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Incidence, predictors and clinical implications of new renal impairment following percutaneous coronary intervention

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ABSTRACT

Background Renal impairment post-percutaneous coronary intervention (post-PCI) is a well-described adverse effect following the administration of contrast media. Within a large cohort of registry patients, we aimed to explore the incidence, predictors and clinical outcomes of renal impairment post-PCI.

Methods The Victorian Cardiac Outcomes Registry is an Australian state-based clinical quality registry focusing on collecting data from all PCI capable centres. Data from 36 970 consecutive PCI cases performed between 2014 and 2018 were analysed. Patients were separated into three groups based on post-procedure creatinine levels (new renal impairment (NRI), defined as an absolute rise in serum creatinine>44.2 µmol/L or>25% of baseline creatinine; new renal impairment requiring dialysis (NDR), defined as worsening renal failure that necessitated a new requirement for renal dialysis; no NRI). Multivariate logistic regression analysis was performed to investigate the impact of NRI and NDR on clinical outcomes.

Results 3.1% (n=1134) of patients developed NRI, with an additional 0.6% (n=225) requiring dialysis. 96.3% (n=35 611) of patients did not develop NRI. Those who developed renal impairment were more comorbid, with higher rates of diabetes (22% vs 38% vs 38%, p<0.001), peripheral vascular disease (3.4% vs 8.2% vs 11%, p<0.001), chronic kidney disease (19% vs 49.7% vs 54.2%) and severe left ventricular dysfunction (5% vs 22% vs 40%, p<0.001). Multivariable analysis found that when compared with the no NRI group, those in the combined NRI/NDR group were at a greater risk of 30-day mortality (OR 4.77; 95% CI 3.89 to 5.86, p<0.001) and 30-day major adverse cardiac events (OR 3.72; 95% CI 3.15 to 4.39, p<0.001).

Conclusions NRI post-PCI remains a common occurrence, especially among comorbid patients, and is associated with a significantly increased morbidity and mortality risk.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Renal impairment is a common complication following PCI, with various well-described risk factors increasing the likelihood of its development.

WHAT THIS STUDY ADDS

⇒ The present study offers data from a large-scale, multicentre registry cohort that contributes to the existing body of knowledge pertaining to renal impairment post-PCI.

⇒ The utilisation of this large sample size allows for meaningful independent risk factors for both the development of renal impairment and pertinent clinical outcomes for those with new renal impairment to be modelled using multivariate analyses.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ The observed association between renal impairment and poor clinical outcomes/mortality found in this study emphasises the importance of recognising readily identifiable risk factors that may predict high-risk groups.

⇒ In turn, this may facilitate the appropriate utilisation of preprocedural/periprocedural prophylactic strategies aimed at mitigating rates of post-PCI renal impairment.

INTRODUCTION

Renal impairment post-percutaneous coronary intervention (post-PCI) remains one of the leading causes of iatrogenic kidney injury,[1–4] historically comprising almost one-third of all hospital-acquired kidney injury,[5] with more modest estimates in contemporary studies.[6] New renal impairment (NRI) is a well-documented complication of the administration of iodinated contrast media (CM).

While the incidence of NRI post-PCI may be as low as 1% in otherwise healthy individuals, this increased to almost 50% in patients with significant pre-existing risk factors.[7] Such a high incidence is compounded by the lack of effective therapies available to treat and prevent NRI. As such, there has been a greater emphasis placed on identifying

patients at high risk of acquiring renal impairment and applying targeted prophylactic therapies within these patient subsets.

NRI also has important prognostic implications for patients. There is a clear association between developing NRI and poor clinical outcomes, including a higher in-hospital mortality rate, higher morbidity rates and a prolonged length of stay.[2 8–10]

This study aimed to explore the incidence, predictors and clinical outcomes of NRI post-PCI within a large Australian population of patients undergoing PCI.

METHODS

Victorian Cardiac Outcomes Registry (VCOR)

VCOR is an Australian state-based clinical quality registry focusing on collecting prospective data of patients undergoing PCI across all 30 PCI-capable public and private hospitals in Victoria with the aim of improving both patient safety and quality of care.[11] Data collected by VCOR include baseline patient demographics, procedural information and both in-hospital and 30-day clinical outcomes for patients undergoing PCI. The data are collected and stored by VCOR personnel in accordance with an opt-out consent policy, with data first being deidentified prior to analysis by researchers.[12] This study analysed VCOR data collected for all patients undergoing PCI between 2014 and 2018 after obtaining ethics approval from the institutional human research ethics committee of the Alfred Hospital, the central healthcare network of the principal investigators.

