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Research article

The relationship between serum 1,5-anhydroglucitol and adverse outcomes in acute coronary syndrome with and without chronic kidney disease patients

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ABSTRACT

Purpose: Individuals with chronic kidney disease (CKD) face an elevated residual risk of cardiovascular events, but the relationship between this residual risk and 1,5-anhydroglucitol (1,5-AG) is uncertain. Our study aimed to examine the effect of 1,5-AG on major adverse cardiovascular events (MACEs) and all-cause mortality in acute coronary syndrome (ACS) individuals.

Methods: 1253 ACS participants hospitalized were enrolled at Beijing Hospital between March 2017 and March 2020. All participants were classified into 2 groups based on their eGFR (60 ml/min/1.73 m^2). The link between 1,5-AG and adverse outcome was investigated in non-CKD and CKD participants.

Results: CKD patients had reduced concentrations of 1,5-AG than those without CKD. Throughout a median follow-up duration of 43 months, 1,5-AG was an autonomous hazard factor for MACEs and all-cause mortality. 1,5-AG<14 μg/ml participants had greater MACEs and all-cause mortality risk than those with 1,5-AG≥14 μg/ml, regardless of renal function. Furthermore, concomitant reduced concentrations of 1,5-AG and CKD portended a dismal prognosis in ACS patients.

Conclusions: 1,5-AG was autonomously linked to MACEs and all-cause mortality in ACS participants with both non-CKD and CKD. Co-presence of reduced concentrations of 1,5-AG and CKD may portend adverse clinical outcomes.

1. Introduction

The strong connection between kidney and cardiac pathological conditions is well-documented [1,2]. Chronic kidney disease

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(CKD) patients experienced an elevated occurrence of adverse cardiovascular events than patients without renal insufficiency [3]. Understanding the status of CKD is essential for risk prediction of adverse outcomes [4]. Even with secondary prevention strategies in place for acute coronary syndrome (ACS) participants with CKD, the hazard of adverse events remains elevated [5,6].

Blood glucose fluctuation could potentially have a crucial role in the advancement of coronary heart disease (CHD) patients [7]. 1, 5-anhydroglucitol (1,5-AG) levels drop quickly with urinary glucose excretion, indicating glucose status over the past two weeks [8]. Recent research have found that 1,5-AG may provide a meaningful addition to glycosylated hemoglobin (Hb1Ac) testing, indicating short-term blood glucose status in contrast to traditional glucose markers [9]. Reduced concentrations of 1,5-AG were autonomously linked to the prevalence and severity of CHD, even after adjusting for fasting blood glucose and HbA1c [7]. Renal function should be considered while interpreting 1,5-AG concentration [10,11]. The link between 1,5-AG and renal insufficiency is a subject of ongoing debate. Our study sought to explore the link between 1,5-AG concentrations and renal function in ACS participants.

2. Methods

2.1. Study participants

Participants were from the prospective cohort of Beijing Hospital Atherosclerosis Study (NCT03072797). 1754 ACS individuals were enrolled between March 2017 and March 2020. The criteria for inclusion were being older than 18 years, hospitalized due to ACS, and having accessible serum 1,5-AG measurements. From an overall pool of 1754 individuals, 1253 were included in the study after excluding 461 patients who did not receive dual anti-platelet therapy and 40 patients lost during follow-up (Supplemental Fig. 1). As a pre-specified study protocol, exclusions were made for patients with severe renal impairment, significant hepatic dysfunction, malignancies, primary pulmonary hypertension, advanced cardiac insufficiency, and severe congenital heart disease. CKD was defined as eGFR$<$60 ml/min/1.73 m^2 [5]. The Ethics Committee of Beijing Hospital (2016BJYYEC-121-02) approved the study protocol. All patients gave written informed consent.

2.2. Clinical data

Patient demographic parameters were extracted from medical records, encompassing current smoking status, diabetes mellitus, hypertension, history of stroke, past myocardial infarction (MI), and prior percutaneous coronary intervention (PCI). We also collected medication use at the time of discharge. To detect the severity of ischemia caused by the lesions of coronary artery, the Gensini score was calculated for ACS patients [12]. 1,5-AG was analyzed in venous blood samples before coronary angiography [7].

