


Risk Factors of Cardiac Death for Elderly Patients with Severe Chronic Kidney Disease after Percutaneous Coronary Intervention

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Abstract

Aims: To identify risk factors for cardiac death of elderly and severe chronic kidney disease (CKD) patients with coronary atherosclerotic heart disease (CAHD) after percutaneous coronary intervention (PCI). **Methods:** 1010 CAHD-CKD patients over 60 years old who had CKD stage 3 or above and underwent PCI were followed up for at least 3 years. Cases of cardiac death were divided into groups. After univariate analysis of all variables, the variables with $P < .2$ were selected for further logistic regression. **Results:** For logistic regression single-vessel disease (SVD) PCI OR = 0.612, 95%CI: 0.416–0.899, $P = .012$, it is the protective factor. There are four risk factors, stable angina pectoris (SAP) OR = 4.723, 95%CI: 1.098~20.322, $P = .037$, combined with lower extremity arteriosclerosis obliterans (LEASO) OR = 2.631, 95%CI: 1.272~5.440, $P = .009$, $K > 4.285$ mmol/L OR = 1.44, 95%CI: 1.002~2.069, $P = .049$, without statins OR = 2.015, 95%CI: 1.072~3.789, $P = .030$. **Conclusion:** In elderly and serious CAHD-CKD patients after PCI, SVD PCI was a protective factor against cardiac death. However, SAP, CAHD-CKD combined with LEASO, $K > 4.285$ mmol/L, and no statins were independent risk factors of cardiac death for elderly patients with severe CKD after PCI.

Keywords

chronic kidney disease, coronary atherosclerotic heart disease, risk factors, prognosis

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Introduction

Coronary atherosclerotic heart disease (CAHD) and chronic kidney disease (CKD) are risk factors for each other.¹ CKD patients are more easily complicated with CAHD,² for example, more than 50% of patients with severe CKD are complicated with CAHD. Meanwhile the proportion of life risk due to CAHD is significantly higher than the aggravation and progression of CKD itself,² elderly CKD patients are 5-10 times more likely to die of cardiovascular disease than to develop end-stage renal disease (ESRD). In addition to the traditional risk factors of atherosclerosis, patients with CAHD and CKD are also exposed to uremia related risk factors such as inflammation, oxidative stress, calcium and phosphorus metabolism disorder. Due to the severity and complexity of coronary artery disease, the drug treatment of CAHD patients with CKD was affected by renal function to varying degrees, which caused restrictions and obstacles to the treatment, especially after coronary intervention, and the overall prognosis was poor.

Especially in elderly patients, age is an established risk factor for both CKD and CAHD patients, and the proportion of patients with CKD and CAHD is higher in the elderly population. Therefore, it is of clinical significance to understand the clinical features and risk factors for cardiac death of elderly

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and severe CKD patients with CAHD after percutaneous coronary intervention (PCI).

Methods

Patient Selection

This was a retrospective analysis of patients hospitalized in Beijing Anzhen Hospital from January 1, 2008, to December 31, 2017. A total of 1010 CAHD-CKD patients over 60 years of age who had CKD stage 3 or above and underwent percutaneous coronary intervention (PCI) as a revascularization method. Hydration therapy was performed 12 h before PCI to 24 h after PCI, and 0.9% sodium chloride 0.5-1 ml/kg.h was injected intravenously. All the patients were followed up for at least 3 years. The medical records of patients included in the study were counted and analyzed. The inclusion criteria were: patient over 60 years old had CKD stage 3 or above and underwent PCI. The exclusion criteria were: Non-Asian, under 60 years of age, CKD stage 1 or 2, acute renal injury or recovery of renal function, infectious diseases, malignant tumors, severe heart diseases (eg, aortic dissection and hypertrophic cardiomyopathy), severe systemic disease, systemic inflammatory disorders, glucocorticosteroid therapies within 2 months, failed PCI or change to coronary artery bypass grafting (CABG). PCI was defined as balloon dilatation and drug-eluting stent implantation after coronary angiography. And "failed PCI" was defined as failure to stent implantation and balloon dilation, or percutaneous coronary balloon dilation only. The flowchart outlining the study is shown in Figure 1.

