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Editor's Choice – Acute Kidney Injury (AKI) in Aortic Intervention: Findings From the Midlands Aortic Renal Injury (MARI) Cohort Study

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WHAT THIS PAPER ADDS

Acute kidney injury (AKI) has been associated with worse post-operative outcomes. The exact proportion of patients who develop AKI after aortic intervention and relevant risk factors are not well described, because of poor study design and inconsistent reporting. This multicentre prospective cohort study has shown that AKI is common after both open and endovascular aortic procedures; this drop in renal function persists after 30 days. Age, baseline renal function and pre-existing cardiovascular disease are the main risk factors. Future research should explore renoprotective strategies in this population, targeting predominantly the elderly, those with existing renal dysfunction or several cardiovascular comorbidities.

Objective: The incidence of acute kidney injury (AKI) after open (OAR) or endovascular (EVAR) aortic repair is unknown. This research assessed the proportion of patients who develop AKI after aortic intervention using validated criteria, and explored AKI risk factors.

Methods: This was a multicentre national prospective cohort study. Eleven centres recruited patients undergoing EVAR or OAR (September 2017—December 2018). Serum creatinine (SCr) and urine outputs were measured over a minimum of 48 h or throughout the index inpatient stay to define post-operative AKI using the Kidney Disease Improving Global Outcomes (KDIGO) criteria. Renal decline at 30 days was calculated using estimated glomerular filtration rate (eGFR) and the Major Adverse Kidney Events (MAKE) 30 day composite endpoint (consisting of: death, new dialysis, $>$ 25% eGFR decline).

Results: 300 patients (mean age: 71 years, standard deviation [SD] 4 years; 9% females) were included, who underwent: infrarenal endovascular aneurysm repair (EVAR) 139 patients, fenestrated EVAR (fEVAR) 30, branched EVAR (bEVAR) seven, infrarenal open aneurysm repair (OAR) 98, juxtarenal OAR 26. Overall, 24% of patients developed stage 1 AKI (defined at 48 h as per KDIGO), 2.7% stage 2 AKI and 1% needed renal replacement therapy before discharge. AKI proportions per intervention were: infrarenal EVAR 18%; fEVAR 27%; bEVAR 71%; infrarenal OAR 41%; juxtarenal OAR 63%. Older age (odds ratio [OR] 1.44 for EVAR, 1.58 for OAR), lower baseline eGFR (OR 0.88 EVAR, 0.74 OAR), and ischaemic heart disease (OR 4.42 EVAR, 5.80 OAR) were the main predictors of AKI for infrarenal EVAR and OAR. Overall, 24% developed the MAKE30 endpoint. All patients who died (0.6%) or developed a major cardiac event (5.6%) at one year had developed AKI.

Conclusion: AKI and short term renal decline after aortic intervention are common. Age, renal function, and cardiovascular disease are the main risk factors. Research should now focus on AKI prevention in this high risk group.

Keywords: Acute kidney injury, Aorta, Cohort study, Endovascular, Vascular surgery
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INTRODUCTION

Acute kidney injury (AKI) after any type of surgical or radiological intervention is independently associated with prolonged hospital stay, overall treatment costs, and excess cardiovascular morbidity.[1–3] Development of post-operative AKI is also independently associated with decreased long term survival in patients undergoing vascular procedures.[4–8] Aortic interventions, such as open (OAR) or endovascular (EVAR) abdominal aortic aneurysm (AAA) repair, are common major vascular procedures, with over 12 000 infrarenal aneurysms and 2300 complex aneurysms treated in the UK between 2015 and 2017. In 2017, 1246 OARs and 2907 EVARs were performed to treat infrarenal AAA, as well as 217 OARs and 1883 fenestrated (fEVAR) or branched (bEVAR) EVARs to treat complex AAAs according to the National Vascular Registry (NVR) report in the UK.[9] Even though similar detailed data are not available for every European country, a recent analysis of 11 national datasets between 2005 and 2013 has shown that the uptake of EVAR has increased across Europe and worldwide. Interestingly mortality after OAR has slightly increased, potentially because of increased complexity of those cases that cannot be addressed with EVAR.[10]

