

# Analysis of the Clinical Effects of Clopidogrel in the Treatment of Unstable Angina and the Influence of Triacylglycerol and Total Cholesterol Levels

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**Abstract:** *Objective:* To explore the clinical value of clopidogrel as the main treatment for patients with unstable angina. *Methods:* From January 2020 to December 2022, we assigned 50 cases to the control group (basic treatment) and 50 cases to the observation group (basic treatment with clopidogrel). The clinical data of both groups of patients were analyzed. *Results:* The total effective rate of the observation group was significantly higher than that of the control group after treatment ( $P < 0.05$ ). Before treatment, there were no significant differences in the duration of angina pectoris and frequency of attacks between the two groups ( $P > 0.05$ ); after treatment, the duration of angina pectoris and frequency of attacks in the observation group were significantly lower than those in the control group ( $P < 0.05$ ). Before treatment, there were no significant differences in hemorheological indices, triacylglycerol (TG), total cholesterol (TC), and platelet aggregation rate between the two groups ( $P > 0.05$ ); however, after treatment, they were significantly lower in the observation group than in the control group ( $P < 0.05$ ). Before treatment, there was no significant difference in the scores of SF-36 between the two groups ( $P > 0.05$ ); after treatment, the SF-36 score of the observation group was significantly higher than that of the control group ( $P < 0.05$ ). *Conclusion:* Clopidogrel can promote the rehabilitation of patients with unstable angina during treatment. Compared with conventional basic treatment, it can reinforce the final therapeutic effect. In addition, our study showed that the duration of angina pectoris and the frequency of attacks were shortened after clopidogrel therapy and the treatment had an effect on improving patients' hemorheological indices, TG, TC, and platelet aggregation rate. Clopidogrel therapy plays an important role in ensuring the quality of life of patients after recovery.

**Keywords:** Unstable angina; Clopidogrel; Triacylglycerol; Total cholesterol

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## 1. Introduction

Unstable angina is a type of acute coronary syndrome common in cardiovascular medicine. According to the severity of symptoms, its clinical manifestation may vary between stable angina and acute myocardial infarction [1,2]. Due to its rapid progression and high incidence, it may transition to acute myocardial infarction, and in severe cases, induce sudden death. Even if the patient survives, their quality of life would be affected [3-6]. It should be noted that unstable angina is reversible and can stabilize and become stable angina. Some literatures have pointed out that in unstable angina, clopidogrel is usually used for treatment. This drug can inhibit platelet aggregation, prevent thrombosis, and minimize the risk of ischemic events, such as ischemic stroke, myocardial infarction, or cardiac death [7,8]. In view of this, we aimed to determine

the clinical value of clopidogrel therapy as the main treatment for patients with unstable angina.

## 2. Materials and methods

### 2.1. General information

From January 2020 to December 2022, we assigned 50 cases to the control group (basic treatment) and 50 cases to the observation group (basic treatment with clopidogrel). The male to female ratio of the control group and the observation group were 25 to 25 and 28 to 22, respectively. The mean age of the patients in the control group was  $63.41 \pm 3.10$  (46–84 years old), while the mean age of those in the observation group was  $63.38 \pm 3.22$  (46–80 years old). There were no significant differences in baseline data between the two groups ( $P > 0.05$ ).

Inclusion criteria: (i) patients who had not received surgical treatment before participating in the study; (ii) patients who understood the significance of the study, cooperated throughout the treatment process, and signed the “Informed Consent”; (iii) patients without active pathological bleeding in the recent period; (iv) patients without major organ dysfunction; (v) patients without abnormal platelet function or coagulation function; (vi) patients without acute myocardial infarction.

Exclusion criteria: (i) patients with communication disorders; (ii) patients with a history of severe allergy to clopidogrel; (iii) patients with malignant tumors, systemic infection, or bleeding tendencies; (iv) patients who left the study midway.

### 2.2. Treatment methods

Patients in the control group received basic treatment, which included oxygen therapy, sedation, myocardial oxygen consumption reduction therapy, *etc.* At the same time, the patients were given enteric-coated aspirin at a dose of 100 mg once a day, Xiaoxintong (with isosorbide dinitrate as the main ingredient) at a dose of 10 mg three times a day, nitroglycerin via a micropump at 10–30  $\mu\text{g}$  per minute, oral calcium channel blocker, simvastatin, angiotensin-converting enzyme inhibitor or  $\beta$ -receptor blocker, and 5000 U of low-molecular-weight heparin calcium (Q12H) via subcutaneous injection. The patients were treated as such for 28 days.

Patients in the observation group received basic treatment with clopidogrel. The basic treatment was the same as that of the control group, followed by an additional dose of 75 mg clopidogrel, QD, for 28 days.

