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ORIGINAL ARTICLE

Impact of intravascular ultrasound-guided minimum-contrast coronary intervention on 1-year clinical outcomes in patients with stage 4 or 5 advanced chronic kidney disease

Katsuaki Sakai[1] · Yuji Ikari[1] · Mamoru Nanasato[2] · Hiroshi Umetsu[3] · Masaaki Okutsu[4] · Tomonobu Takikawa[5] · Satoru Sumitsuji[6] · Kenji Sadamatsu[7] · Masanori Takada[8] · Yasuko Kato[2] · Nobuyuki Ogasawara[9] · Kanichi Otowa[10]

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Abstract

This study aims to elucidate 1-year clinical outcomes using this technique for patients with stage 4 or 5 advanced chronic kidney disease (CKD). Research has proven that imaging-guided percutaneous coronary intervention (PCI) reduces contrast volume significantly; however, only short-term clinical benefits have been reported. Minimum-contrast (MINICON) studies are based on the registry design pattern to enroll PCI results in patients with advanced CKD stage 4 or 5 comorbid with coronary artery disease. We excluded cases of emergency PCI or maintenance dialysis from this study. In this study, we compared the intravascular ultrasound (IVUS)-guided MINICON PCI group ($n=98$) with the angiography-guided standard PCI group ($n=86$). Enrollment of the MINICON studies started in 2006. Before 2012, IVUS-guided MINICON PCI was performed only in 14% (stage 1), but it was 100% after 2012 (stage 2). The enrollment finished in 2016. The IVUS-guided MINICON PCI group exhibited a significantly reduced contrast volume (22 ± 20 vs. 130 ± 105 mL; $P<0.0001$) and contrast-induced acute kidney injury (CI-AKI; 2% vs. 15%; $P=0.001$). The PCI success rate was similarly high (100% vs. 99%; $P=0.35$). At 1 year (follow-up rate, 100%), we observed less induction of renal replacement therapy (RRT; 2.7% vs. 13.6%; $P=0.01$), but all-cause mortality or myocardial infarction was similar in both groups. The IVUS-guided MINICON PCI reduces CI-AKI significantly and induction of RRT at 1 year in patients with stage 4 or 5 advanced CKD.

Keywords Coronary artery disease · Renal failure · Prognosis

✉ Yuji Ikari
ikari@is.icc.u-tokai.ac.jp

1 Department of Cardiology, Tokai University School of Medicine, 143 Shimokasuya, Isehara, Kanagawa 259-1193, Japan

2 Cardiovascular Center, Nagoya Daini Red Cross Hospital, 2-9 Myoukencho, Shouwaku, Nagoya 466-8650, Japan

3 Seirei Fuji Hospital, 3-1 Minamicho, Fuji, Shizuoka 417-0026, Japan

4 Nozaki Tokushukai Hospital, 2-10-50 Tanigawa, Daito, Osaka 574-0074, Japan

5 Kasugai Municipal Hospital, 1-1 Takakicho, Kasugai, Aichi 486-8510, Japan

6 Osaka University Graduate School of Medicine, 2-15 Yamadaoka, Suita, Osaka 565-0871, Japan

7 Saga-ken Medical Centre Koseikan, 400 Kasemachinakabaru, Saga City, Saga 840-0861, Japan

8 Medical Corporation Kawasaki Hospital, 3-3-1 Higashiyamacho, Hyogoku, Kobe, Hyogo 652-0042, Japan

9 Japan Community Health Care Organization Osaka Hospital, 4-2, Fukushima, Fukusimaku, Osaka, Japan

10 Municipal Tsuruga Hospital, 1-6-60, mishimacho, Tsuruga City, Fukui, Japan

Introduction

Contrast-induced acute kidney injury (CI-AKI) is a percutaneous coronary intervention (PCI)-related complication that has been associated with increased mortality and the requirement for renal replacement therapy (RRT) [1, 2]. The established modalities to prevent CI-AKI are peri-procedural hydration [3, 4], statin use [5], and contrast volume reduction [6, 7]. Despite these CI-AKI prevention strategies, PCI in patients with advanced chronic kidney disease (CKD) remains related to a high risk of CI-AKI and requirement for RRT, resulting in under-utilization of PCI in these high-risk patients.