These treatments have previously been associated with rates of AKI as high as 25%, even in the elective setting.[11,12] Developing AKI in this context has been linked to adverse outcomes, over both the short and long term.[11,12]

Unfortunately, most series reporting on post-operative AKI after EVAR or OAR, especially in more complex procedures such as fEVAR and bEVAR, suffer from limitations, such as: retrospective or single centre design, use of non-validated AKI reporting criteria, use of inappropriate outcome measures such as serum creatinine (SCr) or creatinine clearance, and lack of details relating to patient risk factors.[12,13] Moreover, no studies in this clinical area have reported longer term decline using clinical criteria such as the "major adverse kidney events" (MAKE) criteria.[14] Previous literature has assessed a plethora of subclinical markers of renal injury in the setting of surgery or radiological intervention, such as the Nephrocheck panel. These markers, however, have not yet been validated for use in clinical practice.[15]

Therefore, the exact incidence of AKI in this population remains unclear and any causative associations have not been fully explored. It is impossible to design relevant focussed renoprotective strategies, which impact on outcomes after these complex high risk procedures.

Consequently, the main aim of this prospective multicentre cohort study was to report the proportion of patients who develop periprocedural AKI after aortic vascular intervention using up to date validated definitions and also to assess potential AKI risk factors in those patients. The study also aimed to report the proportion of patients who develop the MAKE renal outcome at 30 days, which objectively assesses the degree of renal injury after intervention.

METHODS

Study population

Patients undergoing aortic intervention were recruited prospectively from 11 vascular surgery centres between September 2017 and December 2018 (Table S1).

Inclusion criteria: age $\geq$18 years; scheduled to undergo an abdominal aortic procedure via open or endovascular means; able to provide written informed consent for the surgical procedure and study participation. Patients presenting with an aortic rupture or as an emergency were analysed separately.

Exclusion criteria: age $<$18 years; established cardiac failure with functional status $>$ New York Heart Association (NYHA) class III (severe heart failure); established renal failure requiring replacement therapy; pregnancy or lactation; isolated thoracic aortic procedure; isolated iliac procedure; patient unwilling or unable to provide informed consent in English.

Ethical statement and regulatory approvals

The study was approved by the Health Research Authority (HRA) and an NHS Research Ethics Committee (REC; reference: 16/NI/0239, 15/11/2016); it was sponsored by the University Hospital Coventry and Warwickshire, Department of Research, Development and Innovation (RD&I; reference: CI189516). All collected data were anonymised and the study complied with the Declaration of Helsinki, Health Research Authority (HRA) Good Clinical Practice (GCP) guidance and Caldicott principles.[16] The study was supported nationally by the Vascular and Endovascular Research Network (VERN), an established network of research active vascular surgery trainees,[17] and the National Institute for Health Research (NIHR) Clinical Research Network (CRN), following CRN portfolio adoption. It was funded by the NIHR Leicester Biomedical Research Centre (BRC). The research was named "Midlands Aortic Renal Injury" (MARI) study as it was led and sponsored by a centre in the Midlands.

Study procedures and follow up

Following informed consent, data were recorded prospectively at each site. Baseline biochemistry was performed within two weeks before the surgery. Peripheral venous blood samples were taken 24 h and 48 h after surgery to measure SCr; urine output was recorded during and for at least 48 h after the procedure. Following discharge, patients had a new SCr measurement at 30 days and a clinical assessment at 6 weeks as well as a SCr at one year. The patients then resumed normal clinical follow up. More follow up details are listed in Table S2. Data were entered prospectively in a purpose built remote electronic database. The Research Electronic Data Capture (REDCap) software was used to create the database, which was maintained by staff at the NIHR Leicester Biomedical Research Centre.