Long-term survival status was obtained by linkage to the Australian National Death Index (NDI), a database housed at the Australian Institute of Health and Welfare that contains records of all deaths occurring in Australia since 1980. The following variables for each deceased patient were identified: name, date of birth (or estimated year of birth), age at death, gender, date of death, state/territory of registration and registration number.

Baseline characteristic definitions

Patients who had successful or attempted PCI were included, irrespective of clinical indication. Patients without a measured pre-PCI and post-PCI renal function were excluded from the study. NRI was defined as an absolute rise in serum creatinine (SCr)>44.2 µmol/L or >25% of baseline creatinine up to 5 days after the index PCI, in keeping with international guidelines[13] and multiple previous studies.[14–16] New dialysis requirement (NDR) was defined as worsening renal failure that necessitated a new requirement for renal dialysis (including haemodialysis, peritoneal dialysis, haemofiltration, haemodiafiltration or ultrafiltration). Renal impairment was recorded from creatinine samples taken after PCI, but prior to discharge and/or subsequent catheter lab visits. Baseline renal function was recorded from creatinine samples collected up to 60 days prior to the index procedure, with the estimated glomerular filtration rate (eGFR) derived using the chronic kidney disease (CKD)–EPI formula and stratification into stages of kidney function 1–5 as per the Kidney Health Australia guidelines.[17] In keeping with these guidelines, CKD was defined as a baseline renal function of stage 3a or worse, corresponding with an eGFR of <60 mL/min/1.73 m^2. Baseline demographics were compared among patients grouped as either NRI, NDR or no NRI. Patient, treatment and procedural characteristics were collected and compared between the groups.

Clinical outcome definitions

Both in-hospital as well as 30-day clinical outcomes were collected for analysis between groups. This was inclusive of all-cause mortality, new myocardial infarction (MI), new stent thrombosis (ST), the need for emergency PCI/target vessel revascularisation/target lesions revascularisation/coronary artery bypass graft (CABG) and the incidence of rehospitalisation after the index admission. Major bleeding was also compared between the three groups, and was defined in accordance with the Bleeding Academic Research Consortium (BARC) classification as either BARC 3 or 5 (including overt bleeding with haemoglobin drop>30 g/L necessitating transfusion or surgical intervention, intracranial haemorrhage, cardiac tamponade and/or fatal or probable fatal bleeding).[18] Where follow-up data could not be obtained through medical records, it was acquired through contacting the patient, the patient's next of kin or the patient's general practitioner.

Statistical analysis

The primary hypothesis that the present data analysis sought to explore is that renal impairment post-PCI can be readily predicted by a number of key risk factors identifiable prior to angiogram. The secondary hypothesis of interest is that the development of renal impairment following PCI is associated with adverse clinical outcomes.

A univariate analysis was performed to compare the baseline and procedural characteristics between the NRI, NDR and control groups. Categorical variables were analysed using Pearson's chi-squared test and expressed as a number and percentage. Continuous variables were analysed with a t-test, Manning-Whitney U test or Kruskal-Wallis test as appropriate, and are expressed as a mean and SD. A calculated difference between groups were considered statistically significant if two-tailed p values were <0.05. A multivariable logistic regression was performed to determine adjusted effect measures of baseline demographics and clinical outcomes on the combined endpoint of NRI or NDR, reflected as an OR. A sensitivity analysis was conducted following the inclusion of patients without recorded renal function in order to predict the effects of the unmeasured confounder. The covariates adjusted for in the multivariable analysis included age, sex, treatment at a private hospital, diabetes mellitus, peripheral vascular disease, cerebrovascular disease, CKD, previous PCI, left ventricular ejection fraction (LVEF), baseline

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renal function, emergent PCI, cardiogenic shock, out-of-hospital cardiac arrest (OHCA), in-hospital cardiac arrest (IHCA), preprocedural medications (including thienopyridine, aspirin, ticagrelor), femoral access, requirement for adjunctive device and lesion type (ie, ACC/AHA B2/C). Cox proportion hazard modelling was used to assess for independent predictors of long-term mortality. Univariate variables with p<0.10 were included in our model to obtain adjusted HRs and 95% CIs. Statistical analyses were performed using Stata V.16.

