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Clinical Efficacy Analysis of Rivaroxaban Dosage in Patients with Ventricular Thrombus

Gong Qiang

Department of Cardiology, Baoding NO.1 Central Hospital, Baoding, 071000, China

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Abstract: This study aimed to investigate the efficacy of different dosages of rivaroxaban in the treatment of ventricular thrombus. From February 2023 to February 2025, 86 patients from the Department of Cardiology of our hospital were selected and divided into two groups based on the dosage of rivaroxaban: the low-dose group (10 mg/d) and the standard-dose group (20 mg/d). Post-treatment outcomes, including thrombus area, dissolution rate, bleeding events, cardiac function improvement, and changes in D-dimer levels, were observed. Results indicated that after treatment, the standard-dose group showed superior outcomes in thrombus area reduction ($P < 0.05$), with complete thrombus dissolution and total effective rates of 67.44% and 88.37%, respectively, compared to 44.19% and 74.42% in the low-dose group ($P < 0.05$). However, the incidence of bleeding events was lower in the low-dose group ($P < 0.05$). Additionally, after 3 months of treatment, the standard-dose group exhibited a more pronounced decrease in D-dimer levels ($P < 0.05$). In conclusion, rivaroxaban at a standard dosage of 20 mg/d is more effective than a low dosage of 10 mg/d in reducing thrombus area and D-dimer levels in patients with ventricular thrombus, though it carries an increased risk of mild bleeding. Clinicians should tailor the dosage based on individual patient conditions to optimize therapeutic efficacy and safety.

1. Introduction

Ventricular thrombus is a clinically common and severe pathological event in the progression of cardiovascular diseases, often serving as a serious complication of conditions such as acute myocardial infarction, dilated cardiomyopathy, and heart failure. Once formed, a ventricular thrombus may dislodge, leading to systemic embolism and potentially causing life-threatening consequences like stroke or pulmonary embolism. It also significantly increases the risk of disability and mortality. Therefore, timely and effective anticoagulation therapy is crucial for improving patient prognosis.

Traditionally, vitamin K antagonists (e.g., warfarin) have been used for anticoagulation in such cases. However, these drugs require frequent INR monitoring due to their narrow therapeutic window and complex drug-drug interactions, which limits their clinical application[1]. Rivaroxaban, a novel anticoagulant and highly selective factor Xa inhibitor, promotes thrombin generation and exerts anticoagulant effects without the need for routine coagulation monitoring. Its fewer drug

interactions make it widely applicable in thrombotic diseases.

Notably, there is currently no unified standard for rivaroxaban dosage in treating ventricular thrombus. While guidelines recommend a standard dose of 20 mg/d for stroke prevention in non-valvular atrial fibrillation, whether a lower dose (10 mg/d) is equally effective for ventricular thrombus while reducing bleeding risk remains to be validated through further clinical studies[2]. This study compares the efficacy and safety of rivaroxaban at 10 mg/d versus 20 mg/d for ventricular thrombus treatment, aiming to provide a reference for individualized clinical therapy.

2. Materials and Methods

2.1 General Materials

From February 2023 to February 2025, 86 patients from the Department of Cardiology were enrolled and divided into two groups based on rivaroxaban dosage:

Low-dose group (10 mg/d): 25 males and 18 females, aged 45–78 years (median: 62.35 ± 8.21).

Standard-dose group (20 mg/d): 27 males and 16 females, aged 42–79 years (median: 63.12 ± 7.98).

Baseline characteristics were comparable between groups ($P > 0.05$).

2.1.1 Inclusion Criteria

1) Confirmed ventricular thrombus;
2) Age > 18 years;
3) Cardiac function class II–III;
4) Normal cognitive and communication abilities.

2.1.2 Exclusion Criteria

1) Active bleeding or high bleeding risk within the past 3 months;
2) Allergy to rivaroxaban;
3) Recent thrombolysis or surgical intervention.

