Cardiovascular Topics

Is the transradial approach associated with decreased acute kidney injury following percutaneous coronary intervention in patients not complicated by major bleeding and haemodynamic disturbance?

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Abstract

Background: The impact of the transradial approach (TRA) on the development of acute kidney injury (AKI) after percutaneous coronary interventions (PCI) has been controversial. Methods: We retrospectively analysed 463 patients undergoing PCI for either acute or chronic coronary syndrome. Excluded patients were those with missing laboratory or procedural data, acute/decompensated heart failure, major bleeding, haemodynamic instability, long-term dialysis and mortality. The primary endpoint of the study was the incidence of AKI after PCI, which was defined as an increase in serum creatinine (SCr) level of 0.5 mg/dl or 25% from the baseline. Secondary endpoints were change in SCr level, increase in SCr of ≥ 0.3 and ≥ 0.5 mg/dl, and increase in SCr of ≥ 25 and \geq 50%. We compared the incidence of AKI between the TRA and the transfemoral approach (TFA) in the overall and a propensity score (PS)-matched study population.

Results: The study population included 339 patients. After PS matching, we obtained a well-balanced population of 182 patients. The differences between the incidence of AKI in the TRA and TFA were not significant in both the overall (9.0 vs 11.2%, p = 0.503) and PS-matched (9.9 vs 7.7%, p = 0.601) study population. TRA resulted in a significantly lower incidence of SCr increase of $\geq 50\%$ in unmatched patients. However, after PS matching, there was no difference between the TRA and TFA in any variable of secondary post-PCI renal outcomes. Age, female gender, baseline SCr level, baseline estimated glomerular filtration rate and contrast volume were independent predictors of AKI.

Conclusion: Compared to the conventional TFA, TRA was not associated with a reduced incidence of AKI after PCI in patients not complicated by major bleeding, acute heart failure and haemodynamic disturbances.

Keywords: acute kidney injury, contrast-induced nephropathy, percutaneous coronary intervention, transradial approach

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Acute kidney injury (AKI) following cardiac intervention is associated with a poor short- and long-term prognosis. Depending on the definition and clinical setting, AKI can be observed in up to 25% of patients after cardiac interventions.^{1,2} Established clinical risk factors for AKI after cardiac procedures included patient-related risk factors (older age, chronic renal failure, diabetes, anaemia, heart failure, dehydration and use of nephrotoxic drugs) and procedure-related risk factors (contrast volume, choice of contrast agent and repeat procedure within 48–72 hours).³⁻⁵

Recently, it has been proposed that the transradial approach (TRA) might have a protective role against AKI.⁶⁹ Aside from the usual procedural risk factors, atheroembolism from the abdominal aorta to the renal arteries is thought to be responsible for AKI during the transfemoral approach (TFA), as the abdominal aorta is not catheterised during radial procedures. However, the data have not been clear-cut so far.

There are conflicting results on the benefit of the TRA on AKI,¹⁰⁻¹⁴ particularly when there is a strong negative relationship between TRA and bleeding events after percutaneous coronary interventions (PCI).^{11,12} To some extent, this might explain the lower incidence of AKI after a transradial intervention. In this article, we aimed to analyse our data to see whether the TRA had an advantage over TFA in reducing AKI following PCI.

Methods

Consecutive patients undergoing PCI between July 2016 and April 2018 in a tertiary centre were analysed retrospectively in this study. Both elective procedures and procedures for acute coronary syndromes (ACS) were included. Demographic, laboratory and clinical data were obtained from hospital records. The study was approved by the local institutional ethics committee.

We opted to exclude major complications of ACS and/or PCI so that haemodynamics did not play a central role in the development of AKI. Patients presenting with haemodynamic instability or cardiogenic shock were excluded, as haemodynamic deterioration worsens renal perfusion and function, even without a PCI procedure. Systolic blood pressure below 90 mmHg persisting for more than 30 minutes was considered as haemodynamic instability.