Definition of CKD

Patients with a history of above-normal creatinine for at least 3 months or with a definite diagnosis of CKD were included, except for those with a gradual decline in creatinine levels that returned to normal during hospitalization. The estimated glomerular filtration rate (eGFR) was calculated by the Cockcroft-Gault Formula $18 [(140-\text{age}) \times \text{body weight (kg)} / \text{serum creatinine (mg/dL)} \times 72]$ ($\times 0.85$ in women). Participants with $\text{eGFR} < 60$ (ml/min.1.73 m²) were defined as CKD stage 3 according to the classification of the National Kidney Foundation.

Data Extraction and Quality Assessment

Clinical data were collected on patients' general status, such as age, sex, height, and weight, family history of coronary heart disease, and risk factors or complications of atherosclerosis, such as hypertension, diabetes, dyslipidemia, cerebral infarction, and history of lower extremity arteriosclerosis obliterans (LEASO) defined as patient had symptoms of intermittent claudication or resting pain in the lower limbs, and the arterial pulse of the lower limb arteries was weakened or no pulse. Lower extremity arterial ultrasound showed that the blood flow signal weakened or disappeared, and ankle brachial index (ABI) was < 0.8 . History of revascularization defined as

previous PCI or CABG. Acute kidney injury (AKI) defined as serum creatinine was 25% higher than baseline or the absolute value was higher than 44 $\mu\text{mol/L}$ within 48 hours after coronary angiography. Important laboratory examinations during hospitalization mainly included routine blood examination, liver and kidney function, blood lipid level, ion detection, coronary artery impact data and information about interventional diagnosis and treatment, including lesion characteristics, PCI target vessels, dosage of contrast agent and postoperative effect. An Excel database was established, collating these data. The situation and time of cardiac death were recorded with telephone follow-up as the main form, supplemented by the inquiries of outpatient and inpatient medical records in Anzhen Hospital.

Statistical Analysis

All data were statistically analyzed using SPSS 26.0 for Windows (IBM Corp.), Stata 15.0 (Stata Corp.) All continuous variables were tested for normality using the Kolmogorov-Smirnov method. Normally distributed continuous variables are expressed as the mean \pm standard deviation, nonnormally distributed variables are expressed as the median and interquartile range, and categorical variables are expressed as the number and percentage. Statistical significance was defined as $P < .05$ with two-sided tests. The patients were divided into groups according to whether they died or not and whether there were differences between the two groups. The measurement data with a normal distribution were compared between the two groups by Student's t-test, the measurement data with a skewed distribution were compared by the Mann-Whitney U-test, and the qualitative data were compared by the chi-square test and Fisher's exact test. After univariate analysis of all variables, the variables with $P < .2$ were selected for further logistic regression, and the forward LR method was used to gradually fit the model. Both were bilateral tests, and at $P \leq .05$, the difference was considered statistically significant.

Results

Different Clinical Features Between the two Groups

A total of 1010 patients with CAHD-CKD met the study criteria, and 142 patients had cardiac during at least 3-12 years of follow-up. The two groups were compared with age, sex, body mass index (BMI), length of stay, renal function, family history, smoking history, history of revascularization, contrast media dosage, AKI proportion of patients with hypertension, diabetes, blood lipid abnormalities, heart failure and cerebral infarction, heart rate and Grace score at admission, and none were significantly different. There were no differences in leukocyte count, erythrocyte count, platelet count, liver function, uric acid and blood lipids, myocardial enzymes and ions, double antiplatelet drug treatment and perioperative use of parenteral anticoagulants and II_bIII_a receptor antagonists, beta-blockers, renin-angiotensin-aldosterone system inhibitors (RAASI)