3. Clinical outcomes

The primary endpoint of the study was major adverse clinical events (MACEs), defined as a composite of cardiac death, non-fatal myocardial infarction (MI), non-fatal ischemic stroke, and coronary revascularization. The secondary outcome measured was all-cause mortality. Patients were followed up through clinic visits and telephone interviews until March 2023.

3.1. Statistical analysis

Continuous variables were summarized as mean ± standard deviation or median with interquartile range and compared between groups using the Student's t-test or Mann-Whitney *U* test. Categorical variables were expressed as frequencies (percentages) and analyzed using the chi-square ($\chi 2$) test. Correlations between 1,5-AG and other variables were examined using Pearson or Spearman analysis.

Variables that *p*-value$<$0.10 in the univariate Cox analysis were incorporated into the multivariate analysis. We categorized the ACS subjects into two groups (1,5-AG concentrations$<$14.0 μg/mL, and $\geq$14.0 μg/mL) [11]. Restricted cubic spline (RCS) analysis with four knots was conducted to detect potential nonlinear associations between 1,5-AG and adverse outcome, with the reference point using the value of 1,5-AG concentration (14 μg/ml). We also performed subgroup analysis to explore the association between 1,5-AG and MACE or all-cause mortality. All statistical analyses were conducted using IBM SPSS Statistics version 26.0 and R version 3.6.1 by the R Development Core Team in Vienna, Austria.

4. Results

4.1. Baseline clinical characteristics

1253 ACS patients was screened for eligibility. The age was 66.7 ± 10.7 years, and 31.7 % (397/1253) patients were women. 16.0 % (200/1253) patients had CKD. CKD patients were older and predominantly female, hypertension, diabetes mellitus, history of stroke, and myocardial infarction (MI). CKD patients exhibited significantly larger FPG, HbA1c, and lower 1,5-AG concentrations. CKD patients were less inclined to perform angiotensin-converting enzyme inhibitors/angiotensin receptor blockers and PCI, compared with non-CKD patients, as described in Table 1.

The distribution of 1,5-AG was shown in Supplemental Fig. 2A. The median level of 1,5-AG was 20.7 (11.4–29.4 μg/ml). CKD patients had lower concentrations of 1,5-AG than those without CKD ([17.3 (9.3–25.3) μg/ml vs. 21.3 (12.3–29.9) μg/ml, *p*-value =

0.001]), as described in Supplemental Fig. 2B. A significant inverse link was described between 1,5-AG concentration and FPG (r = −0.341, *p*-value<0.001), Hb1Ac (r = −0.537, *p*-value<0.001), and Gensini score (r = −0.146, *p*-value<0.001), (Supplemental Table 1). However, there was no significant link between 1,5-AG concentration and eGFR (r = 0.023, *p*-value = 0.409), (Supplemental Fig. 3). Moreover, we found that 1.5-AG concentration was significantly lower in ST-segment elevation MI patients than in unstable angina ([14.0 (6.9–24.7) μg/ml vs. 21.5 (12.5–30.0) μg/ml, *p*-value<0.001]), but there were no meaningful variation in 1.5-AG concentration with non-ST-segment elevation MI patients compared to unstable angina ([20.8 (11.7–30.0) μg/ml vs. 21.5 (12.5–30.0) μg/ml, *p*-value = 0.553]), as described in Supplemental Fig. 4.

5. The impact of 1,5-AG on adverse outcomes

Baseline characteristics based on MACEs and all-cause mortality groupings is shown in Supplemental Tables 2 and 3 Throughout a median follow-up duration of 43 months, MACEs occurred in 16.9 % (212/1253) patients, including 61 patients with cardiac death, 25 patients with non-fatal MI, 33 patients with non-fatal ischemic stroke, and 93 patients with coronary revascularization. 109 (8.7 %) patients with all-cause death occurred. Multivariate analysis indicated that the per 5 μg/ml increase of 1,5-AG concentration was autonomously linked to hazard of MACEs (HR 0.81, 95 % CI 0.75–0.88, *p*-value<0.001), as described in Supplemental Table 4. The per 5 μg/ml increase in 1,5-AG was autonomously linked to low risk of overall mortality (HR 0.78, 95 % CI 0.69–0.88, *p*-value<0.001), as described in Supplemental Table 5. The hazard of MACEs increased as 1,5-AG concentration decreased (p-value for non-linearity = 0.022) (Supplemental Fig. 5A). Conversely, the hazard of mortality decreased with higher 1,5-AG concentrations, (*p*-value for non-

Table 1
Baseline clinical characteristics.

