Journal of Clinical and Nursing Research

Research Article

BIO-BYWORD SCIENTIFIC PUBLISHING PTY LTD

Efficacy and Safety of Tirofiban in Thrombolytic Therapy for Ischemic Stroke

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Abstract: ***Objective:*** To evaluate the efficacy and safety of tirofiban hydrochloride in the treatment of ischemic stroke with thrombolytic therapy. ***Method:*** Two hundred patients with acute ischemic stroke thrombolysis were randomly divided into the experimental group and the control group. The experimental group was given tirofiban with the addition of rtpa in the control group and the therapeutic effects of the two groups were compared. ***Results:*** In comparison with the control group, the NIHSS improvement rate was 98% in the experimental group within 14 days. The platelet aggregation rate and efficacy in the experimental group were significantly reduced than the control group ($P < 0.01$). The major adverse reaction in the two groups was hemorrhage with an incidence rate of 3%. ***Conclusion:*** Tirofiban hydrochloride is a highly effective and selective platelet glycoprotein IIb/IIIa receptor inhibitor, which is safe and effective in combination with heparin and aspirin.

Keywords: Acute coronary syndrome, Tirofiban hydrochloride, Receptor, Platelet glycoprotein IIb/IIIa

Publication date: January, 2020

Publication online: 31 January 2020

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1 Introduction

Studies have shown that tirofiban is a selective non-peptide platelet glycoprotein IIb/IIIa receptor antagonist that that blocks specific binding to fibrinogen ligands by binding to platelet surface IIb/IIIa receptors, thereby directly inhibiting platelet aggregation and preventing thrombosis. At present, the safety and efficacy of tirofiban in the treatment of acute coronary syndrome have been verified[1]. The author will summarize the role of tirofiban in acute ischemic stroke as follows.

2 Research object

All patients with progressive stroke in Tianjin Beichen Hospital from January 2016 to February 2018 were diagnosed according to diagnostic criteria: (I) Basic recovery: Functional deficit score decreased by 91% to 100%, and the degree of disability level 0; (II) Significant progress: Functional deficit score reduced by 46% to 90%, and the degree of disability level 1 to 3; (III) Progress: Functional impairment score decreased by 18% to 45%; (IV) No change: Function deficit score 0 to 17%; (V) Deterioration: Functional deficit score increased.

(1)Symptoms and signs of focal nervous system appear on admission;

(2)The symptoms of neurological deficit after onset gradually progressed and increased in a stepwise manner within 36 hours, and the NIHSS score decreased by 2 points or more;

(3)CT examination of head excludes cerebral hemorrhage;

(4)Excluding TIA;

(5)No history of the constitutional hemorrhagic disease;

(6)No history of gastric ulcer in six months and no history of surgery;

(7)Platelet count (PLT), bleeding time (BT) thrombin time (TT), prothrombin time (PT), fibrinogen quantification (FIB) and activated partial thromboplastin time (APTT) were normal;

(8)The patient signs an informed consent form.

3 Research methods

Prior to the study, all patients and control group members agreed to participate in the survey voluntarily, and all data were collected using face-to-face questionnaires. Cerebral stroke type, neurological function, and daily living ability were evaluated by brain CT or MRI, NIHSS score, and Bathel index, respectively. After the questionnaire is withdrawn, it is verified, and the missing part is found and immediately corrected. The returned questionnaire is registered, and the investigation and recovery time are recorded.

4 Observation indicators

(1)Any cause of death within 7 days and 30 days after drug administration, a new myocardial infarction. The criteria are as follows (I) Basic recovery: Functional deficit score reduced by 91% to 100%, disability level 0; (II) Significant progress: Functional deficit score decreased by 46% to 90%, disability level 1 to 3; (III) Progress: Functional impairment score was reduced by 18% to 45% or more; (IV) No change: Functional deficit score 0 to 17%; (V) Deterioration: Functional deficit score increased.

(2)The platelet aggregation rate was measured once before administration and within 24 hours after administration.

(3)Main endpoints: (I) basic recovery; (II) significant progress; (III) progress; (IV) no change; (V) death.

The average treatment time of the experimental group and the control group were (7 ± 0.74) and (30 ± 0.68) days, respectively, and the difference was not statistically significant ($P > 0.05$). The comparison of the primary endpoints of the experimental group and the control group for 7 days and 30 days was shown in Table 1.