Reportedly, the use of imaging and physiology guidance in PCI significantly reduces the contrast volume to almost zero and decreases the incidence of CI-AKI [8]. However, CI-AKI is a surrogate marker. The hard endpoints such as mortality or induction of renal replacement of imaging and physiology-guided PCI remain unknown. Thus, this study aims to investigate the hard endpoints of intravascular ultrasound (IVUS)-guided PCI using minimized-contrast volume for stage 4 or 5 advanced CKD at 1 year.

Methods

Study design

The study design of MINICON studies comprises a prospective, multicentre, non-randomized, open-label registry trial design in patients with coronary artery disease comorbid with advanced CKD stage 4 or 5 excluding maintenance haemodialysis. The MINICON study started in 2006. This first stage was called as MINICON-1 study ($n=100$) which was a registry design to enroll PCI for patients with CKD stage 4 ($n=77$) or 5 ($n=23$) including angiography-guided ($n=86$) and IVUS-guided PCI ($n=14$). After 2012, PCI operators attempted to perform IVUS-guided MINICON PCI using the minimized-contrast volume for all the patients. From April 2012 to June 2016, we enrolled 84 patients with stage 4 ($n=71$) or 5 ($n=13$) CKD for this MINICON-2 study from 13 institutions.

The inclusion criteria were coronary artery disease requiring elective PCI comorbid with high-risk CKD defined as a severe reduction of the estimated glomerular filtration rate (eGFR) as <30 mL/min/1.73 m^2 (CKD stage 4 or 5). In contrast, the exclusion criteria were the maintenance of haemodialysis, insufficient renal protection without saline infusion, planned surgery within 1 year that could deteriorate renal function, postrenal transplantation, and no indication of PCI.

We compared the IVUS-guided MINICON PCI group ($n=98$) with the angiography-guided standard PCI group ($n=86$).

This study was approved by the local Ethics Committee and was conducted per the guidelines of the Declaration of Helsinki.

Technique of IVUS-guided MINICON PCI

Apparently, IVUS-guided minimum-contrast PCI is entirely different from the standard PCI technique merely using IVUS, as the use of angiography is prohibited during the PCI procedure [9]. Based on the diagnostic coronary angiography performed on a different day, a guidewire is advanced without contrast. Both the stent size and length are determined depending on IVUS findings to assess the lesion length and arterial diameter. Then, IVUS-marking technique is performed to ascertain the stent position, and stent expansion and dissection at the stent edges are verified by IVUS. Although zero-contrast PCI is feasible, final single angiography confirms no distal coronary artery injury by a guidewire, because the detection of any distal complication is impossible by IVUS [8]. Hence, this technique is termed ‘minimum-contrast (MINICON)’ and not zero-contrast PCI. Although the MINICON PCI procedure requires only a few millilitre of contrast, a little more might be required in cases of complications. In any case, operators attempted to attain the cut-off contrast dose defined as contrast volume less than the eGFR value of patients based on the previous MINICON-1 study [10].

At 12 h before PCI, the administration of prophylactic infusion with saline or isotonic sodium bicarbonate solution was initiated, continuing even 12 h after PCI. Furthermore, the coronary flow on angiography was determined on the definition of the thrombolysis in myocardial infarction flow grade. Furthermore, complex lesions were defined as type lesions per the American Heart Association classification.

Assessment of renal function and CI-AKI

In this study, the eGFR was evaluated using the Japanese Modification of Diet in Renal Disease study equation reported by the Japanese Society of Nephrology [11]. The eGFR was estimated from serum creatinine, age, and sex using the new Japanese equation as follows: eGFR (mL/min/1.73 m^2) $= 194 \times$ serum creatinine$^{-1.094} \times$ age $- 0.287$ ($\times 0.739$ for female subjects).

Then, CKD stages were categorized according to the 2012 Kidney Disease Improving Global Outcome guidelines [12]. Notably, stage 4 CKD is when the eGFR is between 15 and 29 mL/min/1.73 m^2, and stage 5 CKD is eGFR <15 mL/min/1.73 m^2.