Definitions and reporting

All diagnoses and cardiovascular clinical events were defined as per the American Heart Association (AHA) guidance for cardiovascular studies.[18] Information relating to endovascular procedures were reported based on established reporting standards published by the Society for Vascular Surgery.[19] The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidance was used during protocol design to ensure accurate reporting of study findings.[20]

Estimated GFR was calculated using the Chronic Kidney Disease Epidemiology (CKD-EPI) formula, based on SCr, which is the most precise SCr based index of renal function in populations with established cardiovascular disease.[21]

AKI was defined as per the Kidney Disease Improving Global Outcomes (KDIGO) criteria,[22] the use of which is supported by current National Institute for Health and Care Excellence (NICE) guidance,[22] as follows:

- Rise in SCr $\geq$ 26.5 µmol/L within 48 h compared with baseline (or)
- Rise in SCr more than 1.5 times within 48 h compared with baseline (or)
- Fall in urine output $\leq$ 0.5 mL/kg/hour for $\geq$ 6 h.

Objectives and outcomes

Primary outcome measure. The proportion of patients who developed AKI within 48 h after aortic vascular intervention.

Secondary outcome measures. The proportion of patients who develop AKI after each different type of intervention (EVAR, fEVAR, bEVAR, OAR) and stages of AKI (Table S3).

The proportion of patients requiring renal replacement therapy at 30 days and one year.

The change in estimated glomerular filtration rate (eGFR) at thirty days and one year after intervention.

The proportion of patients who develop the MAKE30 composite renal endpoint (composite outcome of death, new dialysis, and/or 25% or greater decline in eGFR, compared with baseline).

Risk factors associated with development of AKI after infrarenal EVAR or OAR (the most common types of vascular aortic intervention).

The association between development of AKI and absolute change in eGFR at one year.

The association between development of AKI and change in eGFR >20% at one year, a widely accepted definition of medium term renal dysfunction.[12]

Devices, procedures, and peri-operative care

All procedures were performed in an operating theatre with intra-arterial monitoring. A total of six patients (all had infrarenal EVAR) underwent a procedure under loco-regional anaesthetic. All endovascular devices were used within the relevant manufacturer's instructions for use following a discussion at a multidisciplinary meeting (devices used are listed in Table S4). Patients with calcification or thrombus at their proximal aortic neck exceeding 50% of the circumference of the neck did not undergo infrarenal EVAR. Balloon moulding of the proximal aneurysmal neck (infrarenal EVAR) was not performed electively as an adjunct procedure in all cases. All procedures were performed as per instructions for use.

The following nephrotoxic medications were stopped for 48 h before and after all procedures: angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and non-steroidal anti-inflammatory drugs (NSAIDs). Patients on metformin stopped taking it 48 h before their admission and metformin was re-commenced one day after successful completion of the procedure. All patients were asked to stop eating 6 h and stop drinking clear fluids 2 h before the procedure. A urinary catheter was inserted after anaesthetic induction and hourly urine output was recorded until the catheter was removed. If the catheter was removed before the 48th post-operative hour, the patient was asked to void in a container.

Intra-operatively, the anaesthetist was asked to maintain the patient's mean arterial blood pressure (using inotropic support and/or intravenous fluids) within 90% of the baseline for at least 80% of the time, as per the intra-operative protocol of the recent feasibility trial relating to AKI in EVAR;[23] this intra-operative strategy was the result of a national consensus group (anaesthetists, nephrologists, vascular surgeons) regarding fluid administration peri-operatively in patients having aortic intervention.[23] Crystalloid solutions were used as first line intra-operative and post-operative fluid therapy (Hartmann's solution), guided by blood pressure measurements (all patients had intra-arterial monitoring throughout the procedure and for at least 6 h post-operatively) and hourly urine output (target > 0.5 mL/kg/hour).

Post-operatively, a minimum of 2 mL/kg/hour of intravenous Hartmann's solution was administered for 6 h in all patients, unless their requirements (based on blood pressure and hourly urine output) exceeded this volume. No specific renoprotective strategies were used, such as N-acetylcysteine, sodium bicarbonate administration, or aggressive pre-/post-operative intravascular volume expansion. A non-ionic contrast medium was used in all cases (71.44% w/v of iomeprol equivalent to 35% iodine or 350 mg iodine/mL) and contrast volumes were recorded on procedure completion.