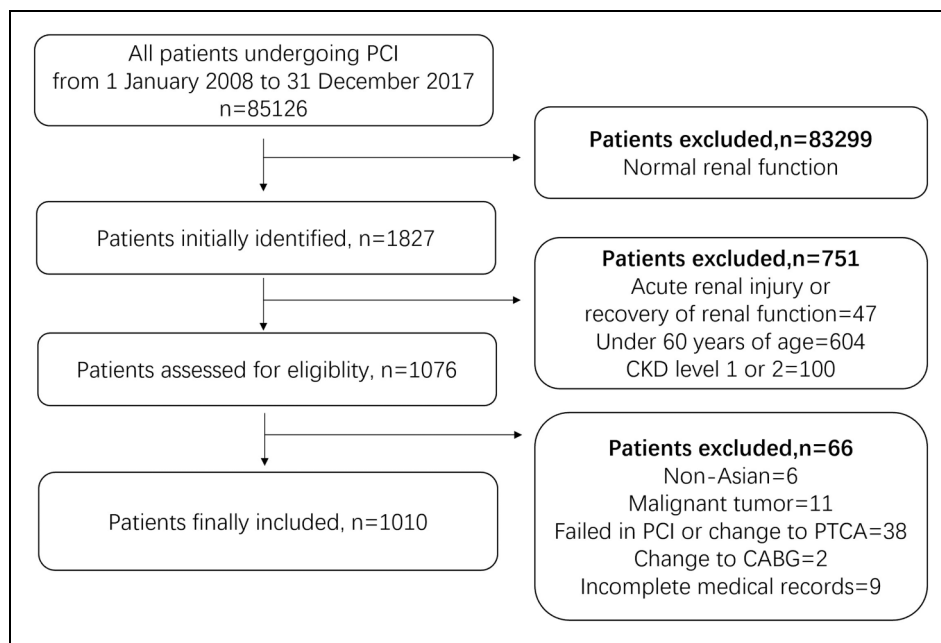


Figure 1. Flow chart of patient selection. PCI, percutaneous coronary intervention; CKD, chronic kidney disease; PTCA, percutaneous transluminal coronary angioplasty; CABG, coronary artery bypass grafting.

calcium channel blockers (CCBs) and nitrates, coronary artery lesion characteristics or surgical approach.

The proportions of patients with systolic blood pressure <90 mmHg and diastolic blood pressure <60 mmHg at admission were higher (5 [3.50%] vs 11 [1.30%]) and (9 [6.30%] vs 27 [3.10%]), the proportion of patients with lower extremity arteriosclerosis obliterans (LEASO) was increased (11 [7.70%] vs 29 [3.30%]), and the proportion of patients receiving single-vessel interventional therapy was decreased (94 [66.20%] vs 656 [75.60%]) in the mortality group compared with the non-mortality group. The above differences were statistically significant ($P < .05$) (Table 1).

Logistic Regression of Cardiac Death

The variables with $P < .2$ that could be included in logistic regression were systolic blood pressure (SBP) <90 mmHg, diastolic blood pressure (DBP) <60 mmHg, revascularization history, smoking history, stable angina pectoris (SAP), ST-segment elevation myocardial infarction (STEMI), LWASO, transfemoral intervention (TFI), single-vessel disease percutaneous coronary intervention (SVD PCI), no statins, white blood cells (WBCs), and serum potassium (K). The continuous variables WBC and K were converted into binary variables according to the cutoff value (the cutoff value of WBC is $6.555 \times 10^9/L$, the cutoff value of K is 4.285 mmol/L) and were included in the regression model. SVD PCI (OR = 0.612, 95% CI: 0.416-0.899, $P = .012$) was a protective factor against cardiac death. SAP (OR = 4.723, 95% CI: 1.098~20.322, $P = .037$), combined with LWASO (OR = 2.631, 95% CI: 1.272~5.440, $P = .009$), $K > 4.285$ mmol/

L (OR = 1.44, 95% CI: 1.002~2.069, $P = .049$), and without statins (OR = 2.015, 95% CI: 1.072~3.789, $P = .030$) were risk factors for cardiac death (Table 2 and Figure 2).

