Prognosis Effects of Interventional Therapy on Patients with Coronary Slow Flow: An Observational Cohort Study

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Abstract

Background: The phenomenon of slow coronary blood flow is common clinically and is related to the patient experiencing repeated chest distress. The mechanism of slow blood flow is still unclear, and the prognosis after combined interventional therapy is unknown. Therefore, we evaluated the role of interventional therapy in this part of the population through the results of a 2-year follow-up.

Methods: A total of 4663 patients who underwent continuous coronary angiography (CAG) were identified. Those patients with primary slow coronary flow were included in the study (n=369). The population was then divided into the PCI group and the N-PCI group. CTFT is used to assess the severity of slow blood flow. The endpoint events were the occurrence of rehospitalization and out-of-hospital death within 2 years. The log-rank test, Kaplan-Meier method, and Cox regression were used to evaluate and analyze the final results.

Results: A total of 36 patients were readmitted to hospital, and 14 died suddenly outside the hospital during the follow-up period. Among these patients, 6 and 2 patients comprised the PCI group, while 30 and 12 patients comprised the N-PCI group. Comparison of the two groups showed no significant superiority (15.1% vs 13%, $P=0.73$). In Cox regression analysis, high BMI (body mass index) was an independent predictor of adverse end events ($P=0.024$).

Conclusions: Interventional therapy may not improve outcomes in patients with slow coronary blood flow. BMI plays an important role in the influence of prognosis. Further research is needed to investigate this conclusion.

Background

Coronary slow flow phenomenon (CSFP) is a common phenomenon in the process of coronary angiography, with an incidence of approximately 1–7%[1]. Patients usually present with typical angina symptoms and visit the hospital repeatedly. However, coronary angiography showed no significant intimal lesions but only a decrease in contrast agent filling.

The mechanism of CSFP is still unclear and mainly includes[2] the following aspects: microcirculatory dysfunction, endothelial dysfunction, subclinical atherosclerosis, inflammation, and anatomic factors. Pathophysiological inaccuracy leads to no targeted treatment. Previous studies have focused on the search for predictors[3–7] and potential therapeutic approaches[8–10], yielded significant results and received widespread recognition. Nevertheless, current guidelines do not provide clear treatment recommendations and evaluation procedures for this population, especially after percutaneous coronary intervention (PCI). It remains unclear whether interventional therapy can improve outcomes in patients with slow blood flow with coronary atherosclerosis.

Thus, we assessed the effect of interventional therapy on 2-year outcomes in patients with slow blood flow compared with conventional treatment.

Method

Study Population

This is a retrospective cohort study conducted between January 2014 and September 2018. Out of 5663 consecutive patients who underwent coronary angiography in the Hangzhou First People's Hospital Heart Center, 409 patients with CSFP were assessed for eligibility. In total, 369 patients were enrolled in the analysis and divided into two groups according to the experimental design. A flow diagram of the progress through the phases is shown in Fig. 1.

Ethics and consent statements

Since this is a retrospective cohort study, ethical and informed consent is not required.

Definitions

CSFP is defined as primary CSFP[2], which essentially is a delay in contrast injection into the coronary arteries. It is important to identify primary CSFP from delayed progression of contrast agents in coronary reperfusion therapy (such as angioplasty or stent implantation for acute myocardial infarction) or from other secondary causes of coronary slow flow. Conditions associated with secondary coronary slow flow include the following categories: coronary ectasia, coronary spasm, embolism, heart failure, angioplasty and stenting of acute myocardial infarction, valvular heart disease and connective tissue disorders.

Corrected TIMI frame count (CTFC)[11] represents the number of frames required to reach the distal boundary marker of the coronary artery. Not only is CTFC a simple, repeatable, objective, quantitative coronary index, but it also standardizes TIMI grading and facilitates angiography time comparisons between flow tests.

Criteria for inclusion and exclusion

Inclusion criteria

Patients with primary CSFP

Patients who underwent interventional therapy showed CSFP on coronary angiography after 1 month

Exclusion criteria

Previous myocardial infarction or interventional therapy

Heart pump failure

Severe liver or kidney failure

Life expectancy of less than 5 years

Preoperative examination

Venous blood samples are extracted within 24 hours of admission. White blood cells, red blood cells, platelets, total cholesterol, low-density lipoprotein, high-density lipoprotein, C-reactive protein, creatinine, uric acid, admission blood glucose, glycosylated hemoglobin, electrocardiogram and echocardiography must be completed and reviewed prior to coronary angiography.

