infarction, as a serious cerebrovascular event, can lead to symptoms of various neurological disorders. The disability rate, mortality rate and recurrence rate are also very high, which is the health and economic burden of individuals, families and even human society. In this paper, 60 patients with cerebral infarction treated in our hospital were divided into observation group and control group. The observation group was treated with Ozagrel sodium combined with Eureka, and the control group was given a single Ozagrel regimen. The results showed that Ozagrel sodium combined with Eureka was effective in the treatment of acute cerebral infarction.

1 Introduction

Although substantial progress has been made in the treatment of patients with cerebral infarction, it is still the main cause of adult death and acquired disability worldwide. Cerebral infarction is divided into ischemic cerebral infarction and hemorrhagic cerebral infarction, in which ischemic cerebral infarction accounts for 70-80% of all cerebral infarction. Therefore, accurate diagnosis and proper management of patients with acute cerebral infarction are very important in saving their lives. Because of the high disability rate, high incidence of cerebral infarction and the younger age of onset with the increase of living standard and the change of diet structure, the early diagnosis, early intervention and the reduction of incidence [1] acute cerebral infarction are particularly important. Ozagrel sodium is one of the commonly used antiplatelet drugs, which has the function of dilating blood vessels and plays an important role in improving circulatory disorders and energy metabolism in patients with acute cerebral thrombosis. As a cerebrovascular and peripheral vascular drug, Eureka can significantly reduce the expansion of cerebral infarction area, improve cerebral hypoxia caused by cerebral infarction, improve glucose metabolism, and have a certain effect on the treatment of cerebral infarction.

Naess studies suggest that elevated plasma homocysteine can promote the pathological process of cardiovascular and cerebrovascular atherosclerosis, and pathological studies show that cardiovascular and cerebrovascular diseases in young people have early manifestations of atherosclerosis, which may be related to dyslipidemia, hypertension, smoking, long-term overload work [2] young people. O'Neill results showed that cardiogenic embolism was the most common cause of ischemic stroke between the ages of 16 and 55, followed

by carotid dissection, atherosclerosis and small vascular occlusion. At the same time, the etiology of young stroke is also related to age stratification [3].

In this paper, the patients with acute cerebral infarction treated in our hospital were selected as the research object, and the patients were divided into observation group and control group. The therapeutic effect of the two schemes was compared.

2 Risk factors and Treatment of Cerebral Infarction

2.1 An Overview of Cerebral Infarction

Although most of the cerebral infarction patients occurred in the population over 65 years old, the epidemiological survey results showed that the incidence of cerebral apoplexy was gradually increasing in the young and middle-aged population.

Currently, the treatment of cerebral infarction mainly focuses on the removal of acute thrombosis or the prevention of thrombosis, which may be of no benefit to a small number of patients with cerebral infarction. Moreover, the data of stroke in children show that all cases of atherosclerosis in children only account for a very small proportion. Therefore, only when clinical trials and clinicians adopt the etiological credit system that includes all the pathogenesis of cerebral infarction, can an effective and accurate treatment plan be formulated, but the correct identification of the risk factors of cerebral infarction is the precursor for diagnosis and prevention.

2.1.1 Early-Onset Atherosclerosis

Dyslipidemia accompanied by vascular endothelial injury is the initial stage of ischemic cardiovascular and

^aZTWXXGC@126.com, ^b13648703686@139.com

cerebrovascular diseases. Several studies have shown that elevated plasma LDL levels can accelerate the progression of atherosclerosis and increase the risk of ischemic vascular events.

2.1.2 *Hyperhomocysteine*

70% of Hcy in plasma is bound to albumin by disulfide bonds, and 30% of Hcy exists in free form, that is, free homocysteine (fHcy). FHcy includes reduced Hcy and disulfide Hcy. Studies have shown that fHcy is strongly correlated with vascular endothelial dysfunction [4]. In addition to B vitamin deficiency, gender, meat diet, hypothyroidism and other diseases and genetic factors can all cause abnormal plasma Hcy level, leading to the occurrence of hyperhomocysteinemia. Folinic acid (in terms of genetic factors, methylene reductase gene loci polymorphism (especially the C677T, A1298G) associated with Hcy concentration and a variety of diseases, observational study results show that the young people MTHFR (C677T gene carriers in the risk of myocardial infarction, ischemic stroke and TIA are carriers of 2-4 times, but also the most common genetic cause of HHcy.