2.2 Methods

After enrollment, all patients received basic treatment including blood pressure control, blood glucose management, improvement of myocardial blood supply, and correction of heart failure. On this basis, patients in the low-dose group took rivaroxaban tablets (Bayer Healthcare Co., Ltd., specification: 10mg per tablet) at 10mg/d, while patients in the standard-dose group took rivaroxaban tablets at 20mg/d. Both groups took the medication at the same time daily with meals for 12 consecutive weeks. During the treatment period, patients' symptom changes were closely observed, and regular laboratory tests and imaging evaluations were performed.

2.3 Outcome Measures

1) Changes in thrombus area: Echocardiography was used to measure the area of ventricular thrombus.

2) Thrombus dissolution status: After 3 months of treatment, cardiac ultrasound was performed to evaluate thrombus dissolution, which was categorized as:

- Complete dissolution (thrombus disappeared)
- Partial dissolution (thrombus volume reduction ≥50%)

- No change (thrombus volume reduction <50%)
- Progression (thrombus volume increased)

The complete dissolution rate and total effective rate (complete dissolution + partial dissolution) served as indicators to evaluate thrombus dissolution efficacy[3].

3) Bleeding events: All bleeding events occurring during treatment were recorded, and the bleeding event incidence rate was calculated. Bleeding was defined according to the BARC bleeding criteria.

4) Cardiac function improvement.

5) Plasma D-dimer levels: Measured before treatment and after 3 months of treatment.

2.4 Statistical Analysis

Statistical analysis was performed using SPSS 26.0 software. Continuous variables were expressed as mean ± standard deviation ($\bar{x}\pm s$) and analyzed using t-tests. Categorical data were presented as frequencies and percentages [n (%)] and compared using χ^2 tests. A P-value <0.05 was considered statistically significant.

3. Results

3.1 Changes in Thrombus Area

Before treatment, the thrombus areas showed comparable measurements between groups (P>0.05). After treatment, the standard-dose group demonstrated superior reduction in thrombus area compared to the low-dose group (P<0.05). See Table 1.

Table 1 Changes in Thrombus Area

Group	Before treatment (cm^3)	After treatment (cm^3)
Low dose	2.87±0.65	1.66±0.52
Standard	2.91±0.72	1.01±0.38
T value	0.247	6.341
P value	0.513	0.000

3.2 Thrombus Resolution Outcomes

The complete thrombus resolution rates were 44.19% in the low-dose group versus 67.44% in the standard-dose group, with corresponding total efficacy rates (complete + partial resolution) of 74.42% and 88.37%, respectively. The standard-dose regimen demonstrated statistically superior performance on both endpoints (P<0.05). Detailed results are shown in Table 2.

Table 2 Thrombus Resolution Outcomes (%)

Group	Complete Resolution n (%)	Partial Resolution n (%)	No Change n (%)	Progression n (%)	Total Efficacy Rate (%)
Low dose	19 (44.19)	13 (30.23)	9 (20.93)	2 (4.65)	74.42%
Standard	29 (67.44)	10 (23.26)	3 (6.98)	1 (2.33)	88.37%
χ^2 value	4.715	0.534	3.486	0.345	3.957
P value	0.030	0.465	0.062	0.557	0.047

3.3 Bleeding Events

The low-dose group demonstrated a significantly lower incidence of bleeding events compared

to the standard-dose group (P<0.05). See Table 3.

Table 3 Bleeding Event Incidence (%)

Group	Minor Bleeding n (%)	Major Bleeding n (%)	Total Bleeding Incidence (%)
Low dose	2 (6.98)	0 (0.00)	9.30
Standard	6 (13.95)	2 (4.65)	18.60
χ^2 value			4.074
P value			0.044

3.4 Cardiac Function

After 3 months of treatment, cardiac function improvement (≥1 grade) was observed in:
Low-dose group: 26 cases (60.47%).
Standard-dose group: 28 cases (65.12%)
No significant intergroup difference was observed between groups (χ^2=0.32, P=0.572).

3.5 D-Dimer Levels

Pre-treatment: No significant intergroup difference (P>0.05)

Post-treatment (3 months): The standard-dose group showed significantly greater reduction in D-dimer levels (P<0.05). See Table 4.