Coronary angiography and evaluation of coronary flow velocity

Selective coronary angiography was performed by two experienced interventional cardiologists using a standard Judkins' technique. When evaluating the results of coronary angiography, the left anterior descending (LAD) and the left circumflex (LCX) should be projected in at least four angles; the right coronary artery (RCA), in at least two angles. The first frame used to evaluate each TFC is when the dye enters the artery completely, and the last frame is when the dye enters the distal marker branch. In our study, the filming speed was 15 frames per second. The calculation of CTFC was performed by an observer who was blinded to the clinical details. The standard numbers of CTFC frames are defined as 36.2 ± 2.6 frames for LAD, 22.2 ± 4.1 frames for LCX, and 20.4 ± 3.0 frames for RCA. Researchers with TFC of any of the three vessels greater than twice the standard deviation of the normal published range were defined as patients with slow coronary blood flow.

Endpoint

The mean follow-up time for all patients was 24 months. Data related to clinical outcomes were obtained mainly through telephone follow-up and partly by reviewing outpatient records. The endpoints of the study included cardiovascular events requiring hospitalization or death. The former includes the occurrence of ACS, heart failure requiring hospitalization, and angina requiring revascularization[12]. All deaths are considered cardiogenic unless an unequivocal noncardiac cause was established.

Statistics

Before statistical analysis, all continuous variables were plotted and tested for normality and homogeneity of variance. Depending on the results, continuous variables are expressed as the means $\pm$ standard deviations (or medians and interquartile ranges). Categorical variables are expressed in terms of frequencies and percentages. Differences were compared between the two groups. Continuous data were compared by the T test or the Mann-Whitney U test (according to data characteristics), and categorical variables were tested by the chi-square test and Fisher's exact test.

To compare the relationships between prognosis and factors, log-rank tests and Cox regression methods were performed. Log-rank tests are accurate based on hypergeometric probability. Time-to-event curves (i.e., age, treatment, diabetes, smoking and BMI) were evaluated by the Kaplan-Meier method. To identify independent risk factors for prognosis, a single-factor regression analysis was conducted on independent variables one by one, and corresponding p-values were recorded. Independent variables with p values less than 0.1 were incorporated into the final regression equation to identify clinical predictors.

Receiver operating characteristic (ROC) curves were constructed for the prediction of endpoint events. Different prediction parameters were analyzed by the logistic regression model, and the prediction probability was calculated for ROC analysis. The area under the curve (AUC) and the sensitivity and specificity of predicting RP were calculated. AUC = 1.00 indicates the highest accuracy, while AUC = 0.50 indicates no accuracy. A p-value of < 0.05 (2-sided) was considered statistically significant. All statistical analyses were carried out using SPSS software (version 23.0).

Results

Baseline Characteristics

During an average of 24 months of follow-up, a total of 369 people were included in the analysis, with 53 in the PCI group and 316 in the N-PCI group. The average age of the patients was 67 (56–78). The majority of the population was male (67.2%), most of the population's BMI levels indicated that they were overweight (25.03 ± 3.62), approximately half of the patients had a long-term history of smoking (45%), and the overall cardiac ejection function was 65.1 (60.7–68.8). Patients in the N-PCI group were younger (67.7 ± 11.0 vs 63.0 ± 11.1 , $p = 0.004$) but had higher levels of LDL ($1.62(1.35-2.00)$ vs $2.22(1.67-2.71)$, $p = 0.0001$) and TC ($3.28(2.86-3.71)$ vs $4.04(3.36-4.61)$, $p = 0.0001$) than those in the PCI group. No significant differences in other eigenvalues were found. The baseline characteristics of the patients are listed in Table 1.

Coronary angiography and medication

In this study, approximately half of the patients had slower blood flow in more than one vessel (50.4%), and most patients had only mild arteriosclerosis or even smooth intima (71.8%). Compared with the N-PCI group, the PCI group had higher use of antiplatelet agents, statins, and B-blockers, but there was no significant difference in the use of nicordil, and even lower use of nondihydropyridine calcium antagonists than the N-PCI group ($1(1.9\%)$ vs $41(13.0\%)$, $p = 0.019$) (Table 2).