Discussion

Patients with renal dysfunction are more prone to cardiovascular disease (CVD), high mortality and poor prognosis.³ CVD is a major cause of morbidity and mortality in patients with CKD.⁴ To date, all randomized controlled studies on myocardial revascularization have excluded CKD patients; therefore, revascularization therapy with CKD is currently based on observational studies.⁵ Current retrospective studies have shown that CKD patients have better survival after revascularization than drug therapy.^{6,7} A large amount of registration data shows that inpatient revascularization is associated with improved survival regardless of the estimated glomerular filtration rate (eGFR).⁸ PCI was superior to CABG in short-term all-cause death and cerebrovascular accident rates.^{2,9} However, some studies have shown that the restenosis rate after PCI in ESRD patients is almost twice that of patients with normal renal function.¹⁰ Cardiovascular event (CV) risk and mortality are inversely proportional to eGFR,⁸ but the eGFR level that begins to increase the risk is not clearly defined.^{10,11} Elderly individuals are more likely to be complicated with CAHD and CKD. Therefore, it is of great practical significance to systematically analyze the clinical characteristics of elderly patients with severe CKD under PCI treatment and to establish a multifactor risk prediction model to improve the identification and management of patients with poor prognosis, PCI treatment strategies and the clinical prognosis of these patients.

Table I. Characteristics and Therapies in the Different Treatment Groups

Characteristics and Therapies	Cardiac death group N = 142	No cardiac death group N = 868	Z/X ²	P
Sex (male), n (%)	109(76.80%)	677(78.00%)	0.108	.743
Age (y)	71.00(66.00,75.00)	71.00(65.00,75.00)	-0.271	.786
SBP < 90 mmHg, n (%)	5(3.50%)	11(1.30%)	3.976	.046
DBP < 60 mmHg, n (%)	9(6.30%)	27(3.10%)	3.698	.045
HR (beats per min)	70.00(64.00,76.00)	70.00(62.00,78.00)	-0.065	.948
BMI (kg/m ²)	24.06(23.44,27.18)	24.22(23.44,26.99)	-0.505	.613
Hospital stay (days)	6.00(4.00,9.00)	6.00(4.00,9.00)	-0.832	.405
PFH, n (%)	15(10.60%)	74(8.50%)	0.631	.427
Revascularization history, n (%)	26(18.30%)	208(24.00%)	2.191	.139
Smoking history, n (%)	69(48.60%)	370(42.60%)	1.767	.184
Recurrent smoking, n (%)	44(31.00%)	230(26.50%)	1.244	.265
Grace Score	146.00(128.00,165.00)	149.00(126.00,173.00)	-1.016	.31
SAP, n (%)	3(2.10%)	5(0.60%)	3.667	.055
UAP, n (%)	73(51.40%)	487(56.10%)	1.09	.296
NSTEMI, n (%)	16(11.30%)	106(12.20%)	0.102	.749
STEMI, n (%)	47(33.10%)	222(25.60%)	3.534	.06
OMI, n (%)	24(16.90%)	175(20.20%)	0.82	.365
HF, n (%)	39(27.50%)	198(22.80%)	1.472	.225
Hypertension, n (%)	107(75.40%)	654(75.30%)	<0.001	.999
Diabetes, n (%)	53(37.30%)	309(35.60%)	0.158	.691
Dyslipidemia, n (%)	37(26.10%)	218(25.10%)	0.057	.811
Cerebral infarction, n (%)	9 (6.30%)	72(8.30%)	0.634	.426
LEASO, n (%)	11 (7.70%)	29 (3.30%)	6.227	.013
CKD			0.015	.992
CKD stage 3, n (%)	109 (76.80%)	666 (76.70%)		
CKD stage 4, n (%)	29 (20.40%)	179 (20.60%)		
CKD stage 5, n (%)	4 (2.80%)	23 (2.60%)		
TFI, n (%)	58 (40.80%)	303 (34.90%)	1.873	.171
Number of diseased vessels			2.37	.306
SVD, n (%)	21 (14.80%)	160 (18.40%)		
DVD, n (%)	51 (35.90%)	261 (30.10%)		
TVD, n (%)	70 (49.30%)	447 (51.50%)		
AKI, n (%)	19 (13.40%)	106 (12.20%)	0.154	.695
CCC, n (%)	7 (4.90%)	43 (5.00%)	<0.001	.999
CTO, n (%)	37 (26.10%)	204 (23.50%)	0.438	.508
SVD PCI, n (%)	94 (66.20%)	656 (75.60%)	5.616	.018
TVD PCI, n (%)	7 (4.90%)	30 (3.50%)	0.751	.386
LM PCI, n (%)	6 (4.20%)	22 (2.50%)	1.294	.255
LVEF(%)	60.00(54.00,67.00)	60.00(53.00,67.00)	-0.172	.864
Dose of contrast media (ml)	200.00(200.00,300.00)	200.00(200.00,300.00)	-0.148	.882
Without aspirin, n (%)	7 (4.93%)	43 (4.95%)	<0.001	.999
Without P2Y12 antagonists, n (%)	5 (3.52%)	28 (3.23%)	0.034	.854
Without statins, n (%)	14 (9.86%)	50 (5.76%)	3.454	.063
Without beta-blockers, n (%)	36 (25.35%)	206 (23.73%)	0.176	.675
Without RAASI, n (%)	48 (33.80%)	332(38.20%)	1.028	.311
Use of LMWH, n (%)	56 (39.40%)	332 (38.20%)	0.073	.787
Use of CCB, n (%)	52 (36.60%)	353 (40.70%)	0.833	.361
Use of nitrate drugs, n (%)	89 (62.70%)	580 (66.80%)	0.937	.333
Use of tirofiban, n (%)	18 (12.70%)	116 (13.40%)	0.05	.823
Use of PPI, n (%)	30 (21.10%)	223 (25.70%)	1.354	.245
Use of ICA, n (%)	73 (51.40%)	481 (55.40%)	0.791	.374
WBC ($\times 10^9/L$)	7.65(6.66,10.30)	7.55(6.28,9.34)	-1.412	.158
RBC ($\times 10^{12}/L$)	4.07(3.60,4.55)	4.01(3.56,4.42)	-0.912	.362
PLT ($\times 10^9/L$)	199.50(170.00,243.75)	207.00(170.00,246.00)	-0.281	.779
K (mmol/L)	4.20(3.90,4.57)	4.16(3.85,4.50)	-1.503	.133
Na (mmol/L)	139.70(137.00,141.88)	139.90(137.30,141.70)	-0.273	.785
Cl (mmol/L)	105.00(103.00,108.00)	105.70(103.00,108.00)	-0.537	.591
Ca (mmol/L)	2.22(2.12,2.32)	2.22(2.13,2.32)	-0.207	.836