RESULTS

Data from a total of 36 970 patients who underwent PCI from 2014 to 2018 were analysed, with 35 611 patients (96%) in the no NRI group, 1134 patients (3.1%) in the NRI group and 225 patients (0.6%) in the NDR group. Additional 14 067 patients who did not have postprocedure creatinine measured were excluded from the study (online supplemental figure 1). A sensitivity analysis including these patients found similar rates of risk factors and clinical outcomes between this cohort and the no NRI group.

Baseline characteristics

Baseline characteristics between the three groups are presented in table 1. Compared with those who did not develop NRI, the NRI group were more likely to be female and had a higher mean age. Average length of stay was significantly increased in the NRI and NDR groups (3.8 days vs 9.2 days vs 15 days, p=0.001) compared with those in the no NRI group. Comorbidities analysed were more common in the NRI and NDR groups. Significantly higher rates were observed for diabetes mellitus, peripheral vascular disease and cerebrovascular disease. CKD was more prevalent in the NRI and NDR groups, while those with preserved kidney function comprised the majority of those without NRI (72.8%). A normal LVEF was more common in those without NRI, whereas moderate and severe reductions in LVEF were more common in those with NRI and NDR. Patients without NRI were less likely to have presented with ST-elevation myocardial infarction and more likely to have presented with a non-ACS. Those who develop NRI or NDR were more likely to have presented with OHCA, IHCA and cardiogenic shock.

Periprocedural and medication characteristics

Periprocedural and medication characteristics across the three groups are presented in table 2. The rate of thrombolysis was higher in both NRI and NDR groups. Aspirin use remained similarly high across all groups, though this comparison did not reach statistical significance. Thienopyridine rates were observed to be higher in those without NRI or NDR and ticagrelor rates higher in the NRI group. Radial/brachial access was significantly higher in those without NRI, with femoral access was higher in patients with NRI and NDR. PCI to the RCA was more commonly seen in those without NRI while those with NRI and NDR were more likely to have had PCI to their left anterior descending, LM or a graft site. Patients without NRI had less complex (ACC/AHA type A/B1) coronary lesions and in turn, those with NRI and NDR were more likely to have complex (ACC/AHA type B2/C) lesions. The use of adjunctive devices employed during the index PCI were associated with increased rates of both NRI and NDR.

Clinical outcomes

The clinical in-hospital and 30-day outcomes are displayed in online supplemental table 2. All-cause in-hospital mortality was significantly higher in those with NRI and NDR (1.2% vs 17% vs 45%, p<0.001). Higher rates of both in-hospital repeat revascularisation by PCI and in-hospital stroke were observed in those with NRI and NDR. Major in-hospital bleeding as defined by the BARC criteria was shown to be significantly higher in both NRI and NDR groups, as were in-hospital ST and CABG, with similar results being reflected at 30-day follow-up. Rehospitalisation rate at 30 days was higher in the NRI and NDR groups; however, target-vessel revascularisation and target-lesion revascularisation rates did not reach statistical significance.

Multivariate analysis of baseline characteristics

The independent predictors for the development of the combined endpoint of NRI or NDR are displayed in table 3 and included age (OR 1.02, CI 1.01 to 1.03, p<0.001), diabetes mellitus (OR 2.01, CI 1.76 to 2.29, p<0.001), peripheral vascular disease (OR 1.43, CI 1.12 to 1.82, p=0.004) and cerebrovascular disease (OR 1.33, CI 1.04 to 1.70, p=0.021). Each stage of CKD predicted the development of NRI/NDR, the strongest being CKD stages IV–V (OR 5.90, CI 4.37 to 8.08, p<0.001). All stages of reduced LVEF were independently associated with combined NRI/NDR, with severely reduced LVEF showing the strongest association (OR 3.81, CI 3.18 to 4.57, p<0.001) (figure 1). Preprocedural characteristics independently associated with NRI/NDR include urgent PCI, cardiogenic shock, OHCA and IHCA (figure 1). Procedurally, the use of femoral access and adjunctive device were associated with the combined endpoint. Lesion complexity B2/C did not reach statistical significance.