2.1.3 *Cardiogenic Stroke*

Cardiogenic stroke refers to the cerebral artery stenosis and occlusion caused by cardiogenic embolus through systemic circulation, which leads to the dysfunction of the corresponding cerebral blood supply area. It is a type of ischemic stroke in young people with poor prognosis.

2.2 Treatment of Acute Cerebral Infarction

General treatment: respiratory and blood oxygen management: for patients with cerebral infarction with airway dysfunction, auxiliary ventilation; body temperature management: keep body temperature within normal range; blood sugar management: when hypoglycemia occurs, high sugar solution can be given; infection, water and electrolyte disorders and other aspects of management [5].

Antiplatelet aggregation therapy: dual antiplatelet therapy reduces the risk of recurrence of cerebral infarction with single antiplatelet therapy, but also increases the risk of bleeding. A meta analysis study included 103 eligible randomized controlled trials that divided the study into a control group (taking aspirin alone) and an observation group (one of the six aspirin combinations). The results showed that the combination of atorvastatin, Ozagrel sodium, low molecular weight heparin, clopidogrel, cilostazol, ginkgo biloba and aspirin were significantly better than that of aspirin alone ($P < 0.05$) [6]. The combination of aspirin and low molecular weight heparin is the most effective intervention, and the cumulative score curve on its surface is 79.1. The study shows that the combination of aspirin and low molecular weight heparin may be a more effective way to treat cerebral infarction.

Thrombolytic therapy: intravenous thrombolysis was initially used within 3 hours of onset and then extended to 4.5 hours. At present, starting within 4.5 hours after the onset of cerebral infarction symptoms can significantly reduce the disability rate of cerebral infarction. But strict requirements and high risk of bleeding.

Intravascular therapy: endovascular therapy is only suitable for cerebral infarction caused by large vessel obstruction. Stent implantation has some advantages over other mechanical thrombectomy methods and can achieve high recanalization rate. All in all, the application of intravascular therapy can improve the recanalization rate and serum cytokine concentration, improve the quality of life of patients with acute cerebral infarction, and have little [7] effect on the fibrinolytic system of patients. However, intravascular therapy also has some limitations, it has a treatment time window, and the treatment cost is relatively expensive, these factors make the development of intravascular therapy limited.

Other: including neuroprotective agents, improved collateral circulation therapy, traditional Chinese medicine treatment, immunotherapy, cell therapy and other treatments.

At present, there are many ways to treat acute cerebral infarction, but there is still no specific treatment for cerebral infarction. It is necessary to find a specific treatment, low cost and small side effects.

2.3 Risk Factors for Acute Cerebral Infarction

In this study, the general data of cerebral infarction group and control group and the results of blood biochemical examination were compared. There was no significant difference in age, race, diabetes, heart disease, smoking, alcohol consumption, BMI, TC, TG and LDL-C between the two groups. However, there were significant differences between the history of hypertension, HB, WBC, UA and serum ferritin. Age, sex, history of diabetes, history of coronary heart disease, smoking, alcohol consumption, BMI, blood lipids were not different between the two groups, consider the following factors: the study sample size included in this topic is relatively small; the subjects included were from our hospital can not represent the overall level of the entire Chinese population; when asking about the history, some patients failed to clarify the diagnosis of past history. Although some patients in this study had dyslipidemia, but compared to the control group, and there was no difference in serum TC, TG, LDL-C levels, Even the level of blood lipids in cerebral infarction group was lower than that in control group, this result may be that different environmental factors (such as diet, lifestyle) and changes in blood lipids before examination (such as non-fasting blood collection, hypolipidemic drugs used before cerebral infarction) all affect the detection of blood lipids. Both the cerebral infarction group and the control group Hb in the normal range. Hemoglobin is considered to be a risk factor for atherosclerotic cardiovascular disease. Increased hemoglobin increases blood viscosity, associated with atherosclerotic factors, may be an independent risk factor for cerebrovascular events.