(continued)

Table 1. (continued)

Characteristics and Therapies	Cardiac death group N = 142	No cardiac death group N = 868	Z/ χ^2	P
UA ($\mu\text{mol/L}$)	415.20(350.18,495.60)	418.80(349.50,495.00)	-0.35	.727
eGFR (ml/min.1.73m^2)	40.03(30.79,48.04)	39.49(30.69,48.89)	-0.307	.759
CK (mmol/L)	84.00(50.25,150.00)	85.00(55.00,161.00)	-0.445	.656
CK-MB (mmol/L)	3.60(1.43,15.15)	4.00(1.50,14.50)	-0.174	.862
TG (mmol/L)	1.53(1.14,2.06)	1.58(1.13,2.10)	-0.289	.773
TCH (mmol/L)	4.23(3.56,4.94)	4.18(3.51,4.88)	-0.145	.885
HDLc (mmol/L)	0.99(0.83,1.16)	0.97(0.83,1.13)	-0.977	.329
LDLc (mmol/L)	2.57(2.01,3.02)	2.51(2.01,3.02)	-0.532	.595
AST (mmol/L)	25.00(18.00,40.50)	24.00(18.00,37.00)	-0.949	.342
ALT (mmol/L)	21.00(14.00,33.00)	20.00(14.00,31.00)	-0.577	.564
TP (mmol/L)	67.55(63.10,71.43)	66.90(62.90,71.30)	-0.897	.37

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; BMI, body mass index; PFH, positive family history; SAP, stable angina pectoris; UAP, unstable angina pectoris; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction; OMI, old myocardial infarction; HF, heart failure; LEASO, lower extremity arteriosclerosis obliterans; CKD, chronic kidney disease; TFI, transfemoral intervention; SVD, single-vessel disease; DVD, double-vessel disease; TVD, three-vessel disease; AKI, acute kidney injury; CCC, coronary collateral circulation; CTO, chronic total occlusion; PCI, percutaneous coronary intervention; LM, left main; LVEF, left ventricular ejection fraction; RAASi, renin-angiotensin-aldosterone system inhibitors; LMWH, low-molecular-weight heparin; CCB, calcium channel blocker; PPI, proton pump inhibitors; ICA, isotonic contrast agent; WBC, white blood cell; RBC, red blood cell; PLT, platelets; UA, uric acid; eGFR, estimate glomerular filtration rate; CK, creatine kinase; CK-MB, creatine kinase isoenzymes; TG, triglyceride; TCH total cholesterol; HDLc, high-density lipoprotein cholesterol; LDLc, low-density lipoprotein cholesterol; AST, aspartate aminotransferase; ALT, alanine aminotransferase; TP, total protein.