Variables	All	Non-CKD	CKD	*p*-value
Patients	1253	1053	200	
Age, years	66.7 ± 10.7	65.0 ± 10.3	75.9 ± 7.9	<0.001
Female, gender	397 (31.7 %)	303 (28.8 %)	94 (47.0 %)	<0.001
BMI, kg/m^2	26.0 ± 6.1	25.9 ± 5.3	27.0 ± 9.1	0.079
Gensini score	28.5 (12.0–57.0)	26.5 (11.5–53.3)	42.0 (17.1–78.8)	<0.001
Current smoking	441 (35.3 %)	399 (38.0 %)	42 (21.0 %)	<0.001
Hypertension	992 (79.2 %)	803 (76.3 %)	189 (94.5 %)	<0.001
Diabetes mellitus	658 (52.6 %)	529 (50.2 %)	129 (64.5 %)	0.004
Prior stroke	139 (11.1 %)	106 (10.1 %)	33 (16.5 %)	0.008
Prior MI	159 (12.7 %)	118 (11.2 %)	41 (20.5 %)	<0.001
Prior hyperlipidemia	592 (47.2 %)	504 (47.9 %)	88 (44.0 %)	0.316
Prior PCI	472 (37.7 %)	389 (36.9 %)	83 (41.5 %)	0.223
Laboratory test				
WBC, mmol/l	6.5 ± 1.7	6.5 ± 1.8	6.7 ± 1.8	0.092
eGFR, ml/min/1.73 m^2	83.0 (68.3–95.5)	87.2 (76.2–97.8)	49.7 (43.3–55.6)	<0.001
LDL-C, mmol/l	2.0 ± 0.7	2.0 ± 0.7	2.0 ± 0.8	0.785
HDL-C, mmol/l	1.0 ± 0.3	1.0 ± 0.3	1.0 ± 0.3	0.137
TC, mmol/l	3.5 ± 0.8	3.5 ± 0.8	3.5 ± 0.9	0.909
TG, mmol/l	1.5 (1.1–2.1)	1.5 (1.1–2.1)	1.6 (1.2–2.1)	0.543
FPG, mmol/l	7.0 ± 2.5	6.8 ± 2.3	7.9 ± 3.3	<0.001
HbA1c, %	6.5 ± 1.1	6.4 ± 1.0	6.9 ± 1.2	<0.001
1,5-AG	20.7 (11.4–29.4)	21.3 (12.3–29.9)	17.3 (9.3–25.3)	0.001
1,5-AG <14 μg/ml	387 (30.9 %)	310 (29.4 %)	77 (38.5 %)	0.011
Type of ACS <0.001				
UA	988 (78.9 %)	1031(86.5 %)	155 (72.8 %)	
NSTEMI	118 (9.4 %)	95 (9.0 %)	23 (11.5 %)	
STEMI	147 (11.7 %)	117 (11.1 %)	30 (15.0 %)	
PCI	894 (71.3 %)	765 (72.6 %)	129 (64.5 %)	0.019
Medication at discharge				
ACEI/ARB	910 (72.6 %)	788 (74.8 %)	122 (61.0 %)	<0.001
β-blocker	1144 (91.3 %)	968 (91.9 %)	176 (88.0 %)	0.071
CCB	455 (36.3 %)	372 (35.3 %)	83 (41.5 %)	0.722
Clinical outcomes				
MACEs	212 (16.9 %)	148 (14.1 %)	64 (32.0 %)	<0.001
Cardiac death	61 (4.9 %)	32 (3.0 %)	29 (14.5 %)	<0.001
Non-fatal MI	25 (2.0 %)	15 (1.4 %)	10 (5.0 %)	0.001
Non-fatal ischemic stroke	33 (2.6 %)	22 (2.1 %)	11 (5.5 %)	0.006
Coronary revascularization	93 (7.4 %)	79 (7.5 %)	14 (7.0 %)	0.804
All-cause mortality	109 (8.7 %)	61 (5.8 %)	48 (24.0 %)	<0.001

Note: Data are expressed as the mean value ± standard deviation, median with 25th and 75th or number (%). 1,5-AG, 1,5-anhydro-*d*-glucitol; ACEI, angiotensin-converting enzyme inhibitor; ACS, acute coronary syndrome; ARB, angiotensin receptor blocker; BMI, body mass index; CCB, calcium-channel blocker; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; FPG, fasting plasma glucose; HbA1c, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MACEs, major adverse clinical events. MI, myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction; TC, total cholesterol; TG, triglyceride; UA, unstable angina; WBC, white blood cell.

linearity = 0.263), (Supplemental Fig. 5B).