Studies have shown that intra-atherosclerotic hemorrhage is a major factor in plaque rupture, During this process Hb iron produced by decomposition can be used as a catalyst for free radical formation, free radicals can modify LDL-c and foam cell [8]. Iron deposition is an important catalyst of reactive oxygen species, which can activate the REDOX state, cause inflammation and lead to unstable arterial plaque. As a result, the increase of Hb in patients with acute cerebral infarction has certain significance. the WBC of leukocytes in cerebral infarction group was higher than that in control group WBC, indicating that inflammation-mediated injury may play a role in cerebral ischemia. elevated WBC and neutrophil counts have similar [9] in predicting long-term cerebrovascular events. Interleukin-6, C reactive protein, and erythrocyte sedimentation rate WBC was found to be positively correlated with neurological dysfunction in patients with acute cerebral infarction in Brazil. these results indicate the [10] significance of inflammation-mediated brain injury after cerebral infarction. WBC may play a role in the occurrence of cerebral infarction.

3 Clinical Trial of Sodium Ozagrel Combined with Eureklin

3.1 Subject Investigated

Sixty patients with acute cerebral infarction treated in our hospital in the past 3 months were divided into control group (single use of Ozagrel sodium) and observation group (combined use of Ozagrel sodium and Eureklin). The general situation of the patients in the group was collected.

3.2 Inclusion Criteria

According to the diagnostic criteria of acute cerebral infarction, eligible patients can be included in the cerebral infarction group: acute onset (not more than 2 weeks); consent and signature of informed consent.

Exclusion criteria: history of intracranial surgery, traumatic fracture, myocardial infarction or symptomatic stroke in the last 3 months; history of major surgery or severe trauma in the last 2 weeks; history of organ bleeding in the last 3 weeks; history of bleeding and blood transfusion in pregnant women, menstrual women, recent history of bleeding and blood transfusion, previous anemia; no patient consent.

3.3 Empirical Method

The control group was treated with Ozagrel sodium for 100 ml, 2 times / d. Intravenous administration of Ozagrel sodium the patients in the observation group were given Eureklin injection 500 mg 0.9% sodium chloride injection 250 ml intravenous drip once a day.

3.4 T Test

The T-test formula used in this paper is as follows:

$$t = \frac{\bar{X} - \mu}{\frac{\sigma_X}{\sqrt{n-1}}} \quad (1)$$

$$t = \frac{\bar{X} - \mu}{\frac{\sigma_X}{\sqrt{n}}} \quad (2)$$

4 Results of the Clinical Trials of Ozagrel and Eureklin

4.1 Comparison of Therapeutic Effects Between Two Groups

Table1. Comparison of treatment effect between the two groups

Group	Cure	Excellent	Effective	Invalid	Effective Rate
Observation group	12	15	2	1	96.67%
Control group	8	12	5	5	83.33%

As shown in Table 1, in the observation group, 12 patients were basically cured, 15 were significantly effective, 2 were effective, 29 were effective, and the total effective rate was 96.67%. In the control group, 8 cases were basically cured, 12 cases were significantly effective, 5 cases were effective, 25 cases were effective, and the total

effective rate was 83.33%. The total effective rate in the observation group was significantly higher than that in the control group ($P < 0.05$).

4.2 Comparison of ADL Scale Scores Between the Two Groups Before and After Treatment

Table2. The scores of ADL scale before and after treatment of the two groups of patients

Group	Before the treatment			After treatment		
	Low value	Medium	High value	Low value	Medium	High value
Observation group	18.67	23.25	27.85	63.25	72.44	81.61
Control group	18.58	23.21	27.79	54.68	61.73	68.86

Figure1. The scores of ADL scale before and after treatment of the two groups of patients

As in Table 2, Figure 1 shows, before treatment, the minimum ADL score in the observation group was 18.67, the maximum is 27.85, and the median value is 23.25. After treatment, the minimum value of the observation group was 63.25, the median value is 72.44, and the maximum value is 81.61; the minimum value in the control group was 54.68, the median is 61.73, and the

maximum value is 68.86. The ADL score of the observation group after treatment was significantly higher than that of the control group ($P < 0.05$).