Table 1 Patients' characteristics

	Angiography-guided PCI ($n=86$)	IVUS-guided PCI ($n=98$)	P
Age (years)	74 ± 7	76 ± 9	0.22
Body height (cm)	159 ± 10	160 ± 9	0.62
Body weight (kg)	60 ± 13	61 ± 12	0.58
Male	53 (62%)	77 (79%)	0.01
Smoking			0.90
Never smoked	38 (45%)	42 (45%)	
Ex-smoker	36 (42%)	41 (44%)	
Current smoker	11 (13%)	10 (11%)	
Diabetes mellitus			0.12
None	45 (52%)	38 (39%)	
Diet only	5 (6%)	15 (15%)	
Oral anti-diabetic agent	19 (22%)	24 (24%)	
Insulin user	17 (20%)	21 (21%)	
Dyslipidemia	60 (71%)	67 (49%)	0.82
Hypertension	79 (92%)	88 (90%)	0.63
Family history	6 (7%)	10 (11%)	0.38
Prior MI	29 (34%)	34 (35%)	0.89
Prior PCI	40 (47%)	57 (58%)	0.11
Prior CABG	7 (8%)	5 (5%)	0.41
Prior HF	26 (30%)	36 (37%)	0.35
Prior stroke	14 (16%)	17 (17%)	0.85
Unprotected left main trunk	8 (9%)	9 (9%)	0.98
Number of vessels diseased			0.54
1	28 (33%)	39 (40%)	
2	33 (38%)	36 (37%)	
3	25 (29%)	23 (23%)	
Primary clinical diagnosis			0.002
Angina pectoris	60 (70%)	54 (55%)	
Old myocardial infarction	13 (15%)	6 (6%)	
Silent myocardial ischaemia	11 (13%)	33 (34%)	
Other	2 (2%)	5 (5%)	
Canadian Cardiovascular Society functional classification (CCS)			0.0002
CCS1	15 (17%)	17 (17%)	
CCS2	30 (35%)	36 (37%)	
CCS3	20 (23%)	7 (7%)	
CCS4	6 (7%)	0	
None	15 (17%)	38 (39%)	
LVEF			0.42
< 30%	4 (5%)	8 (8%)	
30–50%	15 (18%)	23 (23%)	
> 50%	63 (77%)	67 (68%)	
EuroSCORE II (%)	2.8 ± 2.0	3.5 ± 2.6	0.06
ACEI or ARB	66 (77%)	68 (69%)	0.26
Haemoglobin (mg/dL)	10.5 ± 1.7	11.3 ± 1.8	0.002
Creatinine (mg/dL)	2.3 ± 1.0	2.0 ± 0.6	0.008
eGFR (mL/min/1.73 m^2)	19 ± 6	21 ± 6	0.01
CKD stage 4/5	65 (76%)/21 (24%)	83 (85%)/15 (15%)	0.12
BNP	213 (IQR 59–464)	151 (IQR 66–342)	0.75
Proteinuria			0.79
(−)	21 (31%)	22 (26%)	

Table 1 (continued)

	Angiography-guided PCI ($n=86$)	IVUS-guided PCI ($n=98$)	*P*
(±)	9 (13%)	17 (20%)	
(1+)	16 (24%)	20 (24%)	
(2+)	12 (18%)	12 (14%)	
(3+ or 4+)	10 (15%)	14 (16%)	

IVUS intravascular ultrasound, *PCI* percutaneous coronary intervention, *MI* myocardial infarction, *CABG* coronary artery bypass graft, *HF* heart failure, *LVEF* left ventricular ejection fraction, *ACEI* angiotensin-converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *eGFR* estimated glomerular filtration rate, *BNP* B-type natriuretic peptide, *IQR* interquartile range

Furthermore, CI-AKI was defined as an absolute increase of, at least, 0.5 mg/dL or a relative S–Cr increase of 25% over the baseline serum creatinine level 48 h after PCI.