Moreover, an elevated 1,5-AG was consistently linked to the reduced hazard of MACEs and mortality using the threshold value of 1,5-AG concentration (14 μg/ml) and the median (20.65 μg/ml), as shown in Table 2. No significant interactions were detected across different subgroups (Supplemental Figs. 6 and 7).

Kaplan-Meier curve analysis described that the MACEs were greater in the 1,5-AG<14 μg/ml patients, compared with patients in the 1,5-AG≥14 μg/ml, as described in Fig. 1A and B. All-cause mortality rates were also greater in the 1,5-AG<14 μg/ml patients, compared to patients with 1,5-AG≥14 μg/ml, as described in Fig. 1C and D.

Moreover, the risk of MACEs was high in 1,5-AG<14 μg/ml patients after correcting risk factors in CKD and non-CKD patients. However, no statistically significant MACEs increase was detected in the 1.5-AG<20.65 μg/ml patients after correcting factors. Conversely, participants with 1,5-AG<20.65 μg/ml was autonomously linked to mortality in CKD and non-CKD participants. However, no meaningful statistical correlation was detected in 1,5-AG<14 μg/ml with the increased risk of mortality in non-CKD patients (Table 2).

We used RCS to explore the link between 1,5-AG concentration and the hazard of MACEs and all-cause mortality. The hazard of MACEs was significantly increased with the decrease of 1,5-AG concentrations in non-CKD and CKD individuals (p-value for non-linearity = 0.180; p-value for non-linearity = 0.003; respectively), (Supplemental Figs. 8A and 8B). Moreover, the hazard of mortality was significantly increased with the decrease of 1,5-AG concentrations in CKD participants (p-value for non-linearity = 0.145), with the 1,5-AG concentration 14 μg/ml as the reference, (Supplemental Figs. 9A and 9B). Nevertheless, in non-CKD patients, reduced concentrations of 1,5-AG were not substantially linked to an increased risk of mortality (Supplemental Fig. 9B).

Further analysis of MACEs and mortality according to 1,5-AG concentrations and renal function. Kaplan-Meier curves analysis described that CKD participants with 1,5-AG concentration<14 μg/ml had markedly elevated cumulative incidences of MACEs and all-cause mortality (all log-rank p-value <0.001), as shown in Fig. 2.

6. Discussion

There are several findings in our study. First, 1,5-AG concentration was low in CKD individuals. Second, the reduced 1,5-AG concentration was autonomously linked to a high hazard of adverse outcomes regardless of renal function. Furthermore, the co-presence of low concentrations of 1,5-AG and CKD portends a high hazard for adverse outcomes. These results indicated that 1,5-AG might be a valuable marker for predicting adverse outcomes.

1,5-AG is a carbon-1 deoxy pyranose mainly derived from dietary intake [11]. Serum 1,5-AG is not bound to hemoglobin, which is not influenced by the lifespan of erythrocytes [13]. The status of 1,5-AG in blood and tissues remain consistent because of reabsorption

Table 2
The incidence of adverse outcomes in patients with or without CKD stratified by the 1.5-AG levels in the multivariate Cox regression analysis.