Multivariate analysis of clinical outcomes

A multivariable analysis was also conducted for the clinical outcomes of 30-day mortality and major adverse cardiac events (MACE), and are demonstrated in online supplemental table 2 and 3, respectively. The development of NRI or NDR was strongly predictive of 30-day mortality (OR 4.77, CI 3.89 to 5.86, p<0.001) and 30-day MACE (OR 3.72, CI 3.15 to 4.39, p<0.001). Similar predictors of NRI or NDR were also shown to significantly increase the likelihood of both death and MACE, including age, all stages of CKD, moderate–severe LV dysfunction, cardiogenic shock, urgent PCI, OHCA and IHCA (figure 2 and online supplemental

Table 1 Baseline clinical characteristics

	No NRI N=35 611	NRI N=1134	NDR N=225	P value
Age (years)	65.3±12.1	71.2±12.8	65.0±13.3	<0.001
Female sex	8381 (23.5)	345 (30.4)	49 (21.8)	<0.001
Length of stay (days)	3 (1–4)	6 (4–11)	10 (4–19)	<0.001
BMI (kg/m^2)	28.8±5.5	28.2±5.6	28.9±6.0	<0.001
Previous PCI	10 447 (29.3)	270 (23.8)	44 (19.7)	<0.001
Previous CABG	2406 (6.8)	105 (9.3)	15 (6.7)	0.004
Diabetes mellitus	7857 (22.1)	431 (38.0)	85 (38.1)	<0.001
Peripheral vascular disease	1222 (3.4)	93 (8.2)	24 (10.8)	<0.001
Cerebrovascular disease	1331 (3.7)	87 (7.7)	18 (8.1)	<0.001
Renal function (eGFR; mL/min/1.73 m^2)				
Stages I–II (>60)	25 944 (72.8)	488 (43.0)	89 (39.6)	<0.001
Stage IIIa (45–59)	3761 (10.6)	214 (18.9)	40 (17.8)	<0.001
Stage IIIb (30–44)	2144 (6.0)	193 (17.0)	32 (14.2)	<0.001
Stages IV–V (<30)	882 (2.4)	157 (13.8)	50 (22.2)	<0.001
Chronic dialysis	437 (1.2)	59 (5.21)	NA	<0.001
LVEF				
Normal (≥50%)	19 821 (63.1)	358 (34.7)	41 (19.9)	<0.001
Mild (45–49%)	6399 (20.4)	209 (20.3)	34 16.5)	
Moderate (35–44%)	3604 (11.5)	235 (22.8)	49 23.8)	
Severe (<35%)	1569 (5.0)	229 (22.2)	82 (39.8)	
Clinical presentation				
STEMI	9678 (27.2)	595 (52.5)	134 (59.6)	<0.001
NSTEMI	9650 (27.1)	283 (25.0)	50 22.7)	
UAP	2430 (6.8)	43 (3.8)	3 (1.3)	
Non-ACS	13 853 (38.9)	213 (18.8)	37 (16.4)	
Cardiogenic shock	781 (2.2)	204 (18.0)	100 (44.4)	<0.001
OHCA	922 (2.6)	101 (8.9)	56 (24.9)	<0.001
IHCA	560 (1.6)	80 (7.1)	46 (20.5)	<0.001
Urgent PCI	21 749 (61.1)	944 (83.3)	200 (88.9)	<0.001

Values are expressed as mean±SD or n (%). Length of stay expressed as median days (IQR).
ACS, acute coronary syndrome; BMI, body mass index; CABG, coronary artery bypass graft; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; IHCA, In hospital cardiac arrest; LVEF, left ventricular ejection fraction; NDR, new dialysis requirement; NRI, new renal impairment; NSTEMI, non-ST elevation myocardial infarction; OHCA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; STEMI, ST elevation myocardial infarction; UAP, unstable angina pectoris.

figure 2). Though comorbidities such as diabetes, peripheral vascular disease and cerebrovascular disease all predicted the development of NRI or NDR, they were not statistically significant predictors of death nor MACE.

Online supplemental table 4 and figure 3 show independent predictors of long-term mortality. NDI/NDR was an independent predictor of long-term mortality (HR 2.18, CI 1.94 to 2.46, p<0.001) at a mean follow-up of 2.3±1.5 years.