Endpoints

The primary endpoint of this study was a composite endpoint of all-cause mortality, myocardial infarction, and induction of RRT at 1 year following PCI. Moreover, the secondary endpoints were contrast dose, the incidence of CI-AKI after PCI, or cardiac death, stroke, myocardial infarction, and any additional coronary revascularization, including PCI or coronary artery bypass graft, at 1 year.

Statistical analysis

In this study, continuous variables are presented as mean ± standard deviation or median and interquartile range for skewed data. In contrast, categorical variables are expressed as numbers and percentages. We performed the Pearson's bivariate test and the χ^2 test to compare the groups for category covariates and the Student's *t* test for continuous covariates. Furthermore, the Kaplan–Meier method was used to compare 1-year clinical outcomes, including all-cause mortality, cardiac death, non-cardiac death, or requirement of RRT. The event-free survival rate differences in each group were compared using the Wilcoxon test. We performed all statistical analyses using the SAS ver 9.4 or JMP software, version 12.0.1 (SAS Institute Inc., Cary, NC).

Results

Patient characteristics

Table 1 summarises the baseline patients' characteristics. In the IVUS-guided MINICON PCI group, the average age of patients was 76 ± 9 years, 79% were males, and 61% were patients with diabetes mellitus. Of note, patients' backgrounds were typically similar, except for a more silent myocardial ischaemia in IVUS-guided group and a small difference of the eGFR (19 ± 6 in angiography-guided vs. 21 ± 6 in IVUS-guided groups; $P=0.01$). However, the rate of stage 4 and 5 CKD exhibited no statistically significant difference.

PCI procedure

In the IVUS-guided MINICON PCI group, 58% of patients received treatment per the transradial approach, and PCI was performed by primarily using a 6-Fr-guiding catheter (87%; Table 2). The IVUS-guided group comprised a higher number of American Heart Association/College of American Cardiology (AHA/ACC) type C lesions ($P=0.006$); however, the complication rate was less in the IVUS-guided MINICON PCI group (12% vs. 2%; $P=0.009$). The angiography-guided group reported five side-branch occlusions, two guidewire perforations, and three access site bleedings. Conversely, the IVUS-guided group reported one side-branch occlusion, one slow flow and one low blood pressure because of the vagal reflex. Nonetheless, the PCI success rate was similarly high (100% vs. 99%; $P=0.35$).

Contrast dye volume and CI-AKI

The histogram in Fig. 1 shows the distribution of the contrast volume, which in the IVUS-guided MINICON PCI group was significantly less compared with the angiography-guided standard PCI group (22 ± 20 vs. 130 ± 105 mL; $P=0.001$). All patients in the IVUS-guided MINICON PCI group attempted to attain the contrast volume less than the eGFR value (achievement rate, 78%). Furthermore, CI-AKI was observed in the standard angiography-guided or IVUS-guided MINICON PCI groups as 15% and 2%, respectively ($P=0.001$; Fig. 2).

Clinical outcomes at 1 year

In this study, the follow-up rate was 100%. Moreover, the contrast was used for 35 patients in the IVUS-guided