Table 4 D-Dimer Levels ($\bar{x}\pm s$)

Group	Pre-treatment (μg/mL)	Post-treatment (μg/mL)
Control	3.25 ±0.68	1.86 ±0.45*
Observation	3.32 ±0.71	1.25 ±0.32*#
T value	0.467	7.244
P value	0.642	0.000

4. Discussion

Intraventricular thrombus refers to a blood clot that forms within the chambers of the heart. It is a common yet severe complication of various cardiovascular diseases. Under normal circumstances, blood within the heart remains in constant motion, making thrombus formation unlikely. However, when certain factors alter the heart's structure and function, abnormal blood flow can occur. This activates the coagulation system, leading to the gradual accumulation of platelets, fibrin, and other components, ultimately resulting in thrombus formation [4].

The development of intraventricular thrombus is primarily associated with the following factors: blood stasis, endothelial injury, and hypercoagulability. During an acute myocardial infarction, the contractile function of the infarcted myocardium is impaired, causing localized slow or even turbulent blood flow, which predisposes to stasis. Additionally, damaged myocardial cells expose subendothelial collagen fibers, activating platelets and clotting factors and initiating the coagulation cascade, thereby increasing the risk of thrombus formation. In patients with dilated cardiomyopathy, the ventricular cavity enlarges, and myocardial contractility weakens, making it difficult for blood to be effectively ejected from the ventricles. This significantly reduces blood flow velocity, creating favorable conditions for thrombus formation. Furthermore, systemic factors such as heart failure, congenital heart disease, prolonged bed rest, malignancies, and certain inherited coagulation disorders can induce a hypercoagulable state, further elevating the risk of intraventricular thrombus.

Once formed, an intraventricular thrombus poses serious risks. The most critical consequence is

thromboembolism, where the clot dislodges and enters the systemic circulation, traveling through blood vessels to various organs and causing embolic events. For instance, if a thrombus reaches the cerebral arteries, it can lead to a stroke; if it lodges in the pulmonary arteries, it results in a pulmonary embolism. Moreover, the presence of an intraventricular thrombus can impair the heart's normal systolic and diastolic functions, exacerbating heart failure and worsening symptoms such as reduced exercise tolerance and dyspnea [5].

Given these risks, the primary treatment goals for such patients should focus on halting thrombus progression, promoting thrombolysis, preventing embolic events, and improving ventricular function [6-7].

Rivaroxaban is a novel oral anticoagulant belonging to the class of direct factor Xa inhibitors. It exhibits high selectivity by inhibiting the activity of factor Xa, thereby blocking both the intrinsic and extrinsic pathways of the coagulation cascade, reducing thrombin generation, and exerting its anticoagulant effect. Additionally, rivaroxaban is rapidly absorbed after oral administration, reaching peak plasma concentrations within 2–4 hours, with high bioavailability and minimal food interactions, allowing for flexible intake with or without meals. Due to its advantages, no need for routine coagulation monitoring, fewer drug interactions, and stable anticoagulant effects, rivaroxaban is widely used in the prevention and treatment of various thromboembolic disorders.

When used in patients with intraventricular thrombus, where cardiac pathology leads to local myocardial injury and hemodynamic changes, vascular endothelial damage occurs, exposing subendothelial collagen fibers. This activates platelets and clotting factors. Factor Xa plays a pivotal role in the coagulation cascade by converting prothrombin to thrombin, which then transforms fibrinogen into fibrin, ultimately forming a thrombus. Rivaroxaban binds with high affinity to both free and clot-bound factor Xa, inhibiting its activity and blocking the conversion of prothrombin to thrombin. This reduces fibrin generation, fundamentally suppressing thrombus formation and progression.

Under normal physiological conditions, the body maintains a dynamic balance between coagulation and fibrinolysis. However, in intraventricular thrombosis, this equilibrium is disrupted, with hypercoagulability and relative insufficiency of the fibrinolytic system. By inhibiting factor Xa, rivaroxaban reduces thrombin generation, thereby decreasing thrombin-induced PAI-1 (plasminogen activator inhibitor-1) release. Lower PAI-1 levels enhance the activity of plasminogen activators, promoting the conversion of plasminogen to plasmin, which dissolves fibrin clots, facilitating thrombus lysis and absorption.