### 2.3. Observation indicators

- (i) Treatment effect: markedly effective, disappearance of symptoms of angina pectoris and normalized levels of TG and TC after treatment; effective, significant improvement in symptoms of angina pectoris and levels of TG and TC after treatment; ineffective, none of the above standards were met. Total effective rate = 100% – total ineffective rate.
- (ii) Duration of angina pectoris and frequency of attacks.
- (iii) Hemorheological indices (plasma viscosity and whole blood viscosity), TG, TC, and platelet aggregation rate.
- (iv) Quality of life: evaluation of quality of life using the SF-36 assessment scale; the higher the score, the better the quality of life.

### 2.4. Statistical analysis

SPSS 21.0 was used for statistical processing. Count data were expressed in percentage (%), the  $\chi^2$  test was used. Measurement data were expressed in mean  $\pm$  standard deviation, and t-test was used.  $P < 0.05$  indicates statistically significant difference.

### 3. Results

#### 3.1. Treatment effect

After treatment, the total effective rate of the observation group was significantly higher than that of the control group ( $P < 0.05$ ). See **Table 1** for details.

**Table 1.** Comparison of treatment effect

Group	Number of cases	Markedly effective	Effective	Ineffective	Total effective rate
Observation group	50	37 (74.0)	11 (22.0)	2 (4.0)	48 (96.0)
Control group	50	24 (48.0)	20 (40.0)	6 (12.0)	44 (88.0)
$\chi^2$		14.208	7.574	4.348	4.348
$P$		0.000	0.006	0.037	0.037

Data are presented in n (%).

#### 3.2. Duration of angina pectoris and frequency of attacks

Before treatment, there were no significant differences in the duration of angina pectoris and frequency of attacks between the two groups ( $P > 0.05$ ). However, after treatment, the duration of angina pectoris and frequency of attacks in the observation group were significantly lower than those in the control group ( $P < 0.05$ ). See **Table 2** for details.

**Table 2.** Comparison of duration of angina pectoris and frequency of attacks

Group	Number of cases	Duration of angina pectoris (min/time)		Frequency of angina attacks (times/week)	
		Before treatment	After treatment	Before treatment	After treatment
Observation group	50	8.87 ± 3.10	4.47 ± 1.88	4.93 ± 1.70	2.19 ± 1.13
Control group	50	8.99 ± 2.91	5.45 ± 2.08	4.81 ± 1.86	2.98 ± 1.78
$t$		0.200	2.472	0.337	2.650
$P$		0.842	0.015	0.737	0.009

Data are presented in mean ± standard deviation.

#### 3.3. Hemorheology indices, triacylglycerol, total cholesterol, and platelet aggregation rate

Before treatment, there were no significant differences in hemorheological indices, TG, TC, and platelet aggregation rate between the two groups ( $P > 0.05$ ). However, after treatment, the hemorheological indices, TG, TC, and platelet aggregation rate of the observation group were significantly lower than those of the control group ( $P < 0.05$ ). See **Table 3** for details.

**Table 3.** Comparison of hemorheology indices, triacylglycerol (TG), total cholesterol (TC), and platelet aggregation rate

Group	Number of cases	Whole blood viscosity (mPa/s)		Plasma viscosity (mPa/s)		TG (mmol/L)	
		Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Observation group	50	9.34 ± 2.45	6.26 ± 1.87	2.99 ± 0.67	1.54 ± 0.55	12.43 ± 1.09	4.36 ± 0.33
Control group	50	9.51 ± 2.58	7.47 ± 1.98	2.86 ± 0.80	2.06 ± 0.60	12.59 ± 1.12	7.78 ± 0.25

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Group	Number of cases	Whole blood viscosity (mPa/s)		Plasma viscosity (mPa/s)		TG (mmol/L)	
		Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
<i>t</i>		0.338	3.142	0.881	4.518	0.724	58.413
<i>P</i>		0.736	0.002	0.381	0.000	0.471	0.000

  

Group	Number of cases	TC (mmol/L)		Erythrocyte aggregation index		Platelet aggregation rate (%)	
		Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Observation group		5.83 ± 1.28	2.29 ± 0.30	1.97 ± 0.33	1.49 ± 0.28	39.46 ± 10.65	26.85 ± 9.33
Control group		5.77 ± 1.35	3.82 ± 1.34	1.94 ± 0.38	1.62 ± 0.30	38.83 ± 10.94	33.57 ± 9.99
<i>t</i>		0.228	7.879	0.422	2.240	0.338	3.476
<i>P</i>		0.820	0.000	0.674	0.027	0.736	0.001

Data are presented in mean ± standard deviation. Abbreviations: TC, total cholesterol; TG, triacylglycerol.

### 3.4. SF-36 score

Before treatment, there was no significant difference in the scores of SF-36 between the two groups ( $P > 0.05$ ). However, after treatment, the observation group had significantly higher scores than the control group ( $P < 0.05$ ). See **Table 4** for details.