Table 2 Procedure characteristics

	Angiography-guided PCI ($n=86$)	IVUS-guided PCI ($n=98$)	*P*
Approach site			0.06
Radial	46 (53%)	57 (58%)	
Brachial	8 (9%)	18 (18%)	
Femoral	32 (37%)	23 (23%)	
Guiding catheter size (Fr)			0.008
5	4 (5%)	6 (6%)	
6	62 (72%)	85 (87%)	
7	19 (22%)	5 (5%)	
8	1 (1%)	2 (2%)	
Guiding catheter shape			0.02
Judkins	25 (29%)	18 (18%)	
Ikari	23 (27%)	22 (22%)	
Voda/EBU/XB	27 (31%)	27 (28%)	
Amplatz or other	11 (13%)	31 (32%)	
Target vessel			0.39
LAD	38 (44%)	43 (44%)	
LCX	10 (12%)	19 (19%)	
RCA	30 (35%)	31 (32%)	
LM	8 (9%)	5 (5%)	
ACC/AHA lesion type			0.006
A	15 (17%)	7 (7%)	
B1	18 (21%)	29 (30%)	
B2	40 (47%)	32 (33%)	
C	13 (15%)	30 (31%)	
Pre-TIMI			0.11
TIMI 0 (CTO)	6 (7%)	16 (16%)	
TIMI 1	3 (3%)	5 (5%)	
TIMI 2	7 (8%)	3 (3%)	
TIMI 3	70 (81%)	74 (76%)	
Post-TIMI			0.49
TIMI 2	2 (2%)	1 (1%)	
TIMI 3	84 (98%)	97 (99%)	
IABP supported	4 (5%)	0	0.03
Complication	10 (12%)	2 (2%)	0.009
Slow flow/No flow	0	1 (1%)	0.35
Procedure Success	86 (100%)	97 (99%)	0.35
Kind of contrast dye			0.0002
Iopamidol	46 (61%)	45 (46%)	
Iomeprol	23 (30%)	16 (16%)	
Iohexol	5 (7%)	19 (19%)	
Not assessed (unreported)	2 (3%)	18 (18%)	
Dye volume (mL)	130 ± 105	22 ± 20	<0.0001
Dye volume/eGFR <1	0	77(78%)	<0.0001
Fluoroscopy time (min)	32 (IQR 21–42)	24 (IQR 13–38)	0.96
Intravenous sodium bicarbonate	15 (17%)	22 (22%)	0.40
N-acetylcysteine	0	3 (3%)	0.10
HD after PCI	6 (7%)	0	0.008
hANP	2 (2%)	2 (2%)	0.89
CI-AKI	13 (15%)	2 (2%)	0.001

IVUS intravascular ultrasound, *PCI* percutaneous coronary intervention, *LAD* left anterior descending artery, *LCX* left circumflex artery, *RCA* right coronary artery, *LM* left main, *ACC* American College of Cardiology, *AHA* American Heart Association, *TIMI* thrombolysis in myocardial infarction, *CTO* chronic total occlusion, *IABP* intra-aortic balloon pump, *HD* haemodialysis, *hANP* human A-type natriuretic peptide, *CI-AKI* contrast-induced acute kidney injury

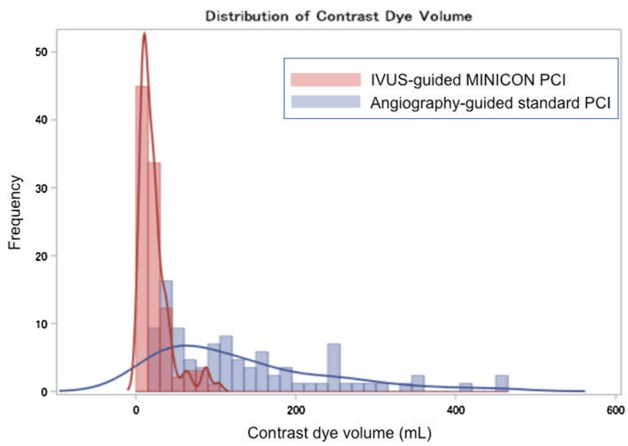

Fig. 1 Distribution of the contrast dye volume. The histogram shows the distribution of the contrast dye volume. The average of the contrast volume in angiography-guided (blue) and intravascular ultrasound-guided minimum-contrast percutaneous coronary intervention (red) was 130 ± 105 and 22 ± 20 mL, respectively ($P = 0.001$)

MINICON PCI group during the follow-up for 16 patients undergoing coronary angiography, 13 PCI, 2 endovascular treatment, 3 computed tomography, and 1 ablation. The average contrast volume during the follow-up was 18 ± 19 mL.

Table 3 shows 1-year clinical outcomes. The induction of RRT within 1 year showed clear difference between the two groups (2.7% vs. 13.6%; $P = 0.01$). The composite endpoint of all-cause mortality, myocardial infarction, and RRT exhibited a similar beneficial tendency of the IVUS-guided group, but did not reach statistical significance (10.7% vs. 19.9%, $P = 0.09$, Fig. 3). However, the IVUS-guided MINICON PCI had less 1-year events in all the observed endpoints.