Table 2. Multivariate Logistic Regression of Cardiac Death.

Variable	Adjusted OR	95% CI	P value
SVD PCI	0.612	0.416-0.899	.012
SAP	4.723	1.098-20.322	.037
LEASO	2.631	1.272-5.440	.009
K > 4.285 mmol/L	1.440	1.002-2.069	.049
Without stains	2.015	1.072-3.788	.030

Abbreviations: OR, odds ratio; CI, confidence interval; SVD, single-vessel disease; PCI, percutaneous coronary intervention; SAP, stable angina pectoris; LEASO, lower extremity arteriosclerosis obliterans.

Currently, it is believed that patients with CKD stage 3 and above receiving invasive strategies do not experience reduced end events,¹² but in the real world, a large number of patients with CKD still need revascularization therapy, which is driven by severe ischemic events. In this study, PCI did not reduce the risk of cardiac death in CKD patients with stable angina. This conclusion was also confirmed by the results of ischemia-CKD clinical trials.¹³⁻¹⁵ Among patients with stable coronary disease, advanced chronic kidney disease, and moderate or severe ischemia, the Ischemia-CKD Clinical Trials did not provide evidence that the initial invasive strategy, in contrast to a conservative strategy with maximal medical management,¹³ reduced the risk of death, myocardial infarction or nonfatal myocardial infarction¹⁴ and relieved angina symptoms.¹⁵

A study¹⁶ including 453 573 subjects showed that LEASO is more common in patients with CKD and is related to lower extremity complications and mortality. CKD is more interrelated with wide vascular disease compared with the general population. Patients with both conditions had lower survival rates.¹⁷ Some studies^{17,18} have suggested that CKD patients with albuminuria have decreased GFR, inflammatory

conditions, and calcium and phosphate metabolism disorders that cause systemic vascular damage. Therefore, a number of authors considered CKD to be a risk factor for vascular disease. Currently, the great majority of organizations suggest the detection of LEASO in CKD patients. A recent study supports this suggestion because the results showed that a low ABI was a predictor of organ failure and death.¹⁹ In addition, we cannot ignore that LEASO is a significant symbol of severe systemic atherosclerosis and is strongly associated with poor prognosis.²⁰

CKD patients are liable to electrolyte disturbance, which is related to poor prognosis. For CKD patients, both hypo- and hyperkalemia can have direct harmful physiological effects. Some meta-analyses²¹ show that the U-shaped curve corresponds to the relationship between potassium levels and the risk of adverse outcomes, with the lowest risk at serum potassium levels of 4-4.5 mmol/L. In our study, the results also confirm this conclusion. Hyperkalemia leads to poor prognosis in patients with CAHD, which may be due to arrhythmia, influence on the use of ACEIs or ARBs, etc.^{22,23}

In actual clinical work, the proportion of CKD patients receiving reperfusion or revascularization treatment is significantly reduced; therefore, the proportion of CKD patients receiving aspirin, clopidogrel, statins, ACEIs or ARBs and β -blockers is significantly lower than that of patients with normal renal function. Some studies²⁴ suggest that statins have a poor effect in reducing cardiovascular complications in CKD patients, and statins are not recommended for dialysis patients. However, in other studies,²⁵ when the indication was extended to all CKD patients, the authors concluded that atorvastatin therapy significantly reduced the relative risk of major outcomes in CKD patients. Focused atorvastatin therapy reduces the cardiovascular risk for CKD patients in the real world compared with conventional treatment, and