Patients with acute or decompensated heart failure were excluded. Similar to cardiogenic shock, the rapid development of heart failure in ACS causes renal malperfusion and dysfunction independent of intervention. Acute heart failure was diagnosed clinically by the attending cardiologist. Rapid onset or deterioration of previous symptoms and/or signs of heart failure were considered acute heart failure.

As a known determinant of AKI, patients with major bleeding were excluded as well. Major bleeding was defined as > 3 g/l drop in haemoglobin level, and bleeding requiring transfusion or surgery. Patients who died immediately after (< 48 hours) PCI was not included either.

Patients with missing baseline or follow-up laboratory or procedural data were excluded. Hospital records were used to detect AKI after the index procedure. Follow-up laboratory was defined as either in-hospital (generally for ACS patients) or short-term visit (generally for elective procedures). The estimated glomerular filtration rate (eGFR) was calculated according to the simplified modification of diet in renal disease formula.¹⁵ Lastly, patients on long-term dialysis were excluded as the diagnosis of AKI is not applicable.

As per institutional protocol, patients with chronic renal failure (eGFR < 60 ml/h/1.73 m²), ACS, and over 70 years old were hydrated with normal saline, starting before the procedure. The usual protocol was 1 ml/kg/h of normal saline infused for six hours before, and 0.5 ml/kg/h for 12 hours after a procedure. Access site (radial or femoral) and size of catheters (6F or 7F) were at the operators' discretion. Low osmolar, non-ionic iohexol was used exclusively at the time of the study. *Ad hoc* interventions were considered as one procedure. Contrast volume and procedure time were calculated from the beginning of diagnostic angiography. Procedural anticoagulation was done with unfractionated heparin 50–70 U/kg, as per institutional protocol. Target vessels, procedure time, contrast volume, procedural complications and post-procedural deaths were recorded.

The primary endpoint of the study was the development of AKI after PCI, which was defined as an increase in serum creatinine (SCr) level of 0.5 mg/dl or 25% from the baseline. Secondary endpoints were absolute (mg/dl) or proportional (%) changes in SCr level, an increase in SCr level of > 0.3 and > 0.5 mg/dl, and an increase in SCr level of > 25 and > 50%.

Statistical analysis

SPSS Statistics for Windows, version 22.0 (Armonk, NY: IBM Corp) was used for the statistical analysis. Continuous variables are presented as means and standard deviations (SD) or medians and interquartile ranges (IQR), according to the normality of distribution. Comparison for continuous variables between the radial and femoral groups were performed either with the *t*-test or Mann–Whitney *U*-test, as appropriate. Categorical data are presented as numbers and percentages, and compared using chi-squared or Fisher's exact test, as appropriate.

A propensity score (PS) analysis was performed to alleviate

the selection bias and other clinical imbalances between the radial and femoral groups due to demographic, laboratory, clinical and procedural characteristics. PS matching was performed using the R-essentials plugin in SPSS Statistics for Windows. The nearest neighbour 1:1 matching with a caliper of 0.1 was performed.

The radial and femoral groups were matched for these variables: age, gender, hypertension, diabetes, history of coronary intervention (PCI and/or coronary artery bypass graft), baseline serum eGFR, white blood cells, haemoglobin, blood cholesterol, clinical presentation (elective or ACS), contrast volume and treated vessel [left anterior descending artery (LAD), circumflex artery (CX), right coronary artery (RCA)]. In addition, independent predictors of AKI following PCI were determined using logistic regression analysis; the model included vascular access site in addition to the aforementioned variables. Odds ratios (OR) and 95% confidence intervals (CI) were reported. Two-sided p < 0.05 was considered significant.

Results

A total of 463 patients were identified. We excluded 124 patients with missing laboratory or procedural data (n = 96), acute/ decompensated heart failure (n = 8), major bleeding (n = 5), haemodynamic instability (n = 3), long-term dialysis (n = 6) and mortality (n = 6), and the remaining 339 patients made up our study population. The radial group included 134 patients (40%) and the femoral group comprised 205 patients (60%).