Statistical analysis

All analyses were performed using SPSS version 24.0 for Windows (IBM, Armonk, NY, USA) based on a statistical analysis plan that was agreed prior to commencing this prospective cohort study as per the primary and secondary outcomes measures.

For continuous variables, means and standard deviation (SD) or medians and interquartile range (IQR) are reported for each group, according to the normality of the distribution, which was assessed using skewness, kurtosis, and the Kolmogorov–Smirnov test.

For categorical variables, the number (and percentage) of patients in each category is reported for each group.

Differences between groups were assessed using a *t* test (for continuous normally distributed variables) or a Mann–Whitney *U* test (for continuous non-normally distributed variables); categorical variables were compared using a Fisher's test (two tailed tests in all instances).

To assess interactions between baseline characteristics in terms of developing peri-operative AKI, a multivariable binary logistic regression was performed separately for those undergoing infrarenal EVAR or OAR (the most common types of vascular aortic intervention), adjusted for factors found to differ in univariable comparison. Previous retrospective research has shown that pre-operative eGFR and cardiovascular reserve are the main predictors of AKI following aortic intervention.[24–26] These have also been shown to predict AKI in other instances[6,7,22] and were entered into the model regardless of baseline differences. Assuming, based on previous series using similar AKI reporting criteria,[23,24,27] that the proportion of patients developing the primary outcome of interest would be 25%, a total of 268 patients would be required to confirm the true proportion of patients developing AKI with 90% power (αset at 0.05) within a range of 15–35%, hence this study aimed to recruit 300 individuals (these sample size calculations were performed a priori). A p value $< .05$ was considered to be statistically significant.

RESULTS

A total of 315 (consecutive) patients who provided written informed consent took part (mean age 71 years, SD 4 years; 29 females, 9%). Of those, 15 patients had emergency surgery (all for a ruptured or symptomatic AAA, median age 77 years: IQR 9, all male) and were therefore not included in the main analysis. An additional four patients had been recruited but withdrew their consent before study completion and eight patients did not have any data collected, hence they are not included in this report. A total of 412 aortic cases were performed in these centres during the study period, i.e. 97 patients did not provide consent or did not meet the inclusion criteria. No patients were excluded because of being diagnosed with NYHA class IV heart failure.

The main study cohort consisted of 300 patients (mean age 71 years, SD 4 years; 27 females, 9%) who underwent elective aortic intervention, including the following procedures: infrarenal EVAR 139, fEVAR 30, bEVAR seven, infrarenal OAR 98, juxtarenal OAR 26 patients (Table 1).

Baseline cardiovascular and renal disease characteristics were representative of a population with established vascular disease, with 38% having a history of a previous major cardiovascular event and 24% having an eGFR <60 mL/min/m^2 (Table 1).

All procedures were completed successfully. The intra-operative blood pressure targets (mean arterial blood pressure within 90% of the baseline for at least 80% of the time) were met in all cases. The mean hospital stay for each procedure type was: infrarenal EVAR 3.5 days; fEVAR 4.6 days; bEVAR 7.6 days; infrarenal OAR 8.5 days; juxtarenal OAR 10.9 days (Table 2). None of the patients returned to theatre within 48 h (AKI reporting window) or developed visceral branch occlusion, gut or limb ischaemia. The median follow up was 14 months (IQR: 4), during which time only two patients died (due to a myocardial infarction in both cases). All procedural and post-operative events/results are summarised in Table 2.

All patients who died or developed a major adverse cardiac event at one year had developed AKI peri-operatively. No further analyses regarding the association between AKI and future events were possible because of the size of the recruited population and the paucity of major adverse events. Of the 300 patients undergoing elective intervention (main study cohort), 83 patients developed AKI (28%). Seventy two patients (24% of the whole cohort) developed stage 1 AKI, eight patients (2.7% of the whole cohort) developed stage 2 AKI and three patients needed transient renal replacement therapy before discharge (1% of the whole cohort), all within 30 days of their index procedure. None developed end stage renal failure requiring permanent renal replacement therapy within 30 days.