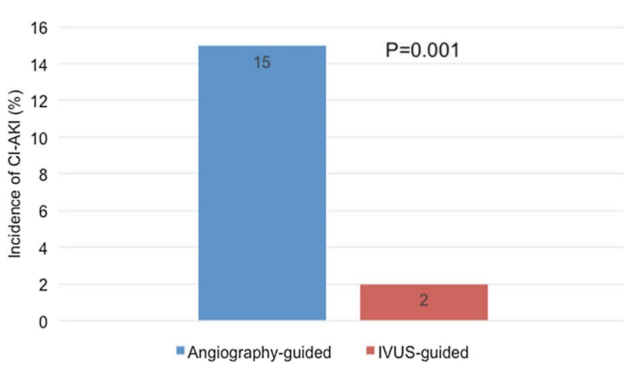

Fig. 2 Incidence of contrast-induced acute kidney injury (CI-AKI). The incidence of CI-AKI is shown in angiography-guided standard percutaneous coronary intervention (PCI) group (blue) and intravascular ultrasound-guided minimum-contrast PCI. Definition of CI-AKI is shown in "Methods"

Table 3 1-year clinical outcomes between angiography-guided and IVUS-guided PCI

	Angiography-guided PCI ($n = 86$)	IVUS-guided PCI ($n = 98$)	*P* value
All-cause death	6 (7.8%)	6 (6.4%)	0.85
Cardiac death	2 (2.6%)	2 (2.2%)	0.98
Non-cardiac death	4 (5.3%)	4 (4.4%)	0.79
RRT	11 (13.6%)	3 (3.2%)	0.01
MI	2 (2.4%)	0 (0%)	0.13
Death or MI	7 (8.9%)	6 (6.4%)	0.85
Death or RRT	16 (19.9%)	9 (9.5%)	0.08
Death, MI or RRT	16 (19.9%)	9 (9.5%)	0.09

IVUS intravascular ultrasound, *PCI* percutaneous coronary intervention, *HD* haemodialysis, *MI* myocardial infarction, *RRT* renal replacement therapy

All percentages were estimated % calculated by Kaplan–Meier method

Discussion

This study demonstrated that IVUS-guided MINICON PCI reduced the contrast volume, incidence of CI-AKI, and induction of RRT at 1 year compared with the standard angiography-guided PCI. Since the established modalities to prevent CI-AKI are peri-procedural hydration [3, 4], all the investigators performed peri-procedural hydration for all the cases without any exclusion.

Even though RRT was significantly less in IVUS-guided group, the primary endpoint as all-cause mortality, MI, and induction of RRT did not reach statistical significance (19.9% vs. 10.7%, $P = 0.07$). It is probably because of low statistical power, since all the endpoints were less in IVUS-guided group. Patients with stage 4 (eGFR < 30 mL/min/1.73 m^2) or stage 5 (eGFR < 15 mL/min/1.73 m^2) advanced CKD are known to have higher annual all-cause mortality (11.36% vs. 14.14%, respectively) [13]. Furthermore, age-standardized cardiovascular events of stage 4 or 5 CKD were 21.80% or 36.60% annually [13]. In this study, 1-year mortality rate in angiography-guided and IVUS-guided MINICON PCI groups was 7.8% vs 6.4%, which has lower mortality rate compared with historical control. This study may also have confirmed the reported better survival of revascularization [14, 15] in patients with stage 4 or 5 CKD [16, 17].

Research has reported a larger contrast volume to be a factor for CI-AKI, and the reduction of contrast volume decreased the incidence of CI-AKI [18]. A new imaging-guided PCI technique minimized the contrast volume to almost zero [8]. Ali et al. reported the treatment of 31 patients with advanced CKD by imaging and physiology-guided minimum-contrast PCI, attaining the contrast