Variables	All (n = 1253)			Non-CKD (n = 1053)			CKD (n = 200)		
	n (%)	Adjusted HR (95 % CI)	p-value	n (%)	Adjusted HR (95 % CI)	p-value	n (%)	Adjusted HR (95 % CI)	p-value
MACEs									
1,5-AG, per 5 μg/ml		0.81 (0.75–0.88)	<0.001		0.84 (0.77–0.93)	<0.001		0.76 (0.64–0.90)	0.001
1,5-AG									
≥14 μg/ml	102/866 (11.8 %)	Reference		76/743 (10.2 %)	Reference		26/123 (21.1 %)	Reference	
<14 μg/ml	110/387 (28.4 %)	2.39 (1.71–3.33)	<0.001	72/310 (23.2 %)	2.07 (1.37–3.11)	0.001	38/77 (49.4 %)	2.77 (1.51–5.09)	0.001
1,5-AG by median									
≥20.65 μg/ml	73/626 (11.7 %)	Reference		56/549 (10.2 %)	Reference		17/77 (22.1 %)	Reference	
<20.65 μg/ml	139/627 (22.2 %)	1.59 (1.14–2.21)	0.006	92/504 (18.3 %)	1.45 (0.98–2.15)	0.061	47/123 (38.2 %)	1.75 (0.92–3.33)	0.089
All-cause mortality									
1,5-AG, per 5 units		0.78 (0.69–0.88)	<0.001		0.83 (0.72–0.96)	0.014		0.70 (0.56–0.87)	0.001
1,5-AG									
≥14 μg/ml	51/866 (5.9 %)	Reference		35/743 (4.7 %)	Reference		16/123 (13.0 %)	Reference	
<14 μg/ml	58/387 (15.0 %)	1.90 (1.22–2.95)	0.004	26/310 (8.4 %)	1.22 (0.66–2.26)	0.525	32/77 (41.6 %)	3.52 (1.68–7.38)	0.001
1,5-AG by median									
≥20.65 μg/ml	24/626 (3.8 %)	Reference		16/549 (2.9 %)	Reference		8/77 (10.4 %)	Reference	
<20.65 μg/ml	85/627 (13.6 %)	2.43 (1.48–3.98)	<0.001	45/504 (8.9 %)	2.14 (1.14–4.02)	0.017	40/123 (32.5 %)	2.88 (1.20–6.93)	0.018

Abbreviations:1,5-AG, 1,5-anhydroglucitol; CI, confidence interval; CKD, chronic kidney disease; HR, hazard ratio; MACEs, major adverse clinical events.