DISCUSSION

The results from our large, multicentre Australian population-based study of patients undergoing PCI showed that NRI occurred in 3.1%, and necessitated dialysis in 0.6% of cases. Comorbid patients were shown to have a higher risk, with diabetes, peripheral vascular disease and cerebrovascular disease each being independently associated with the development of NRI/NDR. In particular, the presence of severe left ventricular dysfunction increased the odds of developing NRI/NDR

Table 2 Preprocedural and periprocedural characteristics

	No NRI	NRI	NDR	P value
Preprocedural medications				
Thrombolysis	1361 (3.8)	73 (6.4)	13 (5.8)	<0.001
Oral anticoagulation	2151 (6.0)	101 (8.9)	12 (5.4)	<0.001
No antiplatelet	1270 (3.6)	39 (3.4)	17 (7.6)	0.006
Aspirin	32 458 (91.4)	1052 (93.3)	198 (90.0)	0.066
Thienopyridine	13 870 (39.0)	329 (29.0)	66 (29.3)	<0.001
Ticagrelor	15 234 (42.8)	593 (52.3)	92 (40.9)	<0.001
Procedural details				
Access site				
Radial/brachial	20 549 (57.7)	499 (44.0)	58 (25.8)	<0.001
Femoral	15 062 (42.3)	635 (56.0)	167 (74.2)	
Adjunctive device required	3838 (10.8)	176 (15.5)	32 (14.2)	<0.001
Intravascular USS	491 (1.4)	21 (1.9)	6 (2.7)	0.111
Optical coherence tomography	216 (0.6)	9 (0.8)	0 (0.0)	0.364
Thrombus aspiration device	1630 (4.6)	109 (9.6)	22 (9.8)	<0.001
Distal or proximal protection device	59 (0.2)	4 (0.4)	0 (0.0)	0.266
Rotational atherectomy	403 (1.1)	13 (1.2)	2 (0.9)	0.942
Fractional flow reserve	1003 (2.8)	16 (1.4)	1 (0.4)	0.002
Coronary vessel				
RCA	11 240 (31.6)	306 (27.0)	52 (23.1)	<0.001
LAD	14 523 (40.8)	478 (42.2)	99 44.0)	
LCx	8566 (24.1)	239 (21.1)	55 24.4)	
Left main	651 (1.8)	68 (6.0)	13 (5.8)	
Graft	631 (1.8)	43 (3.8)	6 (2.7)	
Lesion type				
Lesion A or B1	15 210 (42.7)	392 (34.6)	58 (25.8)	<0.001
Lesion B2 or C	20 401 (57.3)	742 (65.4)	167 (74.2)	

Values are expressed as n (%).
LAD, left anterior descending; LCx, left circumflex; NDR, new dialysis requirement; NRI, new renal impairment; RCA, right coronary artery; USS, ultrasound scan.

by more than threefold, and the presence of CKD stages IV–V increasing the odds almost sixfold. As expected, NRI/NDR was more likely to be observed in patients with more acute or complex presentations; with urgent PCI (for an acute coronary syndrome), cardiogenic shock, requirement for adjunctive device and both OHCA and IHCA being powerful predictors of NRI/NDR. From a clinical perspective, these data highlight the presence of readily identifiable risk factors for the development of NRI and NDR, which may be used to enhance decision-making regarding the appropriateness of an invasive approach, timing of procedures and possible targeted prophylactic measures among high-risk patients, which may lower rates of NRI.[19]

Our data also indicate a clear association between NRI and adverse clinical outcomes, with in-hospital mortality being significantly higher in the NRI (17%) and NDR (45%) groups as compared with those without NRI (1.2%). It is clear that both NRI and NDR also carry a significant morbidity burden, with higher rates of revascularisation (repeat PCI as well as CABG), major bleeding and stroke observed in these patients. The associated increase in morbidity and mortality endpoints persist at 30-day follow-up and are also reflected in subsequent longer-term mortality (Supplementary Figure 3). Similar findings were demonstrated by a pooled analysis from HORIZONS-AMI and ACUITY trial patients conducted by Giacoppo *et al*, who report markedly increased rates of all-cause mortality and MACE among a similar cohort even at the 1-year mark, with contrast-induced nephropathy being the strongest predictor for death.[20] As causality cannot be inferred from our present study, it is likely that a proportion of the observed association between NRI/NDR and poor outcomes is attributable to the more unwell patients within the cohort, such as those requiring urgent PCI and presenting shocked or in cardiac arrest.