**Table 4.** Comparison of SF-36 scores

Group	Number of cases	Mental health		Body pain		Social function		Physiological function	
		Before	After	Before	After	Before	After	Before	After
Observation group	50	81.03 ± 5.03	90.22 ± 8.08	82.24 ± 4.29	93.22 ± 8.08	82.24 ± 8.53	93.53 ± 8.85	82.28 ± 8.05	92.50 ± 10.35
Control group	50	80.02 ± 5.02	80.28 ± 8.05	82.25 ± 4.28	82.23 ± 8.03	82.20 ± 8.52	85.20 ± 8.20	82.25 ± 8.04	85.83 ± 9.08
<i>t</i>		1.005	6.162	0.012	6.822	0.024	4.882	0.019	3.426
<i>P</i>		0.317	0.000	0.991	0.000	0.981	0.000	0.985	0.001

  

Group	Number of cases	Emotional function		Vitality		Physiological function		General health	
		Before	After	Before	After	Before	After	Before	After
Observation group	50	88.00 ± 8.53	95.03 ± 9.43	78.53 ± 8.05	92.89 ± 5.08	75.87 ± 7.80	97.37 ± 8.85	78.54 ± 5.00	90.85 ± 8.00
Control group	50	88.09 ± 8.52	87.48 ± 9.48	78.54 ± 7.07	85.87 ± 5.08	75.87 ± 7.88	88.73 ± 5.08	78.57 ± 5.03	77.85 ± 8.58
<i>t</i>		0.053	3.987	0.007	6.909	0.000	6.014	0.030	7.836
<i>P</i>		0.958	0.000	0.995	0.000	1.000	0.000	0.976	0.000

Data are presented in mean ± standard deviation (points). Before and After refer to before and after treatment, respectively.

## 4. Discussion

Unstable angina is a clinical angina syndrome between stable angina and acute myocardial infarction. In unstable angina, atherosclerosis occurs simultaneously with plaque rupture, while local platelets continue to aggregate and adhere, eventually forming a thrombus<sup>[9,10]</sup>. In addition, vasospasm also occurs, narrowing

or even occluding the vascular cavity, resulting in reduced coronary blood flow, myocardial hypoxia and ischemia, and a continuous increase in acidic metabolites. Intracoronary thrombus is a white thrombus that is rich in platelets and relatively weak in response to fibrinolytic drugs. The coagulation cascade is activated by thrombin and platelets, and the partially occlusive thrombus progresses into a complete occlusive thrombus, which leads to vascular obstruction, induces acute myocardial infarction, and increases the risk of sudden death in severe cases. Therefore, in the treatment of patients with such diseases, the principles of stabilizing the disease, inhibiting the development of the disease, and reducing or preventing the possibility of developing myocardial infarction or death should be adhered to. In recent years, the preferred treatment for patients with unstable angina is active antithrombotic and anti-ischemic therapy <sup>[11,12]</sup>.

In the past, treatment of patients with unstable angina was based on conventional treatment predicated on the expansion of peripheral blood vessels, so that the preload and afterload of the heart can be significantly reduced, the myocardial contractility and rhythm can be improved, and the myocardial oxygen consumption can be reduced as much as possible to ensure sufficient blood supply to the myocardium, thereby relieving myocardial ischemia and angina pectoris <sup>[13,14]</sup>. However, if the patient suffers from coronary atherosclerotic plaque rupture or secondary thrombus, this conventional treatment will be ineffective. As a thienopyridine compound, clopidogrel belongs to a new generation of diphosphate acyl glycoside receptor antagonists. The drug itself does not have antiplatelet activity but mainly forms active antiplatelet substances through the oxidation of cytochrome P450 by the liver enzyme system. The product can selectively and irreversibly bind to adenosine diphosphate (ADP) receptors on the platelet surface to inhibit platelet aggregation and prevent thrombus formation. In addition, the drug can also effectively block the cascade amplification reaction following platelet activation. The inhibitory effect of the drug reaches a stable state after about one week of continuous administration, and the maximum inhibitory effect can be up to three months or even longer. The bleeding time and platelet aggregation return to the baseline five days after the drug is discontinued. The results of the present study showed that the total effective rate of the observation group was significantly higher than that of the control group after treatment; in addition, the duration of angina pectoris and frequency of angina attacks were significantly lower in the observation group than in the control group; the hemorheology indices, TG, TC, and platelet aggregation rate of the observation group were also significantly lower than those of the control group; the SF-36 scores were also observed to be significantly higher in the observation group, compared with control group ( $P < 0.05$ ). The results confirm that the comprehensive intervention with clopidogrel, compared with the conventional treatment plan, can significantly improve the curative effect and various indicators, relieve the degree of angina pectoris, and facilitate the early recovery of patients <sup>[15]</sup>.

In short, clopidogrel should be used in patients with unstable angina to stabilize their condition and reduce the degree of angina. On the basis of ensuring a significant therapeutic effect, clopidogrel can improve the symptoms of unstable angina and the quality of life of patients. However, it should be noted that the antiplatelet mechanism of this drug is very complex. Therefore, further explorations are needed to investigate its long-term therapeutic benefit and ideal period of use in clinical settings.

### **Disclosure statement**

The authors declare no conflict of interest.

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