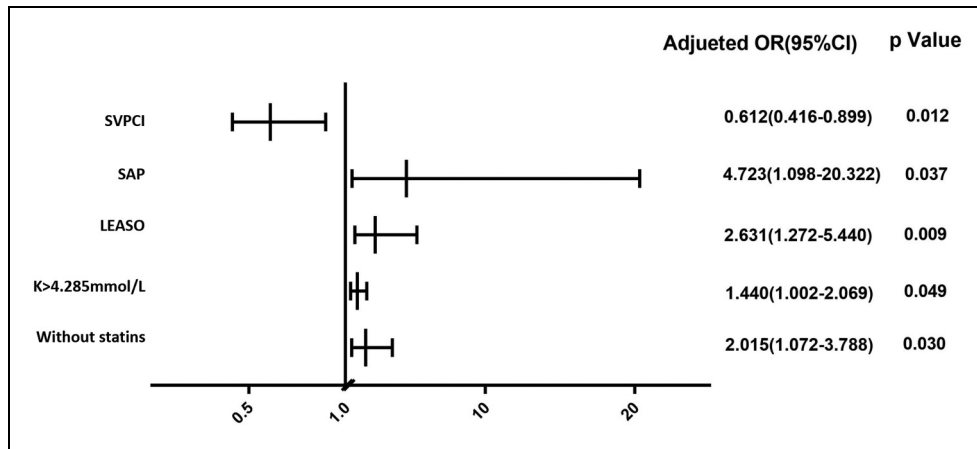


Figure 2. Multivariate logistic regression of cardiac death. OR, odds ratio; CI, confidence interval; SVPCI, single-vessel percutaneous coronary intervention; SAP, stable angina pectoris; LEASO, lower extremity arteriosclerosis obliterans.

there is no significant difference in treatment effects between patients with and without CKD. In addition, the eGFR did not decrease in patients treated with atorvastatin during the study period.

SVD PCI was a protective factor against cardiac death and may be related to mild CAHD and nonsevere and complex vascular disease. SVD PCI can control the dosage of contrast medium and avoid the aggravation of renal function damage or AKI.

CAHD is very common in CKD. CKD alters the traditional risk factors for atherosclerosis, and CKD has some unique mechanisms, such as inflammation and changes in mineral metabolism, playing a major pathophysiological role in the decline of renal function.²⁶ Therefore, it is of great significance for clinical work to understand the prognostic risk factors for patients with severe CKD complicated with CAD.

Limitation

The limitations of this study are mainly reflected in the single-center retrospective study, which may have certain selection bias. Second, due to the study enrollment group is relatively rare and the enrollment time is long, there are great developments and changes in coronary intervention technology during this period. Third, prospective multicenter studies are needed to further verify the results.

Conclusions

In elderly and serious CAHD-CKD patients after PCI, SVD PCI was a protective factor against cardiac death. However, SAP, CAHD-CKD combined with LEASO, K > 4.285 mmol/L, and no statins were independent risk factors of cardiac death for elderly patients with severe CKD after PCI.

Author Contributions

YZ and JLW contributed to the conception or design of the work. GYZ contributed to the acquisition, analysis, interpretation of data for the

work. YZ drafted the manuscript. YJZ critically revised the manuscript. All gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical Approval

This study was approved by the Ethics Committee of Beijing Anzhen Hospital.

Informed Consent

The requirement for informed consent was obtained for this study by follow-up

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References

1. Johnston N, Dargie H, Jardine A. Diagnosis and treatment of coronary artery disease in patients with chronic kidney disease: ischemic heart disease. *Heart*. 2008;94(8):1080-1088.
2. Charytan DM, Li S, Liu J, et al. Risks of death and end-stage renal disease after surgical compared with percutaneous coronary revascularization in elderly patients with chronic kidney disease. *Circulation*. 2012;126(11 Suppl 1):S164-S169.