Table 3 Multivariate analysis for combined new renal impairment or new dialysis requirement

	OR	CI	P value
Age	1.02	1.01 to 1.03	<0.001
Female sex	1.02	0.89 to 1.18	0.750
Diabetes mellitus	2.01	1.76 to 2.29	<0.001
Peripheral vascular disease	1.43	1.12 to 1.82	0.004
Cerebrovascular disease	1.33	1.04 to 1.70	0.021
Previous PCI	0.84	0.72 to 0.98	0.025
Renal function (eGFR; mL/min/1.73 m^2)			
Stages I–II (>60)	0.95	0.74 to 1.22	0.674
Stage IIIa (45-59)	1.91	1.44 to 2.53	<0.001
Stage IIIb (30-44)	2.54	1.89 to 3.42	<0.001
Stages IV–V (<30)	5.90	4.37 to 8.08	<0.001
LVEF			
Mild (45–49%)	1.37	1.15 to 1.62	<0.001
Moderate (35–44%)	2.26	1.91 to 2.68	<0.001
Severe (<35%)	3.81	3.18 to 4.57	<0.001
Urgent PCI (STEMI, NSTEMI or UAP)	2.21	1.85 to 2.64	<0.001
Cardiogenic shock	4.39	3.58 to 5.38	<0.001
OHCA	1.32	1.03 to 1.70	0.027
IHCA	1.52	1.16 to 1.99	0.002
Thienopyridine	0.88	0.73 to 1.05	0.155
Aspirin	1.48	1.16 to 1.90	0.002
Ticagrelor	1.34	1.14 to 1.58	<0.001
Femoral access	1.32	1.16 to 1.50	<0.001
Adjunctive device required	1.35	1.13 to 1.60	0.001
Lesion B2/C	1.14	1.00 to 1.30	0.056

Adjusted for age, sex, private hospital, diabetes mellitus, peripheral vascular disease, cerebrovascular disease, CKD, previous PCI, LVEF, renal function, urgent PCI, cardiogenic shock, OHCA, IHCA, preprocedural medications (including thienopyridine, aspirin, ticagrelor), femoral access, requirement for adjunctive device, lesion type (ie, B2/C).
OR for age is expressed (per year).
CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; IHCA, in hospital cardiac arrest; LVEF, left ventricular ejection fracture; NSTEMI, non-ST elevation myocardial infarction; OHCA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction; UAP, unstable angina pectoris.

Nevertheless, the data strongly correlate NRI/NDR with worse outcomes, clearly establishing both NRI and NDR as important clinical markers that herald poor cardiovascular outcomes for patients.

The reported incidence of NRI (3.1%) in our study is largely in keeping with the incidence of renal impairment post-PCI that has been widely documented in the previous literature, with rates cited as low as 0.7% and as high as 17%,[2 6 10 19 21] depending on the studied population and definitions of NRI applied. However, a large

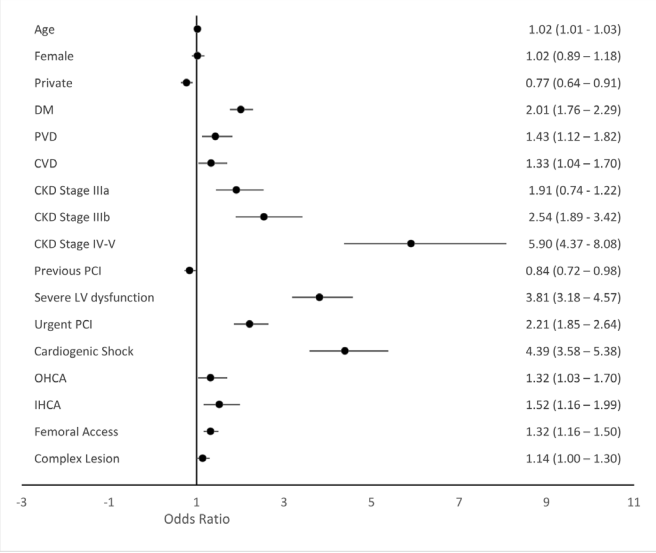

Figure 1 Independent predictors of combined new renal impairment or new dialysis requirement. CKD, chronic kidney disease; CVD, cerebrovascular disease; DM, diabetes mellitus; IHCA, in-hospital cardiac arrest; LV, left ventricular; OHCA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; PVD, peripheral vascular disease.

proportion of these studies have been published over a decade ago, with a distinct lack of population-based contemporary studies. Modern studies looking at the incidence and outcomes of renal impairment can be considered particularly valuable given the recent advances in prophylactic measures aimed at mitigating rates of NRI, with recent Kidney Disease: Improving Global Outcomes