The proportions of patients who developed AKI according to type of intervention were: infrarenal EVAR 18%; fEVAR 27%; bEVAR 71%; infrarenal OAR 41%; juxtarenal OAR, 63% (Table 2). Of those who needed transient renal replacement therapy before discharge (stage 3 AKI), two had undergone juxtarenal OAR and one bEVAR. Of those who developed stage 2 AKI, four had a bEVAR and four had a fEVAR.

The overall change in eGFR between baseline and 30 days was 7 (SD 3.2) mL/min/1.73 m^2 ($p < .001$). Changes in eGFR between baseline, 30 days, and one year are summarised in Table 3; those undergoing fEVAR had the highest drop in eGFR at 30 days (10 mL/min/1.73 m^2) and those undergoing infrarenal OAR had the least change in eGFR in the same time interval (2 mL/min/1.73 m^2). At one year, those undergoing fEVAR had the highest drop in eGFR (9 mL/min/1.73 m^2) and those undergoing infrarenal OAR had the least drop in eGFR (1 mL/min/1.73 m^2).

A total of 99 patients (33%) had an eGFR drop of $>20\%$ at 30 days and 91 patients (30%) at one year, which is a widely acceptable marker of medium term renal decline.[28] Patients having fEVAR were the most likely to have an eGFR drop of $>20\%$ at both 30 days (41%) and one year (37%).

Seventy two patients within the main study cohort developed the MAKE30 composite renal endpoint (24%; three because of requiring renal replacement therapy and 69 because of an eGFR drop exceeding 25% of baseline). The proportions of patients who developed the MAKE30 endpoint according to type of intervention were: infrarenal EVAR 19%; fEVAR 21%; bEVAR 43%; infrarenal OAR 17%; juxtarenal OAR 43%.

Table 4 summarises the differences in baseline characteristics between those who did and did not develop AKI among patients having infrarenal EVAR or infrarenal OAR.

Based on an adjusted multivariable logistic regression, the main predictors of AKI for those undergoing infrarenal EVAR were: age (odds ratio [OR]: 1.44, $p = .03$), history of ischaemic heart disease (OR: 4.42, $p = .01$), and baseline eGFR (OR: 0.88, $p < .001$) (Table 5). The main predictors of AKI development

Table 1. Baseline characteristics for all groups of patients having aortic intervention ($n = 300$)