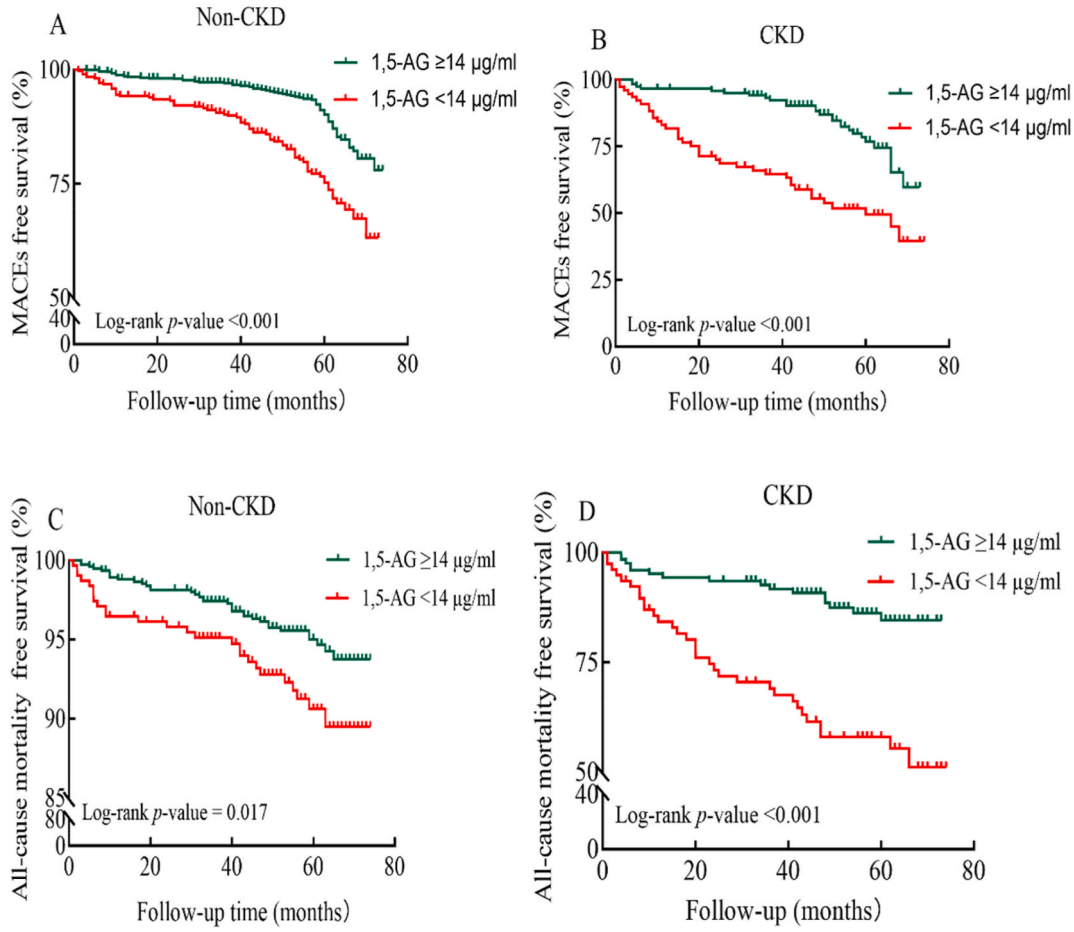

Fig. 1. Kaplan–Meier curves for clinical outcomes according to 1,5-AG concentration (<14 μg/ml or ≥14 μg/ml) in ACS patients. (A) MACEs without CKD patients; (B) MACEs with CKD patients; (C) All-cause mortality without CKD patients; (D) All-cause mortality with CKD patients. 1,5-AG, 1,5-anhydroglucitol; ACS, acute coronary syndrome; CKD, chronic kidney disease; MACEs, major adverse clinical events.

in the kidney's proximal tubules [14]. 1,5-AG is eliminated through the urine and its serum levels decrease expeditiously [15]. 1,5-AG might help prevent increases in blood glucose levels by inhibiting sucrase and intestinal glucose absorption, making it particularly valuable as an indicator of short-term fluctuations in blood glucose [16].

Kim et al. reported that mild or moderate renal insufficiency does not affect 1,5-AG concentrations [17]. Selvin et al. collected 1,5-AG concentrations from more than 10,000 participants in the ARIC study, found that 1,5-AG<6 μg/ml is autonomously linked to diabetic retinopathy during 20 years follow-up. The study revealed that diabetes mellitus patients with 1,5-AG<6 μg/ml has a nearly 3-fold increased hazard of CKD compared to 1,5-AG≥10 μg/ml [18]. Hasslacher et al. found that for every 10 ml/min/1.73 m^2 increases in eGFR, 1,5-AG decreased by approximately 0.32 μg/ml in 269 patients with diabetes mellitus, which is probably attributed to differences in renal handling of 1,5-AG under conditions of glomerular hypo- or hyperfiltration. When CKD progresses to stages 4–5, 1,5-AG loses its role in reflecting glycemic changes due to a severe decrease in eGFR [10]. Changes or fluctuations in blood glucose, above average blood glucose levels, may damage the vascular system, which may lead to the advancement of kidney disease [19]. Recent studies described that sodium-glucose cotransporter 4 as a potential transporter for 1,5-AG. As the renal tubular injury progresses, 1,5-AG uptake may decrease due to decreased SGLT4 receptors and increase glucose cotransporter injury [17,20]. Our study found that 1,5-AG concentration were lesser in ACS participants with CKD than those without CKD.

The link between 1,5-AG levels and clinical prognosis remains controversial [21]. 1,5-AG might be implicated in the progression of ACS, concerning oxidative stress, inflammatory response, and endothelial dysfunction of the body [22,23]. 1,5-AG was better predictor of CHD prevalence than glycated hemoglobin [7]. 1,5-AG might indicate an increased likelihood of coronary plaque rupture among 114 ACS patients [24]. Reduced concentration of 1,5-AG were linked to cardiovascular events in CHD participants following the initial PCI, even in HbA1c<7.0 % [25]. At present, the adverse prognosis of 1,5-AG in ACS individuals with CKD has not been explored. Our study initially found that low concentrations of 1,5-AG predicted hazard of MACEs and mortality in ACS individuals, both with and without CKD. Furthermore, our data indicated that reduced concentrations of 1,5-AG were more strongly associated with adverse outcomes in ACS patients with CKD. 1,5-AG could function as a valuable marker for hazard assessment of CKD patients and identify