A total of seven (2.1%) access site crossovers was observed. The reasons for a radial-to-femoral crossover were failure to access the radial artery in three patients (2.2%) and severe tortuosity of the brachiocephalic artery in two patients (1.5%). A femoral-to-radial crossover was needed in severe iliac tortuosity in two patients (1%). Procedures included in the radial or femoral groups according to the access site procedure were successfully carried out.

Demographic, laboratory and procedural data are presented in Table 1. There was no difference between the two groups in terms of baseline demographic characteristics. There was also no difference between the two groups in the laboratory data, except for white blood cell count, which was higher in the femoral group.

There were 110 patients (32%) undergoing elective PCI, 137 patients (40%) with non-ST segment elevation myocardial infarction (NSTEMI), and 92 patients (27%) with ST-segment elevation myocardial infarction (STEMI). The operators more frequently chose the TRA in elective procedures, whereas TFA was more common in STEMI. In NSTEMI, the rate of TRA and TFA were similar. *Ad hoc* and/or primary PCI were performed more commonly through the femoral route.

The distribution of target vessels was comparable between the groups. Procedure time and contrast volume used were also similar. Total failed procedures were five (1.5%) overall, one (0.7%) in the TRA and four (2.0%) in the TFA (p = 0.652). After PS matching, we had a well-balanced population of 182 patients (Table 1).

SCr level was controlled after the procedure at a median of three days (range two to seven days). According to our definition, AKI developed in 35 patients (10.3%) in the overall study population. The differences between the TRA and TFA in AKI incidence were not significant in both the overall (9.0 vs