Characteristics	Infrarenal EVAR ($n = 139$)	Infrarenal OAR ($n = 98$)	p^*	fEVAR ($n = 30$)	bEVAR ($n = 7$)	Juxtarenal OAR ($n = 26$)
Age − years	73 ± 3	71 ± 5	.19	72 ± 2	74 ± 3	71 ± 3
Male sex	126 (91)	89 (91)	.83	26 (87)	7 (100)	24 (92)
Weight − kg	85 ± 17	84 ± 16	.77	88 ± 17	73 ± 8	86 ± 15
Systolic BP	140 ± 22	142 ± 21	.84	136 ± 18	140 ± 25	142 ± 23
Diastolic BP	80 ± 13	81 ± 11	.72	78 ± 9	84 ± 11	81 ± 14
COPD	35 (25)	18 (18)	.69	8 (27)	2 (29)	7 (27)
Hypertension	99 (71)	74 (76)	.41	18 (60)	6 (86)	20 (77)
Angina	26 (19)	10 (10)	.27	5 (17)	0 (0)	3 (12)
Myocardial infarction	44 (32)	17 (17)	.06	8 (27)	2 (29)	4 (15)
Stroke	7 (5)	4 (4)	.33	0 (0)	1 (14)	0 (0)
Diabetes	31 (22)	11 (11)	.16	3 (10)	2 (29)	6 (23)
PAD	22 (16)	6 (6)	.17	3 (10)	0 (0)	2 (8)
Atrial fibrillation	35 (25)	11 (11)	<.001	11 (37)	0 (0)	2 (8)
Any MACE	35 (58)	38 (39)	<.001	11 (54)	0 (44)	2 (52)
Current smoker	71 (51)	47 (48)	.65	13 (43)	5 (71)	11 (42)
Pack years − n	32 ± 23	35 ± 31	.33	28 ± 16	NA	39 ± 18
ACEi	63 (45)	30 (31)	.29	12 (40)	3 (43)	8 (31)
ARB	14 (10)	15 (15)	.81	4 (13)	1 (14)	2 (8)
Statin	118 (85)	71 (72)	.20	23 (76)	6 (86)	21 (80)
Aspirin	75 (54)	57 (58)	.15	12 (40)	4 (57)	19 (73)
Clopidogrel	32 (23)	14 (14)	.51	6 (20)	1 (14)	0 (0)
Diuretic	3 (18)	14 (14)	.32	2 (20)	0 (43)	1 (12)
NSAIDs	3 (2)	3 (3)	.74	2 (7)	0 (0)	1 (4)
Ca^{2+} blocker	25 (18)	15 (15)	.27	6 (21)	1 (21)	5 (20)
β blocker	58 (42)	31 (32)	.13	15 (50)	2 (29)	6 (23)
SCr − mmol/L	98 ± 27	96 ± 28	.21	94 ± 20	115 ± 40	91 ± 19
eGFR − mL/min/kg^2	66 ± 21	71 ± 17	<.001	70 ± 19	54 ± 6	73 ± 16
Urea − mmol/L	6.4 ± 2.4	6.4 ± 2.1	.91	6.6 ± 2.4	9.5 ± 2.4	5.8 ± 1.8
Haemoglobin − g/L	141 ± 17	142 ± 22	.88	133 ± 21	124 ± 11	142 ± 12
Proximal neck length − mm	19 ± 2	15 ± 1	.82	9 ± 4	NA	11 ± 4
Proximal neck angulation − degrees	34 ± 7	37 ± 6	.08	34 ± 7	NA	

Data are given as n (%) or mean ± standard deviation. ACEi = angiotensin converting enzyme inhibitors; ARB = angiotensin receptor blocker; bEVAR = branched EVAR; BMI = body mass index; BP = blood pressure; COPD = chronic obstructive pulmonary disorder; eGFR = estimated glomerular filtration rate; EVAR = endovascular aneurysm repair; fEVAR = fenestrated EVAR; MACE = major adverse cardiovascular event; NA = data not available; NSAIDs = non-steroidal anti-inflammatory drugs; OAR = open aneurysm repair; PAD = peripheral arterial disease; SCr = serum creatinine; SD = standard deviation.
* For comparisons between infrarenal EVAR and OAR.

for those undergoing infrarenal OAR were: age (OR: 1.58, $p = .02$), history of ischaemic heart disease (OR: 5.20, $p < .001$), baseline eGFR (OR: 0.74, $p = .01$), and anaemia (OR: 0.74, $p = .001$) (Table 5). Multivariable adjusted analyses were not possible for patients having complex EVAR (f/b EVAR) because of the limited number of participants; this was not part of this study's objectives. There were no differences (univariable analysis) between those who did and did not develop AKI in terms of contrast volume for fEVAR (199 *vs.* 193 mL, $p = .42$) and bEVAR (298 *vs.* 258 mL, $p = .09$).

The mean eGFR drop between those who did and did not develop AKI at one year was: 12 mL/min/1.73 m^2 *vs.* 4 mL/min/1.73^2 ($p < .001$). Patients who developed AKI were more likely to experience an eGFR drop >20% at one year: 45% *vs.* 21% ($p < .001$, univariable analysis) (Fig. 1). These associations persisted in a logistic regression model, when adjusted for age, sex, baseline eGFR, history of ischaemic heart disease, infrarenal/juxtarenal aortic disease, and type of procedure (f/b/infrarenal EVAR or OAR): OR 2.92 (95% CI: 0.44−4.32, $p < .001$) for eGFR drop at one year > 20%.