Patients with intraventricular thrombus often exhibit a hypercoagulable state. Rivaroxaban mitigates this by suppressing excessive thrombin generation, thereby reducing coagulation system overactivation and improving blood rheology. It also indirectly inhibits platelet activation and aggregation, lowering thrombotic risk. Furthermore, rivaroxaban provides endothelial protective effects, reducing coagulation system activation due to endothelial injury, helping maintain vascular integrity and function, and further preventing thrombus formation [8].

The clinical dosage of rivaroxaban is primarily determined based on specific indications. The standard dose of 20 mg/day (for patients with creatinine clearance ≥50 mL/min) is approved for the treatment of venous thromboembolism (VTE) and stroke prevention in atrial fibrillation, while the lower dose of 10 mg/day is typically used for perioperative thromboprophylaxis or in patients at high risk of bleeding.

In the treatment of intraventricular thrombosis, dose selection presents greater challenges. A low-dose regimen may lead to inadequate anticoagulation intensity, potentially resulting in incomplete thrombus resolution or recurrence. Although the standard dose can enhance anticoagulation efficacy, it may increase the risk of bleeding, particularly in patients receiving concomitant antiplatelet therapy or those with renal impairment.

This study compared the 10 mg/day and 20 mg/day rivaroxaban regimens, with the following key findings.

(1) Thrombus Reduction Efficacy

The standard-dose group (20 mg/day) demonstrated superior thrombus area reduction, confirming that this regimen provides more potent suppression of coagulation pathways, enhances anticoagulation efficacy, and promotes thrombus dissolution. Additionally, the standard dose more effectively modulates the dynamic balance between coagulation and fibrinolysis.

(2) Dose-Dependent Anticoagulation Effects

The standard-dose group showed significantly higher complete thrombus resolution rates (67.44% vs. 44.19%) and greater D-dimer reduction (1.25 ± 0.32 vs. 1.86 ± 0.45 μg/mL) compared to the low-dose group. These findings align with existing pharmacokinetic-pharmacodynamic studies on rivaroxaban, supporting its concentration-dependent anticoagulant effect. The higher dose achieves more sustained inhibition of factor Xa, further suppressing thrombin generation, thereby more effectively halting thrombus progression and accelerating dissolution. The marked decline in D-dimer levels reflects the standard dose's stronger control over hypercoagulability, consistent with its mechanism in venous thrombosis treatment.

(3) Bleeding Risk and Clinical Strategy

The standard-dose group had a higher incidence of bleeding events (18.60% vs. 9.30%), confirming a positive correlation between anticoagulation intensity and bleeding risk. Notably, all major bleeding episodes (4.65%) occurred in the standard-dose group, predominantly in elderly patients (≥75 years) and those on concomitant antiplatelet therapy. These results highlight the need for individualized risk assessment:

High thrombus burden (e.g., diameter >2 cm, mobile thrombus) + low bleeding risk (HAS-BLED ≤2): Standard dose (20 mg/day) should be prioritized for optimal thrombus clearance.

High bleeding risk (HAS-BLED ≥3) or dual antiplatelet therapy: Low dose (10 mg/day) provides baseline anticoagulation while minimizing life-threatening risks (e.g., intracranial hemorrhage).

(4) Cardiac Function Improvement

Both groups showed similar cardiac function improvement rates (60.47% vs. 65.12%), suggesting that thrombus resolution alone does not solely determine functional recovery. Factors such as myocardial repair, reverse remodeling, and underlying disease progression also play critical roles. Although the standard dose demonstrated better thrombus dissolution within the 12-week observation period, its impact on long-term structural and functional cardiac changes may require further evaluation.

5. Conclusions

In conclusion, for the treatment of intraventricular thrombus, the standard 20 mg/day dose of rivaroxaban demonstrates superior efficacy in reducing thrombus size and lowering D-dimer levels compared to the 10 mg/day low-dose regimen. However, this benefit comes with an increased risk of minor bleeding events. Clinicians should carefully evaluate individual patient characteristics to select the most appropriate rivaroxaban dosage, ensuring optimal therapeutic outcomes while maintaining an acceptable safety profile.

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