4.3 Comparison of Adverse Drug Reactions Between Two Groups

Figure2. The occurrence of adverse drug reactions in the two groups

As shown in Figure2, one patient in the observation group had nausea, headache, palpitations and other symptoms, and the incidence of total adverse drug reactions was 13.33% in the observation group. In the control group, nausea was 2 cases, headache 1 case, palpitation 3 cases, and the total incidence of adverse drug reactions was 20. And the total incidence of adverse drug reactions between the two groups was statistically significant ($P < 0.05$).

5 Conclusions

Cerebral infarction, as a serious cerebrovascular event, can lead to symptoms of various neurological disorders. The disability rate, mortality rate and recurrence rate are also very high, which is the health and economic burden of individuals, families and even human society. In this paper, Ozagrel sodium combined with Yuclin was used to treat cerebral infarction. The results showed that the total effective rate in the observation group was significantly

higher than that in the control group ($P<0.05$), and the ADL score in the observation group was significantly higher than that in the control group ($P<0.05$). Eureka combined with Ozagrelina can significantly improve the cure rate of cerebral infarction patients, with high clinical application and popularization value.

Acknowledgment

The successful completion of this thesis is due to my tutor's strict supervision, patient guidance and devoted help. I would like to thank my mentor for his advice and encouragement over the past seven years. Secondly, I would like to thank my Alma mater for giving me the opportunity to study here for seven years, so that I can continue to learn and make progress. Finally, I would like to thank my classmates for their care and help. I hope I can repay the cultivation of my Alma mater and mentor with practical actions in the future.

References

1. Naess H , Nyland H I , Thomassen L , et al. Mild depression in young adults with cerebral infarction at long-term follow-up: a population-based study.[J]. *European Journal of Neurology*, 2015, 12(3):194-198.
2. O'Neill B P , Dinapoli R P , Okazaki H . Cerebral infarction as a result of tumor emboli[J]. *Cancer*, 2015, 60(1):90-95.
3. Naess H , Kurtz M , Thomassen L , et al. Serial NIHSS scores in patients with acute cerebral infarction[J]. *Acta Neurologica Scandinavica*, 2016, 133(6):415-420.
4. Shen B , Liu Q , Gu Y , et al. Efficacy and Safety Evaluation on Arterial Thrombolysis in Treating Acute Cerebral Infarction[J]. *Cell Biochemistry and Biophysics*, 2015, 73(2):297-304.
5. Zhang S , Wang S , Wan X , et al. Clinical evaluation of post-operative cerebral infarction in traumatic epidural haematoma.[J]. *Brain Injury*, 2017, 31(2):215-220.
6. Tun-Pin H , Yu-Chiang H , Wen-Long H U . Impact of acupuncture therapy in postoperative aortic dissection patients with cerebral infarction: A case study[J]. *Chinese Journal of Integrative Medicine*, 2017(01):76-79.
7. Naoi T , Kameda T , Oku K , et al. Internal carotid artery occlusion and cerebral infarction in a case of juvenile systemic lupus erythematosus and positive for phosphatidylserine dependent antiprothrombin antibody (aPS/PT)[J]. *Neurology & Clinical Neuroence*, 2017, 5(2):68-70.
8. Korbakis G , Prabhakaran S , John S , et al. MRI Detection of Cerebral Infarction in Subarachnoid Hemorrhage[J]. *Neurocritical Care*, 2016, 24(3):428-435.
9. Hiroshi S , Shiro Y , Yasutaka M , et al. A case of recanalization of innominate artery and right middle cerebral artery embolism due to cardiogenic cerebral infarction with anticoagulation therapy[J]. *Rinsho Shinkagaku*, 2017, 57(7):391-394.
10. Hua W , Wu H , Zhou M , et al. [Protective effects of recombinant human erythropoietin on oligodendrocyte after cerebral infarction].[J]. *Zhonghua Bing LI Xue Za Zhi*, 2015, 44(5):323-328.