Table 1. Baseline demographic, laboratory, clinical and procedural data									
		Overall study population PS-matched study po			population				
	Total	Radial	Femoral		Total	Radial	Femoral		
Baseline data	(n = 339)	(n = 134)	(n = 205)	p-value	(n = 182)	(n = 91)	(n = 91)	p-value	
Age, years	61.5 ± 11.4	61.6 ± 10.8	61.5 ± 11.8	0.913	61.1 ± 11.5	60.9 ± 11.2	61.4 ± 11.8	0.748	
Female gender, $n(\%)$	98 (29)	37 (28)	61 (30)	0.670	55 (30)	28 (31)	27 (30)	0.872	
Hypertension, n (%)	196 (58)	79 (59)	117 (57)	0.866	105 (58)	53 (58)	52 (57)	0.967	
Diabetes, $n(\%)$	53 (16)	20 (15)	33 (16)	0.903	29 (16)	14 (15)	15 (16)	0.923	
Active smoking, n (%)	84 (25)	31 (24)	53 (26)	0.776	46 (25)	25 (27)	21 (23)	0.707	
History of CABG, <i>n</i> (%)	17 (5.0)	7 (5.3)	10 (4.9)	0.8/4	9 (4.9)	4 (4.4)	5 (5.5)	1.000	
History of PCI, n (%)	49 (14.5)	21 (15.7)	28 (13.7)	0.606	28 (15)	10(11)	18 (20)	0.100	
History of heart failure, n (%)	15 (3.2)	4 (2.9)	11 (3.4)	0.765	6 (3.3)	2 (2.2)	4 (4.4)	0.172	
Capatining market	0.04 ± 26	0.01 ± 0.26	0.00 ± 0.20	0.120	0.04 ± 0.27	0.02 ± 0.25	0.00 ± 0.20	0.207	
Creatinine mg/dl	0.94 ±. 26	0.91 ± 0.26	0.96 ± 0.30	0.120	0.94 ± 0.27	0.92 ± 0.25	0.96 ± 0.30	0.297	
$eGFR m/n/1.73 m^2$	$8/\pm 24$	90 ± 23	85 ± 24	0.101	$8/\pm 25$	88 ± 23	85 ± 26	0.428	
$eGFR < 60 \text{ mi/n/1.73 m}^2, n (%)$	31(15)	18 (13)	33(10)	0.502	30(17)	15(17)	15(17)	1.000	
$eGFR < 30 \text{ m}/n/1.73 \text{ m}^2, n (\%)$	2(0.6)	0(0.7)	2(1.0)	0.521	1(0.5)	0(0.7)	1(1.1)	0.770	
wBC 107di	9.2 ± 3.2	8.0 ± 3.0	9.0 ± 3.2	.003	9.1 ± 3.0	9.0 ± 3.3	9.1 ± 2.0	0.770	
	13.7 ± 1.7	13.9 ±1.8	15.0 ± 1.05	0.170	15.7 ± 1.8	13.8 ± 1.8	13.0 ± 1.7	0.410	
Anaemia. $n (\%)$	74 (22)	29 (22)	43(23)	0.872	41(23)	20(22)	21(23)	0.859	
Tatal shalastaral mg/dl	234 ± 69	231 ± 03	230 ± 71	0.487	233 ± 73	234 ± 60	230 ± 83	0.882	
mmol/l	5.21 ± 1.30	5.28 ± 1.27	5.15 ± 1.32	0.555	5.13 ± 1.35	5.15 ± 1.30	5.10 ± 1.40	0.775	
LDL-C, mg/dl mmol/l	129 ± 47 3.34 ± 1.22	130 ± 52 3.37 ± 1.35	128 ± 44 3.32 ± 1.14	0.601	127 ± 53 3.29 ± 1.37	129 ± 59 3.34 ± 1.53	127 ± 47 3.29 ± 1.22	0.792	
HDL-C, mg/dl mmol/l	42 ± 19 1.09 ± 0.49	44 ± 22 1.14 ± 0.57	41 ± 17 1.06 ± 0.44	0.168	43 ± 20 1.11 ± 0.52	45 ± 25 1.17 ± 0.65	$41 \pm 15 \\ 1.06 \pm 0.39$	0.166	
TG, mg/dl mmol/l	138 (103–187) 1.56 (1.16–2.11)	143 (103–190) 1.62 (1.16–2.15)	135 (102–186) 1.53 (1.15–2.10)	0.594	136 (101–187) 1.54 (1.14–2.11)	132 (101–187) 1.49 (1.14–2.11)	139 (98–187) 1.57 (1.11–2.11)	0.592	
Medications									
Antiplatelets, n (%)	336 (99)	132 (99)	204 (100)	0.334	180 (99)	90 (99)	90 (99)	1.000	
Beta-blockers, n (%)	228 (68)	86 (65)	142 (70)	0.393	130 (72)	64 (71)	66 (73)	0.739	
RAS blockers, n (%)	156 (47)	51 (39)	105 (52)	0.025	87 (49)	41 (46)	46 (51)	0.551	
CCB, <i>n</i> (%)	14 (4.2)	1 (0.8)	13 (6.4)	0.012	4 (2.2)	1 (1.1)	3 (3.3)	0.621	
Diuretics, n (%)	32 (9.6)	12 (9.2)	20 (9.9)	0.834	16 (8.9)	8 (8.9)	8 (9.0)	0.981	
Statins, n (%)	220 (66)	80 (61)	140 (69)	0.155	122 (68)	61 (69)	61 (68)	0.913	
Clinical presentation									
Elective PCI, n (%)	110 (32)	62 (46)	48 (23)	0.001	54 (30)	27 (30)	27 (30)	1.000	
NSTEMI, n (%)	137 (40)	58 (43)	79 (39)	0.384	100 (55)	50 (55)	50 (55)	1.000	
STEMI, <i>n</i> (%)	92 (27)	14 (10)	78 (38)	0.001	28 (15)	14 (15)	14 (15)	1.000	
All ACS (%)	229 (68)	72 (54)	157 (77)	0.001	128 (70)	64 (70)	64 (70)	1.000	
Procedural findings									
Primary PCI, n (%)	104 (31)	17 (13)	87 (42)	0.001	39 (21)	17 (19)	22 (24)	.366	
Ad hoc PCI, n (%)	265 (78)	97 (72)	168 (82)	0.037	139 (76)	71 (78)	68 (75)	0.601	
Target vessel									
LMCA, <i>n</i> (%)	4 (1.2)	1 (0.7)	3 (1.5)	1.000	2 (1.1)	0 (0)	2 (2.2)	0.497	
LAD, <i>n</i> (%)	135 (40)	53 (40)	82 (40)	0.934	75 (41)	37 (41)	38 (42)	0.880	
CX, n (%)	79 (23)	31 (23)	48 (23)	0.952	46 (25)	19 (21)	27 (30)	0.172	
RCA, <i>n</i> (%)	140 (41)	53 (40)	87 (42)	0.598	72 (40)	38 (42)	34 (37)	0.544	
SVG, <i>n</i> (%)	3 (0.9)	0 (0)	3 (1.5)	0.281	0 (0)	0 (0)	0 (0)		
Failed procedure, n (%)	5 (1.5)	1 (0.7)	4 (2.0)	0.652	2 (1.1)	0 (0)	2 (2.2)	0.497	
Contrast volume, ml	196 ± 92	191 ± 92	200 ± 92	0.398	201 ± 97	199 ± 100	204 ± 94	0.735	
Procedure time, min, n (%)	23 (16–32)	23 (15–31)	23 (16–34)	0.391	24 (16–33)	24 (15–30)	24 (17–35)	0.243	
AKI, <i>n</i> (%)	35 (10.3)	12 (9.0)	23 (11.2)	0.503	16 (8.8)	9 (9.9)	7 (7.7)	0.601	
ACS: acute coronary syndrome, AKI: acute kidney injury, CABG: coronary artery bypass grafting, CCB: calcium channel blockers, CX: circumflex artery, eGFR: estimated glomerular filtration rate, HDL: high-density lipoprotein, LAD: left anterior descending artery, LDL: low-density lipoprotein, LMCA: left main coronary artery, NSTEMI: non-ST-segment elevation myocardial infarction, PCI: percutaneous coronary intervention, RCA: right coronary artery, STEMI: ST-segment eleva- tion myocardial infarction, SVG: saphenous vein graft, TG: triglycerides, WBC: white blood cells.									

