The impact of coronary angiography on renal transplant function

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Abstract

Background

There may be reluctance to perform coronary angiography in kidney transplant patients due to perceived risk of iodinated contrast, despite an increased risk of cardiovascular disease compared with the general population.

Aim

We sought to determine if renal transplant function was adversely affected within 7, 30 and 180 days of coronary angiography.

Methods

Renal transplant recipients undergoing coronary angiography in a single centre (01/2006–02/2018) were identified retrospectively. Baseline and highest SCr within 7, 30 and 180 days of coronary angiography were extracted from the electronic patient record. Rise in creatinine >26 micromol/l was considered significant (equivalent to Acute Kidney Injury (AKI) Network criteria stage 1 AKI) and case note review performed to determine circumstance of renal decline.

Results

There were 127 coronary angiographies conducted in 90 patients: 67.7% were male and mean age was 58.0 (±10.1) years. There was AKI within 7 days in 18.9% cases, but SCr returned to baseline within 7 days or there was an alternative explanation for AKI in 83.3% of these. In the remaining 4 cases, there was progressive decline in renal transplant function. In the absence of critical illness, no patient required dialysis or extended hospital stay for contrast-associated AKI.

Conclusions

In this cohort of renal transplant recipients undergoing coronary angiography, AKI occurred in a minority of cases, and in more than 95% of such cases this effect was transient, with progressive renal decline a rare and predictable event. Renal transplant should not be regarded as a contraindication to coronary angiography.

Background and aim

Renal impairment is an independent risk factor for type 1 myocardial infarction or cardiac death at 1 year, independent of other known risk factors for cardiovascular disease[1]. Cardiovascular disease remains common after renal transplantation, with an incidence of 3-5 times that of the general population[2]. Death from cardiovascular disease in the renal transplant population has reduced in recent years, but still accounts for around 22% of deaths in prevalent renal transplant recipients, and around 26% of deaths in those aged <65 years[3].

Patients with renal transplant are deemed to be at higher risk of acute kidney injury (AKI) after receiving iodinated contrast[4], along with those with chronic kidney disease (CKD; particularly estimated glomerular filtration rate (eGFR) <40ml/min/1.73m^2), heart failure, age >75 years, hypovolaemia, high- or repeated-dose intravenous contrast or intra-arterial contrast administration[4].

Large, population-level analyses have recently been reassuring regarding the overall risk of acute kidney injury after intravenous contrast in those with normal renal function[6] and with chronic kidney disease[7]. A meta-analysis of the existing data suggests that acute kidney injury after contrast exposure is common in kidney transplant patients[8]. Despite the elevated level of risk of cardiovascular disease in the renal transplant population, there may be reluctance to perform coronary angiography because of perceived risk of contrast-induced nephropathy. This risk must be weighed against the potential increased risk of death with a functioning graft in those with significant and sub-optimally treated coronary artery disease. We sought to determine the impact of iodinated contrast on renal transplant function on an individual case basis in renal transplant recipients undergoing coronary angiography for any indication.

Methods

We retrospectively included all renal transplant recipients from a single centre undergoing coronary angiography from January 2006 to February 2018 inclusive. Baseline demographic and biochemical data were extracted from the electronic patients record, including: sex, age at angiography (years), serum creatinine (SCr – micromol/l) values at baseline, within 7 days, 30 days and 180 days following coronary angiography, time since transplant, time since first renal replacement therapy and date of death. We reviewed case notes for additional clinical data, including heart failure status and whether patients had an active prescription of ACE inhibitor (ACEi) or angiotensin receptor blocker (ARB) at time of coronary angiography, and date and type of cardiac surgery up to 180 days after coronary angiography. eGFR was calculated from SCr using the CKD-EPI equation[9]. Patients were not routinely administered IV fluid peri-procedure, but some received intravenous fluid before or after coronary angiography at the discretion of the responsible physician. Coronary angiography reports and images and electronic patient records were reviewed for indication and procedure conducted at angiography and route taken for angiography (transradial versus transfemoral). Ethical approval was not required on the basis that this was analysis of routine clinical data. Data were fully anonymised and Caldicott Guardian approval was granted by the information governance manager of NHS Greater Glasgow and Clyde.

We defined baseline SCr as the nearest SCr prior to date of coronary angiography. In 31 cases when the SCr was not available within 48 hours of angiography, the most recent available SCr was checked to ensure it was in keeping with SCr over the 3 months prior to coronary angiography: if it was not, the average SCr over 3 months prior to coronary angiography was considered the baseline SCr. The highest SCr within 7 days of coronary angiography was extracted and compared with baseline values. Acute kidney injury was defined as a rise in SCr >26 micromol/l (equivalent to Acute Kidney Injury Network criteria stage 1 AKI)[10] within 7 days of coronary angiography. In cases when there was AKI after coronary angiography, electronic case notes were reviewed for cause of creatinine rise. In

the absence of a clear alternative explanation for rise in SCr, AKI was attributed to iodinated contrast. To explore longer-term effects on renal transplant function, we extracted highest SCr values within 30 and 180 days of coronary angiography. When no creatinine values were available within 30 days, the next available SCr after 30 days was extracted.