Of those having emergency surgery (all for a ruptured or symptomatic AAA) who were not analysed as part of the main study group, four patients had an infrarenal EVAR and 11 patients had an OAR (all under a general anaesthetic). A total of 11 patients (73%) developed AKI (nine of those having OAR and two of those having EVAR) and four patients had died at 30 days (27%), all of whom had required renal replacement therapy (AKI stage 3). The remaining seven patients developed stage 1 AKI. All surviving patients had an eGFR drop >20% at 30 days. None completed one year follow up (all lost). Because of the small number of patients, comparative or adjusted analyses were not possible in this subgroup.

DISCUSSION

This multicentre prospective cohort study has defined the incidence of post-operative AKI following aortic intervention, using widely acceptable AKI definition criteria which include precise urine output monitoring and standardised SCr measurements. Overall, 28% of patients having elective aortic intervention developed post-operative AKI; the

Table 2. Intra- and post-procedural events per type of aortic intervention (n = 300)

Event	Infrarenal EVAR (n = 139)	Infrarenal OAR (n = 98)	p*	fEVAR (n = 30)	bEVAR (n = 7)	Juxtarenal OAR (n = 26)
Number of fenestrations or branches⇒	NA		NA	4 (2–4)	4	NA
Suprarenal clamp time – mins	NA					24 ± 4
Contrast volume – mL	118 ± 10	NA	NA	196 ± 57	276 ± 124	NA
Procedure duration – mins	98 ± 12	127 ± 23	<.001	144 ± 11	192 ± 29	144 ± 9
ITU stay – days	0.4 ± 2.8	2.1 ± 3.7	<.001	1.7 ± 1.0	4.7 ± 9.2	4.3 ± 6.4
Hospital stay – days	3.5 ± 3.5	8.5 ± 4.4	<.001	4.6 ± 2.0	7.6 ± 8.9	10.9 ± 8.5
Vasopressors used	101 (72)	92 (94)	<.001	21 (70)	6 (80)	26 (100)
Duration of vasopressors – mins	33 ± 12	61 ± 15	<.001	71 ± 22	90 ± 37	65 ± 25
RBC transfusion	6 (4)	9 (9)	<.001	1 (4)	1 (12)	2 (9)
Units RBCs⇒	1 (0.2)	2 (1)	.06	1 (0.4)	2 (1)	2 (1)
Accessory renal covered	6 (4)	NA NA	0	0 (0)		
Urine output day 1 – mL	1 707 ± 968	1 496 ± 858	<.001	1 506 ± 636	2 234 ± 1 696	1 235 ± 510
Urine output day 2 – mL	1 444 ± 711	1 876 ± 1 361	<.001	1 605 ± 615	1 197 ± 1 221	1 567 ± 953
Anuria ≥6 h (first 48 h)	0 (0)	4 (4)	.08	0 (0)	0 (4)	0 (0)
SCr – mmol/L, day 1	94 ± 28	102 ± 38	<.001	105 ± 39	109 ± 44	99 ± 25
Urea – mmol/L, day 1	6.4 ± 6.8	7.0 ± 2.2	.04	7.1 ± 2.3	8.7 ± (3.6	6.6 ± 1.7
Haemoglobin – g/L, day 1	122 ± 48	129 ± 54	.82	121 ± 28	123 ± 37	135 ± 46
SCr – mmol/L, day 2	101 ± 37	104 ± 47	.41	109 ± 49	126 ± 56	107 ± 50
Urea – mmol/L, day 2	5.3 ± 2.6	7.3 ± 3.2	.07	7.3 ± 3.0	8.8 ± 5.9	7.5 ± 3.4
Haemoglobin – g/L day 2	124 ± 33	119 ± 32	.82	111 ± 21	112 ± 44	125 ± 33
AKI	25 (18)	38 (39)	<.001	8 (27)	5 (71)	10 (37)
MAKE30	24 (17)	30 (31)	<.001	7 (24)	4 (60)	8 (31)
MACE 30 days	3 (2)	2 (2)	.81	1 (4)	0 (3)	1 (5)
MACE 1 year	6 (4)	6 (6)	.43	1 (4)	0 (6)	3 (11)
Mortality 30 days	0 (0)	0 (0)	NA	1 (0)	0 (0)	3 (0)
Mortality 1 year	0 (0)	1 (1)	NA	0 (0)	0 (0)	1 (4)