11.2%, p = 0.503) and PS-matched (9.9 vs 7.7%, p = 0.601) study population (Fig. 1).

Multivariate logistic regression analysis of AKI following PCI is presented in Table 2. The overall success of the models for unmatched and matched patients was 90.2 and 93.2%, respectively. Age, female gender, baseline SCr level, baseline

eGFR and contrast volume were independent predictors of AKI in all patients. The same predictors were found in the matched patients; the only exception was baseline SCr level.

In order to avoid data loss due to dichotomisation, we analysed post-PCI SCr changes individually. There was no difference between TRA and TFA in terms of absolute and



proportional change in SCr level in PS-matched patients as well as in the overall study patients (Table 3). We also analysed different AKI endpoints through dichotomisation. TRA resulted in a significantly lower incidence of SCr increase of > 50% in unmatched patients. However, after PS matching, there was no difference between TRA and TFA in any variable of post-PCI renal outcomes. Notably, none of the patients required post-procedural dialysis in the study.

Discussion

The main finding of this study is that TRA did not decrease the incidence of post-PCI AKI compared to TFA in patients not complicated by major bleeding, acute heart failure or haemodynamic disturbance. This was further confirmed after PS matching of both groups. In addition to the conventional AKI definition (> 0.5 mg/dl or > 25% increase), different cut-off values for AKI yielded similar results in the matched population, although there was a trend towards less renal injury with TRA in the unmatched patients. Importantly, absolute and proportional changes in SCr level after intervention did not differ between TRA and TFA. Our analysis differs from previous ones in that it did not include patients with haemodynamic deterioration, acute heart failure and major bleeding. As these patients were extremely high risk, they were susceptible to deterioration in renal function, not only due to intervention, but also due to impaired renal haemodynamics. The majority of excluded patients during the study period developed AKI after the intervention.