Data are presented as mean ± standard deviation for normally distributed data; median and interquartile range for non-normally distributed data. Comparisons between groups were made using Student's t test, Mann-Whitney test or Chi-square test as appropriate. Data were collated and analysed using Microsoft Excel 2016 MSO and *stats* and *pastecs* packages for R statistical software (R Studio (version 1.0.136) available at http://www.R-project.org and distributed under the GNU (http://www.gnu.org) General Public License). Data are presented according to STROBE reporting guidelines for observational studies.

Results

There were 127 coronary angiographies conducted in 90 renal transplant patients over 12.2 years. There was a male preponderance of 67.7%. The mean age was 58.0 years (SD 10.2) and the median time since transplant was 6.2 years (IQR 2.5-16.1). Further baseline demographics can be found in **Table I**. The most common indications for coronary angiography were angina (27.6%), non-ST elevation myocardial infarction (23.6%) and staged percutaneous coronary intervention procedures (16.5%) (**Table II**). Diagnostic angiography was conducted in 59.1%; angioplasty or stenting in 40.9% (**Table III**).

The incidence of AKI was 18.9% (n=24/127; median rise 52 micromol/l, range 28-163). Those who had AKI within 7 days were younger (53.6 vs 59.0 years, p=0.01) with lower GFR (29.6 vs 44.7 ml/min/1.73m^2, p=0.003). There were no significant differences between time since renal transplant or duration of end-stage renal disease (**Table I**). There was a higher proportion of hypertension (95.8 vs 92.2%, p<0.001) but lower proportion of diabetes (29.2 vs 35.9%, p<0.001) in the group who had AKI (**Table I**).

SCr values were available within 7 days for 98 patients. In 56 instances when there was any rise in creatinine within 7 days, the median maximum creatinine rise was 23 micromol/l (range 2-163), and median time to maximum creatinine was 3 days (IQR 1-4). **Figure 1** shows the maximum change in serum creatinine for individual coronary angiography events (from baseline and within 7, 30 and 180 days).

The overall rate of confirmed AKI within 7 days was more common in those with GFR <30 ml/min/1.73m^2 (Chi-square p=0.01) but not significantly more common in those with heart failure, who were prescribed ACEi or ARB, who were undergoing percutaneous coronary intervention compared with diagnostic angiography alone or when undergoing angiography via femoral versus radial route (**Table III**). Amongst those with confirmed AKI within 7 days, 58.3% were conducted via transradial route; 41.7% via transfemoral route.

SCr returned to baseline within 7 days or there was an alternative explanation for rise in SCr in 20/24 cases (**Table IV**). In the remaining 4 cases with AKI attributed to administration of contrast (3% overall), there was known severe and progressive renal transplant dysfunction (baseline SCr median 354 micromol/l, range 274-464).

Amongst those with no available SCr within 7 days (n=29/127), none had a persistent rise in SCr extracted from days 7-30 post angiography, or the next available SCr after 30 days (median rise in SCr 3 micromol/l, range -31 - 25). In the absence of critical illness at time of coronary angiography, no patient required dialysis or extended hospital stay for contrast-associated acute kidney injury.

There were 2 patients who underwent cardiac surgery (coronary artery bypass grafting, open valve replacement/repair or combined surgery) within 30 days of coronary angiography. Neither patient showed deterioration in renal transplant function within 180 days. Of 6 patients undergoing cardiac surgery between 30 and 180 days after coronary angiography, two patients demonstrated progressive renal transplant dysfunction within 180 days

(baseline and 180 day change in SCr: 153 + 58 micromol/l; 144 + 48 micromol/l respectively).

Discussion

In this cohort of prevalent renal transplant recipients, there was a low rate of clinically significant AKI within 7 days of coronary angiography, with very few cases of AKI that could be attributed to administration of iodinated contrast, and the majority had only a transient rise in SCr.

We acknowledge some limitations in the findings of this study. First, this is a single-centre study and includes a relatively small cohort of patients. Second, coronary angiography necessarily requires administration of iodinated contrast, and therefore there is no control group available for comparison. Third, though some physicians reported efforts to minimise contrast exposure, we can make no comment on the volume or type of contrast administered – which may impact on risk of AKI[8] - as this was inconsistently recorded. Fourth, there was inconsistent measurement of SCr within 7 days of coronary angiography and we cannot be sure to have captured all episodes of AKI in this period. Nevertheless, in the group of patients who did not have available SCr within 7 days, there was no change to transplant function beyond 7 days compared with baseline. The strengths of this study lie in the individual case analysis, including qualifying the cause and degree of acute kidney injury. Based on a population-level assessment, we estimate 18.9% rate of AKI after coronary angiography, but 7.9% rate of contrast-induced AKI and only 3.1% associated with 6-month decline in transplant function, all of whom already had progressive deterioration in transplant function.