Endpoint

The overall prognosis for patients with slow blood flow was good, with the vast majority remaining in the outpatient follow-up; only 36 patients were rehospitalized. However, 14 patients died suddenly outside the

hospital (Table 3). The Kaplan-Meier curves of various influencing factors for patients are shown in Fig. 2. Compared with the N-PCI group, the PCI group did not show an advantage on the prognosis, and there was no statistically significant difference ($p = 0.73$). Among the factors associated with the occurrence of slow blood flow, diabetes and smoking were not associated with the occurrence of events. Only age showed a significant difference, while BMI showed a trend of significant difference.

ROC analysis and prediction of endpoint

We constructed the ROC curve to explore the possibility of predicting the occurrence of terminal events through the area under the curve. No strong predictors were found (AUC of 0.661, AGE + BMI, $P = 0.042$) (AUC of 0.683, TC + BMI, $P = 0.039$), even though we used joint diagnoses to increase the efficacy of the diagnosis (Fig. 3). Through a single-factor regression analysis, we extracted the following potential variables: age, BMI, Lvids, FS, EF, Hb, Plt, LDL, HDL, CRP, TC, and Scr. Combined with clinical factors, smoking and sex were added. BMI was found to be an independent risk factor for endpoint events.

Discussion

The main findings of our study are as follows: 1. Interventional therapy did not show an advantage over drug therapy during the two-year observation period. 2. BMI was an independent risk factor for endpoint events in patients with slow blood flow. 3. Patients with slow coronary blood flow may be at potential risk of sudden death.

Prevalence of CSFP

Compared with previous epidemiological findings[1], the CSFP incidence in our heart center was quite common, at approximately 8%. At the same time, the slow flow in our center usually involved more than one vessel.

It is not unusual for different populations and different studies to have slight fluctuations in the incidence of CSFP, and there are some significant differences in our study that may be due to the following two factors. First, the epidemiological data included the normal population, and the inclusion of coronary angiography in our heart center was more rigorous. Second, because of the differences in inclusion criteria of research design, our research subjects were often at higher risk. According to the pathophysiology of small vessels in CSFP, slow coronary blood flow may be an early manifestation of atherosclerosis[13, 14], which is associated with more risk factors due to the presence of fixed stenosis in our enrolled population. Our reasonable conjecture is that it is due to the mapping of the atherosclerotic conditions to the microcirculation, which is manifested as diffuse small vessel lesions, so that the angiography is more likely to show multiple branches of slow blood flow. Of course, these bold conjectures need to be confirmed by further basic research.

Outcome

Based on 2 years of follow-up, we did not find a strong association between interventional therapy and a reduction in adverse events, suggesting that we should not be too aggressive when patients with slow blood flow are combined with critical lesions and should use intravascular ultrasound (IVUS) and fractional flow reserve (FFR) comprehensive judgment to maximize the benefit to patients. This strategy can also avoid the adverse reactions and poor compliance problems caused by patients taking many drugs. From a pathophysiological point of view[15, 16], stents can solve the problem of large vessels, while the fundamental problem of microcirculation is not improved.

Previous studies[14, 17, 18] have shown that obesity is an important component in the development of slow blood flow. Obesity will lead to insulin resistance, produce a large number of adipocytokines, and then induce the production of reactive oxygen and lead to the breakdown of endogenous vasodilator nitric oxide, resulting in the imbalance between endothelial systolic and diastolic substances and ultimately leading to endothelial dysfunction. Studies have shown that adiponectin is secreted by adipose tissue and that plasma adiponectin levels are negatively correlated with obesity. Adiponectin[18] protects the body through anti-inflammatory and anti-atherosclerosis mechanisms, and low levels of adiponectin[19] are associated with cardiovascular events. The conclusion of this paper is a good proof of this view. Lower BMI has been shown to improve markers of endothelial dysfunction[20], and further randomized controlled trials are needed to demonstrate clinical efficacy.

Current studies suggest that slow blood flow may lead to sudden death due to malignant arrhythmias by affecting the length of QT[21] in the electrocardiogram or by inducing Brugada syndrome[22]. Slow blood flow should not be defined only as a functional diagnosis because the majority of patients have a relatively good prognosis[23]. Long-term and chronic management of these patients should be taken seriously, especially as the use of B blockers, which can improve symptoms and reduce the risk of sudden death, may be a good option.