3. Herzog CA, Ma JZ, Collins AJ. Long-term outcome of dialysis patients in the United States with coronary revascularization procedures. *Kidney Int.* 1999;56(1):324-332.
4. Bansal N, Katz R, Robinson-Cohen C, et al. Absolute rates of heart failure, coronary heart disease, and stroke in chronic kidney disease: an analysis of 3 community-based cohort studies. *JAMA Cardiol.* 2017;2(3):314-318.
5. Neumann F-J, Sousa-Uva M, Ahlsson A, et al. 2018 ESC/EACTS guidelines on myocardial revascularization. *Eur Heart J.* 2019;40(2): 87-165.
6. Hemmelgarn BR, Southern D, Culleton BF, et al. Survival after coronary revascularization among patients with kidney disease. *Circulation.* 2004;110(14):1890-1895.
7. Huang HD, Alam M, Hamzeh I, et al. Patients with severe chronic kidney disease benefit from early revascularization after acute coronary syndrome. *Int J Cardiol.* 2013;168(4):3741-3746.
8. Bhandari S, Jain P. Management of acute coronary syndrome in chronic kidney disease. *J Assoc Physicians India.* 2012;60:48-51.
9. Wu P, Luo F, Fang Z. Multivessel coronary revascularization strategies in patients with chronic kidney disease: a meta-analysis. *Cardiorenal Med.* 2019;9(3):145-159.
10. Reddan DN, Szczech LA, Tuttle RH, et al. Chronic kidney disease, mortality, and treatment strategies among patients with clinically significant coronary artery disease. *J Am Soc Nephrol.* 2003;14(9):2373-2380.
11. Szummer K, Lundman P, Jacobson SH, et al. Influence of renal function on the effects of early revascularization in non-ST-elevation myocardial infarction. *Circulation.* 2009;120(10):851-858.
12. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *JACC.* 2014;64(24):e139-e228.
13. Arif H, Yadav A. Approach to stable angina in patients with advanced chronic kidney disease. *Curr Opin Nephrol Hypertens.* 2021;30(3):339-345.
14. Bangalore S, Maron DJ, O'Brien SM, et al. Management of coronary disease in patients with advanced kidney disease. *N Engl J Med.* 2020;382(17):1608-1618.
15. Paul TK, Mamas MA, Shanmugasundaram M, et al. Medical therapy versus revascularization in patients with stable ischemic heart disease and advanced chronic kidney disease. *Curr Cardiol Rep.* 2021;23(4):23.
16. Bourrier M, Ferguson TW, Embil JM, et al. Peripheral artery disease: its adverse consequences with and without CKD. *Am J Kidney Dis.* 2020;75(5):705-712.
17. Bosevski M. Peripheral arterial disease and chronic kidney disease. *Pril (Makedon Akad Nauk Umet Odd Med Nauki).* 2017;38(2):29-33.
18. Provenzano M, Andreucci M, Garofalo C, et al. The association of matrix metalloproteinases with chronic kidney disease and peripheral vascular disease: a light at the end of the tunnel? *Biomolecules.* 2020;10(1):154.
19. Patel SI, Chakkera HA, Wennberg PW, et al. Peripheral arterial disease preoperatively may predict graft failure and mortality in kidney transplant recipients. *Vasc med.* 2017;22(3):225-230.
20. Ghoshal S, Freedman BI. Mechanisms of stroke in patients with chronic kidney disease. *Am J Nephrol.* 2019;50(4):229-239.
21. Kovesdy CP, Matsushita K, Sang Y, et al. Serum potassium and adverse outcomes across the range of kidney function: a CKD prognosis consortium meta-analysis. *Eur Heart J.* 2018;39(17):1535-1542.
22. Gilligan S, Raphael KL. Hyperkalemia and hypokalemia in CKD: prevalence, risk factors, and clinical outcomes. *Adv Chronic Kidney Dis.* 2017;24(5):315-318.
23. Fu EL, Evans M, Clase CM, et al. Stopping renin-angiotensin system inhibitors in patients with advanced CKD and risk of adverse outcomes: a nationwide study. *J Am Soc Nephrol.* 2021;32(2):424-435.
24. Agrawal H, Aggarwal K, Littrell R, et al. Pharmacological and non pharmacological strategies in the management of coronary artery disease and chronic kidney disease. *Curr Cardiol Rev.* 2015;11(3):261-269.
25. Koren MJ, Davidson MH, Wilson DJ, et al. Focused atorvastatin therapy in managed-care patients with coronary heart disease and CKD. *Am J Kidney Dis.* 2009;53(5):741-750.
26. Galougahi KK, Chadban S, Mehran R, et al. Invasive management of coronary artery disease in advanced renal disease. *Kidney Int Rep.* 2021;6(6):1513-1524.