Contrast-induced nephropathy is thought to be a form of acute tubular injury. The mechanism is not completely understood but is likely to be multifactorial[11]. There remains significant anxiety in administering iodinated contrast to high-risk patients including renal transplant recipients. In a survey of 421 radiologists (2000 were offered participation), fewer

than 30% state that they would frequently administer iodinated contrast for CT scanning to patients with renal transplant and 11% reported they would never use contrast for renal transplant patients[12]. Amongst radiologists who would consider using contrast, the average cut-off serum creatinine was 145 micromol/l (equivalent to eGFR >30ml/min in most cases), but varied between 132-177 micromol/l for the majority of respondents[12].

AKI is common after coronary angiography: Tsai et al estimated that over a quarter of high-risk patients (including those with renal transplant) undergoing coronary angiography suffer post-procedure acute kidney injury[13]. We found a greater proportion of AKI in those with lower GFR. In a large, propensity score-matched analysis, McDonald et al[7] showed similar significant increase in AKI with decreasing GFR (n=12 508, p<0.0001) but did not find any association with contrast exposure. In a follow-up study to address groups with CKD 3 (GFR 30-59 ml/min/1.73m^2) and CKD 4/5 (GFR <30 ml/min/1.73m^2) specifically[14], AKI, dialysis requirement and mortality were not significantly higher in those who received iodianted contrast. These data cannot be directly extrapolated to renal transplant patients, who may have additional risk factors compared to those with CKD, including calcineurin-based immunosuppression and possibly greater duration and severity of diabetes and cardiovascular disease. In renal transplant patients, Haider et al reported no impact of baseline serum creatinine on risk of contrast-associated AKI[15], albeit in a population with better baseline transplant function (GFR >70ml/min/1.73m^2) than our own cohort (42 ml/min/1.73m^2). In a meta-analysis of renal transplant patients, incidence of AKI varied according to the procedure: 16% for cardiac catheterisation, 10% for other angiography and 6% for contrast-enhanced CT scan[8], though no study reported a persisting requirement for dialysis relating to iodinated contrast[8].

Strategies to reduce the risk of contrast-associated AKI have been implemented in recent years. Peri-procedure intravenous hydration is evidence-based in reducing the frequency and severity of AKI after contrast exposure[16],[17], though not routinely administered in our unit. There has been a move away from high-osmolar ionic contrast agents to lower-osmolar

agents and European Society of Cardiology guidelines now recommend reduction or restriction of contrast volumes in high-risk patients undergoing coronary angiography[18]: both are likely to contribute to lower AKI rate[8],[19]. The choice of approach for coronary angiography also impacts risk of AKI. The AKI-MATRIX study randomised 8210 patients to trans-radial or trans-femoral approach for coronary angiography in a 1:1 ratio[20]. Those undergoing transradial coronary angiography had significantly fewer AKI episodes (15.4% vs. 17.4%; p=0.018) compared with those undergoing transfemoral angiography[20]. Approximately 80.5% of coronary angiographies are conducted via the transradial route in the UK [21],[22]; over 90% coronary angiographies in Glasgow are transradial[23]. Proportionately fewer patients had transradial coronary angiography in our cohort (64.6%): lack of radial access due to previous fistula creation was the predominant explanation.

Conclusion

Consistent with previous reports, our findings are reassuring that risk of contrast-associated AKI is low, including in this cohort with sub-optimal kidney transplant function, without routine administration of intravenous hydration peri-procedure and undergoing proportionately more transfemoral angiographies than the general UK population. There is a clinically significant rise in SCr in only a very small minority of cases and does not substantially or permanently affect renal transplant function. Given the burden of cardiovascular disease in this patient group, renal transplant should not be regarded as a contra-indication to coronary angiography.