Study Limitations

This study has the following deficiencies. First, the nature of this study is a retrospective cohort study, and the efficacy of the conclusions is insufficient; therefore, a multicenter prospective randomized controlled trial is needed to further confirm the conclusions. Second, the outcome events in the follow-up events of this study were relatively insufficient, and further extension of follow-up events and an increased number of samples are needed to obtain higher positive results. Third, after two years of follow-up, we did not reassess cardiac function or perform coronary angiography in all patients; thus, it cannot be ruled out that in some patients, slow coronary blood flow has been alleviated. Finally, the low proportion of patients using nondihydropyridine calcium antagonists and nicordil in the population may influence the results to a certain extent, resulting in a certain deviation.

Conclusion

Interventional therapy may not improve the prognosis of patients with slow coronary blood flow, and BMI is an independent predictor of readmission and sudden death in patients with CSFP. Further randomized controlled studies are needed to determine the mechanisms of these relationships and to determine whether treatment for slow coronary flow improves outcomes.

Abbreviations

Coronary slow flow phenomenon (CSFP)

percutaneous coronary intervention (PCI)

Corrected TIMI frame count (CTFC)

Left anterior descending (LAD)

Left circumflex (LCX)

Right coronary artery (RCA)

Receiver operating characteristic (ROC)

intravascular ultrasound (IVUS)

fractional flow reserve (FFR)

Declarations

Ethics approval and consent to participate:

Not applicable

Consent for publication:

Not applicable

Availability of data and materials:

All data that support the findings of this study are included in this published article [and its supplementary information files]. The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests:

We declare that we do not have any commercial or associative-interest that represents a conflict of interest in connection with the work submitted.

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Authors' contributions:

JYH provided the general direction of the thesis, while QXG mainly completed data extraction, statistical analysis and article writing. YH, GXT, YHG, YS and SSM provide their own Suggestions for revision when writing is completed. All authors read and approved the final version of the manuscript.

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Tables

Table 1

Table 1 Population Characteristics				
	Overall	Pci	N-Pci	P
	n=369	n=53	n=316	
Demographic data				
Age, yrs	67.6±11.2	67.7±11.0	63.0±11.1	0.004
Sex Male	248 (67.2%)	39 (73.6%)	209 (66.1%)	0.285
Female	121 (32.8%)	14 (26.4%)	107 (33.9%)	
BMI, kg/m ²	25.03±3.62	24.66±3.39	25.10±3.65	0.419
Hypertension	221 (59.9%)	30 (56.6%)	191 (60.4%)	0.598
Diabetes	80 (21.7%)	14 (26.4%)	66 (20.9%)	0.366
Hyperlipidemia	54 (14.8%)	4 (7.5%)	50 (15.8%)	0.115
Smoking	166 (45.0%)	23 (43.4%)	143 (45.3%)	0.801
Alcohol	184 (49.9%)	26 (49.1%)	158 (50%)	0.899
WBC	5.9 (5.0-6.9)	5.4 (4.6-6.2)	6.0 (5.2-7.0)	0.012
HB, g/l	139±15	136±13	139±15	0.111
PLT	183 (152-215)	176 (140-208)	185 (153-216)	0.188
LDL, mmol/l	2.11 (1.59-2.61)	1.62 (1.35-2.00)	2.22 (1.67-2.71)	0.0001
HDL, mmol/l	1.07 (0.91-1.26)	1.09 (0.95-1.27)	1.07 (0.90-1.25)	0.735
CRP, mg/l	3.40 (2.10-6.00)	4.00 (2.75-7.00)	3.25 (2.0-6.0)	0.227
GLU, mmol/l	5.08 (4.63-5.92)	5.35 (4.65-6.35)	5.06 (4.63-5.84)	0.276
TC, mmol/l	3.91 (3.25-4.48)	3.28 (2.86-3.71)	4.04 (3.36-4.61)	0.0001
UA, umol/l	346 (290-395)	363 (279-407)	344 (292-5.25)	0.528
SCR, umol/l	83 (75-91)	86 (72-97)	83 (76-90)	0.325
HbA1C	5.75 (5.40-6.18)	5.90 (5.54-6.29)	5.70 (5.40-6.10)	0.062
Echocardiographic				
LVIDd, cm	4.92 (4.63-5.20)	4.92 (4.71-5.25)	4.92 (4.62-5.19)	0.622
LVIDs, cm	3.14 (2.88-3.39)	3.15 (2.87-3.42)	3.13 (2.89-3.39)	0.459
FS	35.8 (32.6-38.5)	35.9 (32.1-38.8)	35.8 (32.6-38.5)	0.7
EF	65.1 (60.7-68.8)	65.1 (60.2-68.9)	65.1 (60.7-68.7)	0.671