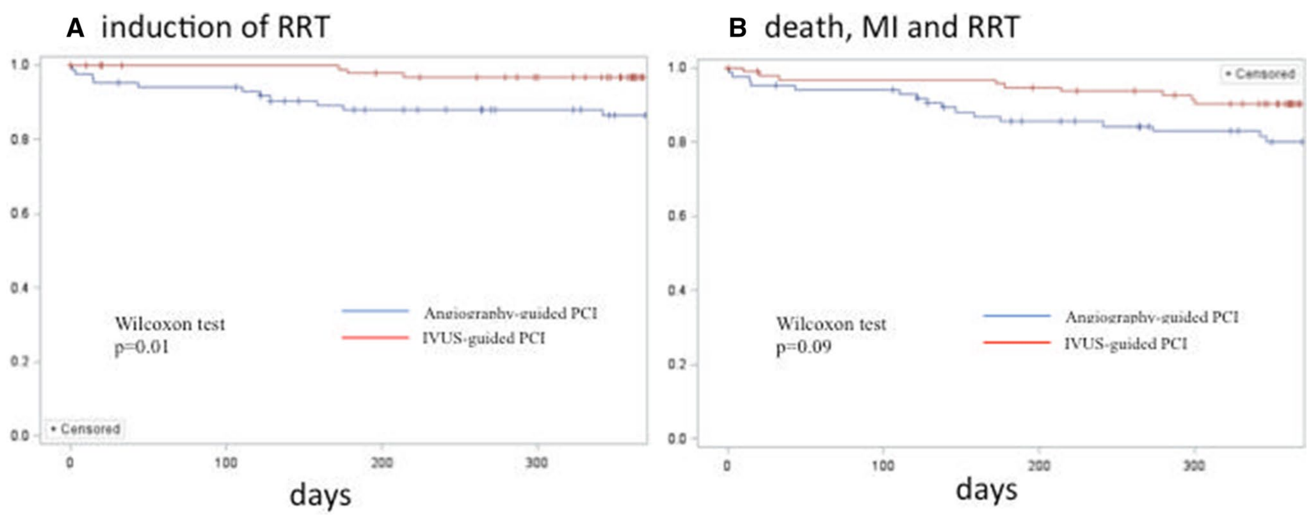

Fig. 3 Kaplan–Meier event-free survival curves to 1 year. The Kaplan–Meier event-free survival curves of the induction of renal replacement therapy (RRT; **a**) and a composite endpoint of all-cause mortality, myocardial infarction, and induction of RRT (**b**). Blue line, angiography-guided standard percutaneous coronary intervention (PCI); red line, intravascular ultrasound-guided minimum-contrast PCI

volume/eGFR < 1 in all patients without major cardiac events or need for RRT within a follow-up time of 79 days [8]. However, these studies did not include hard endpoints. In this study, we included 98 patients with stage 4 or 5 advanced CKD and followed up to 1 year with 100% follow-up rate. To the best of our knowledge, this is the first study to report the clinical benefits of MINICON PCI at 1 year following PCI.

As IVUS completely replaces angiography in the IVUS-guided MINICON PCI, it is different from the standard PCI technique. Thus, expertise in IVUS is imperative. Moreover, IVUS is superior to angiography in the assessment of vessel diameter, plaque volume, and dissection at stent edges. However, stent positioning is complicated without angiography even with IVUS. Furthermore, a critical drawback of IVUS is that prompt detection of complications, such as coronary slow flow/no reflow, dissection, and coronary perforation by stents or guidewires, is difficult. Any delay in the detection of complications might induce serious adverse events. Although no severe complications were reported in this study, safety concerns are potential limitations of this technique. Hence, carefully using the PCI technique through the IVUS-guided MINICON PCI is essential.

This study has several limitations. First, the study is not randomized. Thus, selection bias cannot be ignored. However, it is ethically challenging to conduct randomized research for these very high-risk groups of patients. Second, this study analyzed data from two periods, which were conducted in the same institution but with different enrolment timepoints; this might be another bias in this study. Third, this study did not include a control group without PCI. If the control group without PCI was included, significance of mortality rate might be clearly observed. Finally, the sample size was small. However, prior studies have included even smaller number patients to assess the efficacy of PCI for stage 4 or 5 CKD. In comparison with the literature, this study included a significant number of extremely high-risk patients.

In conclusion, IVUS-guided MINICON PCI significantly reduces contrast volume and CI-AKI and exhibits benefits after 1 year of PCI. Technique of IVUS-guided MINICON PCI is difficult; however, it should be considered for patients with advanced CKD.