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Tables

Table I

Baseline data	No AKI (7 days) (n=103)	AKI (7 days) (n=24)	All (n=127)	P-value
Male (%)	67.0	70.8	67.7	0.72
Age (years)	59.0 (10.3)	53.6 (8.8)	58.0 (10.2)	0.01
Hypertension (%)	92.2	95.8	92.9	<0.001
Diabetes (%)	35.9	29.2	34.6	<0.001
Baseline SCr (micromol/l)	146 (107-209)	264 (161-429)	255 (110-220)	<0.001
Baseline eGFR (ml/min/1.73m^2)	44.7 (21.5)	29.6 (21.2)	41.9 (22.2)	0.003
Time since transplant (years)	5.7 (2.5-16.0)	8.5 (2.9-16.4)	6.2 (2.5-16.1)	0.97
Time since first RRT (years)	12.10 (6.3-23.3)	15.6 (8.1 - 26.2)	13.4 (6.8-25.0)	0.45
Dead at end follow-up (%)	16.5	33.3	19.7	0.11
Dialysis at end follow-up (%)	4.9	0.1	6.3	0.36

Baseline demographics of included patients. Data are represented as mean (SD) or median (IQR) for normally and non-normally distributed data respectively.

Table II

Indication	**n (%)** **(127 total)**
Angina	35 (27.6)
Non-STEMI	30 (23.6)
Staged PCI	21 (16.5)
Pre-operative valve replacement	18 (14.2)
STEMI	14 (11.0)
Investigation of heart failure	9 (7.1)

Indications for coronary angiography. PCI: percutaneous coronary intervention. STEMI: ST-elevation myocardial infarction.

Table III

	No AKI within 7 days (%)	AKI within 7 days (%)	Total
eGFR CATEGORY			
eGFR >60 ml/min/1.73m^2	28 (90.3)	3 (9.7)	31
eGFR 30-60 ml/min/1.73m^2	44 (88.0)	6 (12.0)	50
eGFR >30 ml/min/1.73m^2	31 (67.4)	15 (32.6)	46
Rate of AKI according to GFR category: Chi-square p=0.01			
POST-TRANSPLANT HEART FAILURE			
No heart failure	76 (82.6)	15 (16.3)	92
Heart failure	27 (75.0)	9 (25.0)	36
Rate of AKI according to heart failure status: Chi-square p=0.27			
ACEi OR ARB PRESCRIBED AT TIME OF CORONARY ANGIOGRAPHY			
No ACE or ARB	67 (79.8)	17 (19.5)	84
ACE or ARB	36 (83.7)	7 (16.3)	43
Rate of AKI according to prescription of ACE or ARB: Chi-square p=0.59			
PROCEDURE CONDUCTED AT ANGIOGRAPHY			
Diagnostic angiography	65 (86.7)	10 (13.3)	75
Diagnostic angiography & PCI	26 (70.3)	11 (29.7)	37
PCI only	12 (80.0)	3 (20.0)	15
Rate of AKI after diagnostic angiography vs intervention: Chi-square p=0.11			
ROUTE OF ANGIOGRAPHY (TRANSRADIAL VS. TRANSFEMORAL)			
Transradial	68 (82.9)	14 (17.1)	82
Transfemoral	35 (77.8)	10 (22.2)	45
Rate of AKI according to route of angiography: Chi-square p=0.48			
TOTAL	103 (81.1)	24 (18.9)	127

Rate of acute kidney injury after coronary angiography according to estimated glomerular filtration rate (eGFR), history of post-transplant heart failure, active prescription of ACE inhibitor (ACEi) or angiotensin receptor blocker (ARB) at time of coronary angiography,

procedure conducted at coronary angiography or route of coronary angiography. AKI: acute kidney injury. PCI: percutaneous coronary intervention (balloon angioplasty or stenting).

Table IV

Cause of deterioration	n= (24 total)	Peak SCr rise within 7 days: median (range)	Peak SCr within 30 days: median (range)	SCr difference at 180 days: median (range)
RELATED TO CONTRAST				
Genuine rise: Back to baseline within 7 days	6	43 (36 – 45)	43 (36 – 45)	-2 (-173 – 50)
Genuine rise: Later decline in renal function	4	47 (28 – 85)	76 (42 – 140)	167 (59 – 172)
UNRELATED TO CONTRAST				
SCr at angiography lower than usual baseline	4	35 (28 – 138)	39 (33 – 138)	25 (15 – 39)
Drug-induced AKI	1	30	30	na*
Usual variability in baseline SCr	1	33	33	16
Requiring dialysis for AKI	4	93 (58 – 129)	139 (58 – 195)	174**
Critical illness	4	72 (59 – 163)	72 (59 – 163)	na***

Cause of rise in creatinine >26 micromol/l within 7, 30 and 180 days of coronary angiography on review of electronic case records. SCr: serum creatinine. AKI: acute kidney injury. * no SCr available at 180 days. ** single SCr value available at 180 days. *** all patients died within 180 days of coronary angiography.

Figure legend

Figure 1: Bar chart to represent serum creatinine (SCr) kinetics for individual coronary angiography events at baseline and within 7, 30 and 180 days. Cases are ordered from lowest to highest baseline SCr. Positive or negative changes in SCr are represented in shaded bars going up or down from the baseline value of SCr respectively.