The pathophysiology of AKI after coronary interventions is multifactorial. It includes direct nephrotoxicity due to the contrast agent, systemic and renal haemodynamic conditions, and direct cholesterol/atheroma embolisation from the abdominal aorta.¹⁶ By excluding haemodynamic disturbances and major bleeding, and the fact that contrast volume in both access site groups was similar, we can conclude that the effect of cholesterol/atherosclerotic embolism to the renal arteries on the development of AKI after transfemoral PCI was either minimal or absent.

We found a 10.3% incidence of AKI post-PCI, according to the conventional definition. The rate of AKI was in line with published studies where the majority of patients had a normal pre-procedural renal function. The determinants of AKI were age, female gender, baseline SCr level, baseline eGFR and contrast volume. Female gender was not among the commonly reported determinants of AKI.¹⁷ Our analysis did not reveal other conventional risk factors such as diabetes or haemoglobin level. This might be related to the moderate size of the study.

Some studies have reported a reduction in AKI with the TRA, however, the protective effect of TRA came from a reduction in bleeding events. A meta-analysis of six observational studies concluded that the TRA decreased incidence of post-PCI AKI (OR 0.51, 95% CI 0.39–0.67, p < 0.0001) compared to the TFA.¹⁰ The authors reported two co-variates: it was more pronounced in patients with STEMI, and the protective role of the TRA was associated with a reduction in access-site bleeding.

AKI-MATRIX was a randomised, multi-centre study comparing TRA and TFA in patients with ACS. AKI was defined as 0.5 mg/dl or 25% increase in SCr level. The incidence

Table 2. Multivariate logistic regression analysis of AKI									
_	Overall study population $(n = 339)$				Matched study population $(n = 182)$				
Variables	X^2	p-value	Odds ratio	95% CI	X^2	p-value	Odds ratio	95% CI	
Age	5.648	0.017	1.055	1.01 - 1.103	4.379	0.036	1.075	1 005–1 151	
Female gender	8.447	0.004	7.284	1.909-27.791	4.417	0.036	5.856	1 127–30 438	
eGFR	10.296	0.001	1.065	1.025-1.107	8.683	0.003	1.051	$1\ 017 - 1\ 087$	
Contrast volume	5.958	0.015	1.005	1.001 - 1.009	4.036	0.045	1.006	1.000 - 1.012	
Baseline creatinine	4.254	0.039	21.212	1.164-386.617					
CI: confidence intervals, eGFR: estimated glomerular filtration rate.									

Table 3. Secondary renal outcomes of TRA vs TFA in the overall study and PS-matched population									
	Overal	l study population (n =	339)	PS-matched study population $(n = 182)$					
Variables	TRA	TFA	p-value	TRA	TFA	p-value			
Post-PCI SCr	0.89 (0.79–1.01)	0.94 (0.78–1.10)	0.063	0.89 (0.79–1.01)	0.96 (0.79–1.12)	0.099			
Change in SCr, mg/dl	0.04 (-0.02-0.09)	0.06 (-0.04-0.14)	0.212	0.02 (-0.04-0.07)	0.06 (-0.06-0.13)	0.199			
Change in SCr, %	3.8 (-2.6-10.0)	6.0 (-5.0-14.7)	0.344	2.5 (-4.5-9.3)	5.5 (-6.0-13.5)	0.274			
SCr increase > 0.3, mg/dl	4 (3.0)	16 (7.8)	0.097	4 (4.4)	6 (6.6)	0.747			
SCr increase > 0.5, mg/dl	1 (0.7)	9 (4.4)	0.096	1 (1.1)	2 (2.2)	1.000			
SCr increase > 25%	12 (9.0)	23 (11.2)	0.503	9 (9.9)	7 (7.7)	0.601			
SCr increase > 50%	0 (0)	10 (4.9)	0.007	0 (0)	3 (3.3)	0.246			
PCI: percutaneous coronary intervention, SCr: serum creatinine, TRA: transradial approach, TFA: transfemoral approach.									