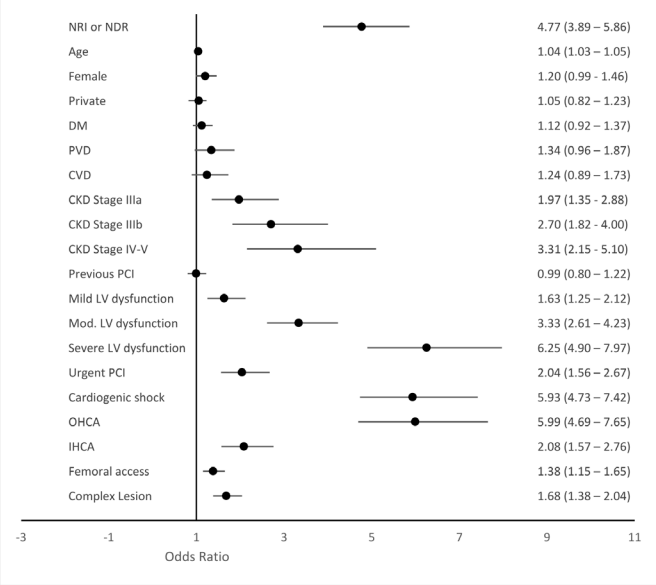

Figure 2 Independent predictors of 30-day mortality. CKD, chronic kidney disease; CVD, cerebrovascular disease; DM, diabetes mellitus; IHCA, in-hospital cardiac arrest; LV, left ventricular; NDR, new dialysis requirement; NRI, new renal impairment; OHCA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; PVD, peripheral vascular disease.

international guidelines recommending a careful pre-PCI selection process, judicious use of CM, intravenous volume expansion and low/iso-osmolar media agents.[13] Contemporary cardiac guidelines support these sentiments, recommending all patients undergoing angiography to be evaluated for the risk of developing NRI and to use adequate intravenous hydration and high-dose statins to minimise the risk of renal impairment.[22]

The definition selected for NRI plays an important role in determining the incidence of renal impairment post-PCI. Indeed, a common explanation for the heterogeneity in the reported incidence of renal impairment post-PCI has been the use of various different definitions for renal impairment.[20 23] The definition used for inclusion into the NRI group in this study is an absolute increase in SCr of 44.2 µmol/L or relative increase in SCr of 25%. This definition is the most consistently used in the literature and, after comparison with various other definitions, has been shown to consistently predict MACE and mortality after PCI.[14–16] A large, contemporary study by Tsai *et al* concluded the rate of renal impairment post-PCI to be 7.1% among their 985 000 patients, with 0.3% requiring dialysis.[24] The reportedly higher incidence of renal impairment may be in part due to their use of the more sensitive definition of acute kidney injury (AKI) adopted by the Acute Kidney Injury Network: a>0.3 mg/dL absolute or >50% relative increase in SCr.[25] The utilisation of more sensitive definitions has also been seen in other studies[26] and comes with the potential benefit of detecting additional patients at an increased risk of poorer outcomes, but will tend to produce heterogenous groups inclusive of low-risk patients.[27] A key strength of our study is therefore our use of a widely accepted definition for renal impairment post-PCI that enables the stratification of only the highest risk patients most susceptible to adverse cardiovascular outcomes.[28]

Study limitations

There are a number of study limitations to note. The key drawback to this study is its observational nature. Powerful associations were made with morbidity and mortality outcomes; however, we cannot ascertain causality, and it is likely that a number of cardiovascular outcomes such as MI and revascularisation were also contributory to the observed rates of renal impairment, as has been previously described by the cardiorenal relationship.[20 22 29] The retrospective nature of the study makes it difficult to ascertain whether the recorded renal impairment was truly due to contrast from the invasive procedure, as VCOR does not collect data to rule out other causes of renal failure. For this reason, the umbrella term NRI was used in preference to contrast-induced nephropathy, acknowledging that many of our patient group may have multifactorial aetiologies of the renal impairment, overestimating the true incidence of contrast-induced nephropathy. Moreover, as this analysis was not prespecified during dataset generation, certain variables previously linked with renal impairment such as the dose of contrast administered and concomitant renotoxic medications[17 19 22] were not collected by VCOR, and thus their relationship to NRI/NDR in our population was unable to be examined.

CONCLUSIONS

In this population-based study of contemporary PCI practices, NRI remains common, occurring in over 3% of patients. Renal impairment post PCI is associated with significant morbidity and mortality, emphasising the role of preprocedural planning, clinical governance and policies to mitigate this risk.

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