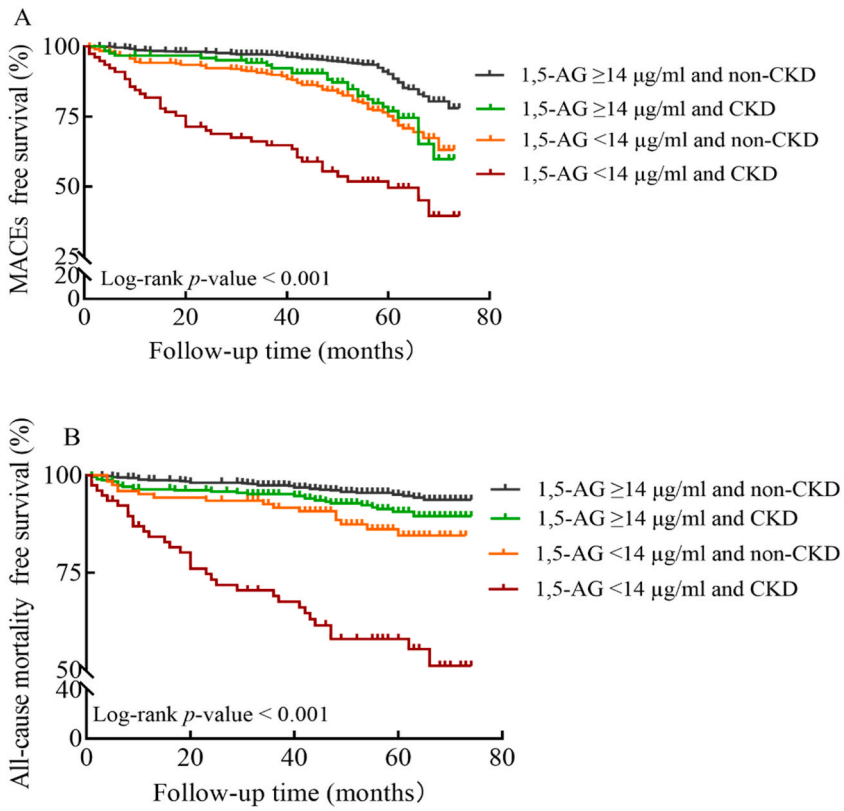

Fig. 2. Kaplan–Meier curves for clinical outcomes according to the combination of 1,5-AG concentration (<14 μg/ml or ≥ 14 μg/ml) and renal function (CKD or Non-CKD). (A) MACEs; (B) All-cause mortality. 1,5-AG, 1,5-anhydroglucitol; CKD, chronic kidney disease; MACEs, major adverse clinical events.

individuals at high hazard who require careful follow-up. Therefore, we recommend enhanced control of 1,5-AG in ACS patients, particularly in those with concomitant CKD.

This study still had some limitations. First, we did not collect information on glucose-lowering medications, which may lead to biased information. Our study did not collect postprandial blood glucose data, and additional research is required to analyze the correlation between 1,5-AG and postprandial blood glucose. Second, we cannot exclude the presence of other unmeasured confounders for the multivariate Cox analysis model. Third, our study could not explore the effect of 1,5-AG on the type of ACS. Due to limited statistical power given our sample size, we couldn't analyze data for eGFR <60 ml/min/1.73 m^2. Further study are required to validate our results.

7. Conclusions

In our study, reduced concentrations of 1,5-AG were autonomously linked to adverse outcomes in ACS patients. Co-presence of CKD and reduced concentrations of 1,5-AG portend a dismal prognosis. Our research indicates that 1,5-AG could be beneficial for categorizing risk in the secondary prevention of ACS.

Ethics statement

The study received approval from Beijing Hospital ethics committee (Number: 2016BJYYEC-121-02). All participants provided informed consent.

Consent for publication

Participants were informed that data would be shared with their names and identities concealed, as per their consent.

Data availability statement

Data will be available upon request from the corresponding author.

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CRediT authorship contribution statement

Yijia Wang: Writing – original draft, Software, Methodology, Investigation, Data curation. **Zhe Wang:** Writing – original draft, Methodology, Investigation. **Ruiyue Yang:** Methodology. **Xinyue Wang:** Software, Resources. **Siming Wang:** Validation, Supervision, Project administration. **Wenduo Zhang:** Validation, Supervision. **Jun Dong:** Writing – review & editing. **Xue Yu:** Writing – review & editing, Writing – original draft. **Wenxiang Chen:** Writing – review & editing, Supervision. **Fusui Ji:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e34179.

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