Table 1. Comparison of primary endpoints of the experimental group and the control group at 7 days and 30 days

Primary endpoint	The experimental group (100 cases)	The control group (100 cases)
Basic recovery		
7 d	30	33
30 d	34	35
Significant progress		
7 d	60	25
30 d	58	20
No change		
7 d	7	20
30 d	5	22
Deterioration		
7 d	3	20
30 d	2	26
Death		
7 d	0	2
30 d	1	3

The platelet aggregation rate of patients in the experimental group was significantly lower than that of before drug administration [(29.1 ± 26.1)%: (54.0 ± 19.3)%, $P < 0.01$]; there was no significant change in the control group compared before and after drug administration [(57.0 ± 20.4)%: (52.1 ± 19.5)%, $P > 0.05$]; the difference between the two groups was statistically significant ($P < 0.01$). The main adverse reactions in the two groups were mild to moderate hemorrhage which expressed as skin and mucous membrane bleeding. There were 16 cases and 8 cases of bleeding in the experimental group and the control group, respectively. There was no significant difference between the groups.

5 Discussion

With the advancement of interventional techniques for coronary artery disease in recent years, it has been recognized that acute cerebral infarction has common pathogenesis[3]. Thrombosis occurs during atherosclerosis especially in the case of unstable plaque formation. Therefore, the use of effective antithrombotic drugs in the treatment of acute cerebral infarction, especially drugs that have an inhibitory effect on platelet function is critical. At present, the commonly used antiplatelet drugs including aspirin and clopidogrel can only prevent one of the pathways of platelet activation to improve the prognosis of patients. In recent years, great progress has been made in the

study of platelet molecules and cell biology. Among them is the discovery of the coagulation factor I and platelet GPIIb/IIIa receptor binding which are the ultimate common pathways for platelet aggregation. Platelet activation induces conformational changes in GPIIb/IIIa receptors which results in a significant increase in the affinity of the receptor to coagulation factor I, and the bound coagulation factor I can cross-link the platelets and cause platelet aggregation. Therefore, regardless of the cause of thrombosis, platelet activation, adhesion, and aggregation are key steps in the process of arterial thrombosis, in which GPIIb/IIIa receptors play an important role in platelet aggregation and thrombosis. Tirofiban hydrochloride, a highly specific non-peptide platelet GPIIb/IIIa receptor inhibitor which blocks the final pathway leading to platelet aggregation and prevents platelet thrombosis, thereby reducing the incidence of ischemic endpoints. GPIIb/IIIa receptor inhibitors can benefit significantly from early interventional ACS patients, and whether the benefits of patients without interventional therapy are unaffirmed. Recently, many studies at local and abroad have shown that GPIIb/IIIa receptor inhibitors can significantly reduce the mortality and morbidity rate of patients with acute cerebral infarction. Platelet glycoprotein IIb/IIIa receptor inhibitors which block the final pathway leading to platelet aggregation, and the evaluation of its effect mainly based on platelet aggregation function with an ideal suppression rate of about 80%[3]. The results of this study showed that the platelet aggregation rate was significantly lower in the experimental group before and after administration ($P < 0.01$), while the platelet aggregation rate in the control group did not change significantly. It shows that the combined use of tirofiban is safe and effective after thrombolysis.

The main adverse reactions of tirofiban hydrochloride were various types of hemorrhage. The results of Boersma et al showed that the incidence of major bleeding was higher in the GPIIb/IIIa antagonist group than in the control group (2.4%: 1.4%, $P < 0.01$), but intracranial hemorrhage was similar in the two groups (0.09%: 0.06%, $P > 0.05$)[3]. The incidence of adverse reactions (12.7%) in this study was higher than that in the control group (7.0%). Although the difference was not statistically significant, it suggested that tirofiban was used within 24 hours after thrombolysis can increase the risk of bleeding.

In conclusion, tirofiban hydrochloride is a highly effective and selective GPIIb/IIIa receptor inhibitor with a novel mechanism and is safety and clinically efficacy. It can be used immediately after thrombolysis to significantly reduce lethality and disability. It is a very promising therapeutic drug.

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