BMI: Body mass index, WBC: white blood cells, HB: Hemoglobin, PLT: platelet, LDL: Low density lipoprotein, HDL: High-density lipoprotein, CRP: C-Reactive Protein, GLU: Blood glucose, TC: Total cholesterol, UA: Uric acid, SCR: Serum creatinine, LVIDs: Left ventricular end-systolic dimension, LVIDd: Left ventricular end-diastolic dimension, FS: fractional shortening, EF: Ejection fraction.

Table 2

Table 2				
	Overall	Pci	N-Pci	P
	n= 369	n=53	n=316	
Coronary angiography				
Slow blood vessel				0.13
LAD	99 (26.8%)	20 (37.7%)	79 (25.0%)	
LCX	6(1.6%)	2 (3.8%)	4 (1.3%)	
RCA	78 (21.1%)	8 (15.1%)	70 (22.2%)	
M ultiple vessel	186 (50.4%)	23 (43.4%)	163 (51.6%)	
Cu brit artery	176 (47.7%)			0.064
No lesions	193 (52.3%)	21 (39.6%)	172 (54.4%)	
LAD	95 (25.7%)	12 (22.6%)	83 (26.3%)	
LCX	16 (4.3%)	5 (9.4%)	11 (3.5%)	
RCA	19 (5.1%)	5 (9.4%)	14 (4.4%)	
M ultiple vessel	46 (12.5%)	10 (18.9%)	38 (11.4%)	
Degree of the lesion				0.008
<30	193 (52.3%)	21(39.6%)	172 (54.4%)	
30-50	72 (19.5%)	11(20.8%)	61 (19.3%)	
50-70	75 (20.3%)	10 (18.9%)	65 (20.6%)	
>70	29 (7.9%)	11(20.8%)	18 (5.7%)	
Drug use				
Anti platelet	305 (82.7%)	51(96.2%)	254(80.4%)	0.005
N itrates	125 (33.9%)	26(49.1%)	99 (31.3%)	0.012
Dihyridin type of calcium antagonists	67 (18.2%)	11(20.8%)	56 (17.7%)	0.596
Non-dihydropyridine calcium antagonists	42 (11.4%)	1(1.9%)	41 (13.0%)	0.019
Beta-blocker	243 (65.9%)	48(90.6%)	195 (61.7%)	0.0001
N icorandil	44 (11.9%)	7(13.2%)	37 (11.7%)	0.755
ACEI/ARB	183 (49.6%)	31(58.5%)	152 (48.1%)	0.162

LAD: Left anterior descending artery, LCX: left circumflex artery, RCA: Right coronary artery, ACEI: Angiotensin converting enzyme inhibitor, ARB: Angiotensin receptor blockers.

Table 3

Table 3 Endpoints			
	Overall	Pci	N-Pci
	n= 369	n=53	n=316
Number			
Readmission	36 (9.8%)	6(11.3%)	30 (9.5%)
Sudden death	14 (3.8%)	2(3.8%)	12 (3.8%)
Follow-Up Clinic	321 (87.0%)	45(84.9%)	275 (87.0%)

Figures

Figure 1

Study Flow Chart

Figure 2

Kaplan-Meier survival for patients groups. The cutoff values for Figures a, b, c, d and e were whether the patient was older than 75 years, had PCI surgery, had A BMI greater than 30, had diabetes, and smoked, respectively. In figure a, there was a significant statistical difference. Figure c, d, and e show a trend of significant differences.

Figure 3

Receiver operating characteristic (ROC) curves of (AGE+BMI) and (TC+BMI). In spite of the combined approach, the AUC values did not achieve statistically significant predictive differences.