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References

1. McCullough PA. Contrast-induced acute kidney injury. J Am Coll Cardiol. 2008;51:1419–28.
2. Giacoppo D, Madhavan MV, Baber U, Warren J, Bansilal S, Witzenbichler B, et al. Impact of contrast-induced acute kidney injury after percutaneous coronary intervention on short- and long-term outcomes: pooled analysis from the HORIZONS-AMI and ACUITY trials. Circ Cardiovasc Interv. 2015;8:e002475.
3. Brar SS, Aharonian V, Mansukhani P, Moore N, Shen AY, Jorgensen M, et al. Haemodynamic-guided fluid administration for the prevention of contrast-induced acute kidney injury: the POSEIDON randomized controlled trial. Lancet. 2014;383:1814–23.
4. Stacul F, Adam A, Becker CR, Davidson C, Lameire N, McCullough PA, et al. Strategies to reduce the risk of contrast-induced nephropathy. Am J Cardiol. 2006;98:59K–77K.
5. Giacoppo D, Gargiulo G, Buccheri S, Aruta P, Byrne RA, Cassese S, et al. Preventive strategies for contrast-induced acute kidney injury in patients undergoing percutaneous coronary procedures: evidence from a hierarchical Bayesian network meta-analysis of 124 trials and 28 240 patients. Circ Cardiovasc Interv. 2017;10:e004383.
6. Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol. 2004;44:1393–9.
7. Nyman U, Bjork J, Aspelin P, Marenzi G. Contrast medium dose-to-GFR ratio: a measure of systemic exposure to predict contrast-induced nephropathy after percutaneous coronary intervention. Acta Radiol. 2008;49:658–67.
8. Ali ZA, Karimi Galougahi K, Nazif T, Maehara A, Hardy MA, Cohen DJ, et al. Imaging- and physiology-guided percutaneous coronary intervention without contrast administration in advanced renal failure: a feasibility, safety, and outcome study. Eur Heart J. 2016;37:3090–5.
9. Ogata N, Matsukage T, Toda E, Tamiya S, Fujii T, Nakazawa G, et al. Intravascular ultrasound-guided percutaneous coronary interventions with minimum contrast volume for prevention of the radiocontrast-induced nephropathy: report of two cases. Cardiovasc Interv Ther. 2011;26:83–8.
10. Ogata N, Ikari Y, Nanasato M, Okutsu M, Kametani R, Abe M, et al. Safety margin of minimized contrast volume during percutaneous coronary intervention in patients with chronic kidney disease. Cardiovasc Interv Ther. 2014;29:209–15.
11. Matsuo S, Imai E, Horio M, Yasuda Y, Tomita K, Nitta K, et al. Revized equations for estimated GFR from serum creatinine in Japan. Am J Kidney Dis. 2009;53:982–92.
12. KDIGO. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int Suppl. 2012;2013(3):1–150.
13. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. N Engl J Med. 2004;351:1296–305.
14. Reddan DN, Szczech LA, Tuttle RH, Shaw LK, Jones RH, Schwab SJ, et al. Chronic kidney disease, mortality, and treatment strategies among patients with clinically significant coronary artery disease. J Am Soc Nephrol. 2003;14:2373–80.
15. Hemmelgarn BR, Southern D, Culleton BF, Mitchell LB, Knudtson ML, Ghali WA, et al. Survival after coronary revascularization among patients with kidney disease. Circulation. 2004;110:1890–5.
16. Tsai TT, Patel UD, Chang TI, Kennedy KF, Masoudi FA, Matheny ME, et al. Contemporary incidence, predictors, and outcomes of acute kidney injury in patients undergoing percutaneous coronary interventions: insights from the NCDR Cath-PCI registry. JACC Cardiovasc Interv. 2014;7:1–9.
17. Dangas G, Iakovou I, Nikolsky E, Aymong ED, Mintz GS, Kipshidze NN, et al. Contrast-induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. Am J Cardiol. 2005;95:13–9.
18. Gurm HS, Dixon SR, Smith DE, Share D, Lalonde T, Greenbaum A, et al. Renal function-based contrast dosing to define safe limits of radiographic contrast media in patients undergoing percutaneous coronary interventions. J Am Coll Cardiol. 2011;58:907–14.