of AKI was less with TRA (15.4 vs 17.3%). The authors reported TRA mainly reduced incidence of AKI by reducing bleeding events. When drop in haemoglobin level and blood transfusion were included in the multivariate analysis, the association of TRA with AKI was lost.¹¹

In a PS-matched retrospective analysis of patients undergoing PCI for myocardial infarction, TRA was not found to be independently associated with AKI in both the non-matched and PS-matched cohorts.¹² The authors concluded that the lower incidence of AKI in TRA might be influenced substantially by confounding factors, especially bleeding.

Similar to our study, some studies have reported no benefit of TRA on AKI. A study by Kolte *et al.* on patients with STEMI showed a non-significant change in the incidence of AKI, defined by > 0.5 mg/dl increase in SCr level, with TRA in the overall and PS-matched cohorts.¹³ A more recent study from Italy collected data from 4 199 patients undergoing angiography and/or PCI between 2007 to 2016, and concluded that TRA was not superior to TFA with regard to the development of AKI.¹⁴ The definition of AKI was the same as in our study, and AKI was observed at 13.2% in the radial and 11.7% in the femoral approach, which are similar to our results.

The effect of TRA on AKI was found to be variable in previous studies. It appears that incidence of bleeding determined frequency of AKI more than TRA did. The protective effect of TRA on AKI appeared largely to be due to a reduction in bleeding events. Therefore, TRA should be preferred over TFA whenever possible. Exceptions include complex procedures that usually need larger catheters, which may not be suitable for TRA, a need for better guiding catheter support from the transfemoral route, and the presence of radial artery occlusion following previous transradial intervention.

Numerous studies have shown that TRA reduces accesssite complications and bleeding events. The benefit is more pronounced in patients with ACS. Therefore, it is irrelevant whether TRA is associated with lower incidence of AKI after coronary intervention because of a reduction in bleeding episodes or atheroembolism from the aorta. It is also important to remember that the incidence of cholesterol embolism syndrome was reported as 0.15% in clinical studies and 25–30% in pathological series.¹⁸ Pathophysiologically, TRA probably reduces the incidence of cholesterol embolism to the renal arteries compared to the TFA, however, it seems the effect is small and difficult to differentiate from the effect of reduction in bleeding events.

Limitations

The relatively small sample size of the study is a limitation. In order to overcome the size issue, we analysed not only the conventional dichotomised AKI endpoint, but also absolute and relative changes in SCr level after PCI. They were very similar in both the overall and PS-matched patients. Another limitation of this study is its single centre and retrospective nature. Although we applied several statistical methods to reduce selection bias and to adjust for different variables in multivariate analysis, unknown or residual confounding factors could not be adjusted for.

As a retrospective analysis based on hospital records, AKI was detected between two and seven days (median three days)

after the intervention. A specific time point, instead of within seven days, would have been more accurate for the analysis. Echocardiographic data were not available for all patients, therefore an important predictor of AKI, left ventricular ejection fraction, was not evaluated and included in the regression analysis.

The application of periprocedural hydration was not systematic. Although it was used in most patients, some patients received it only post-procedurally (STEMI patients), some received it both pre- and post-procedurally, and some elective patients may not have received any hydration at all. This might have affected the incidence of AKI, although it was valid for both radial and femoral patients.

Conclusion

Compared to the conventional femoral approach, the radial approach was not associated with reduced incidence of AKI after PCI in patients not complicated by major bleeding, acute heart failure and haemodynamic disturbances.

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