Data are given as n (%) or mean ± standard deviation unless stated otherwise.
EVAR = endovascular aneurysm repair; fEVAR = fenestrated EVAR; bEVAR = branched EVAR; OAR = open aneurysm repair; NA = not applicable; ITU = intensive treatment unit; RBC = red blood cells; SCr = serum creatinine; AKI = acute kidney injury; MAKE = major adverse kidney events; MACE = major adverse cardiovascular event.
* For comparisons between infrarenal EVAR and OAR.
⇒ Presented as median (range).

proportions of patients who developed AKI according to type of intervention were: infrarenal EVAR 18%; fEVAR 27%; bEVAR 71%; infrarenal OAR 41%; juxtarenal OAR 63%. Furthermore, this study confirmed that the main risk factors for developing AKI in this clinical setting include age, history of ischaemic heart disease, and baseline renal function (based on eGFR levels). Therefore, it has been shown that post-operative AKI is a common complication of both open and endovascular aortic surgery, when using appropriate definitions, and this calls for research to explore potential renoprotection strategies in this high risk group.

Research surrounding the incidence and mechanisms of AKI after major aortic intervention has suffered from some methodological issues which have not allowed meaningful literature syntheses or exploration of the relevant pathophysiology to then address AKI prevention. Some of the published literature has used outdated reporting criteria for both short and long term renal outcomes in this population of patients, as discussed in previous literature reviews.[12,27]

Reporting criteria conceived specifically to define acute renal decline, which has been referred to as "acute kidney injury" in recent years, have only recently been reported in the EVAR or OAR literature in a systematic manner.[29–34] eGFR, which is the optimal outcome measure for long term renal decline, has also not been used consistently in

Table 3. Estimated glomerular filtration rate (eGFR) during follow up (in mL/kg/1.73 m^2)

Post-procedural time	Infrarenal EVAR	Infrarenal OAR	fEVAR	bEVAR	Juxtarenal OAR
Baseline	66 ± 21	71 ± 17	70 ± 19	54 ± 6	73 ± 16
Day 1	64 ± 21	62 ± 33	62 ± 18	55 ± 11	61 ± 22
Day 30	61 ± 27	69 ± 26	60 ± 17	50 ± 8	68 ± 12
1 year	60 ± 24	70 ± 14	61 ± 16	50 ± 7	69 ± 11

Data presented as mean ± standard deviation. bEVAR = branched EVAR; EVAR = endovascular aneurysm repair; fEVAR = fenestrated EVAR; OAR = open aneurysm repair; SD = standard deviation.

Table 4. Characteristics of patients who did and did not develop acute kidney injury (AKI), having either endovascular aneurysm repair (EVAR) or open aortic repair (OAR) of an infrarenal abdominal aortic aneurysm (AAA)

Characteristics	Infrarenal EVAR (n = 139)			Infrarenal OAR (n = 98)		
	AKI	No AKI	p	AKI	No AKI	p
Patients	25 (18)	114 (82)	NA	40 (41)	58 (59)	NA
Age − y	77 ± 5	69 ± 2	<.001	73 ± 3	66 ± 5	<.001
Male sex	22 (89)	105 (92)	.45	39 (97)	49 (85)	.05
Weight − kg	88 ± 15	85 ± 18	.42	84 ± 14	84 ± 17	.92
Systolic BP	138 ± 19	140 ± 23	.62	140 ± 19	144 ± 22	.38
Diastolic BP	77 ± 11	81 ± 13	.18	78 ± 10	83 ± 11	.03
COPD	12 (46)	23 (20)	.01	7 (18)	11 (19)	1
Hypertension	16 (65)	84 (74)	.47	33 (82)	41 (70)	.24
Angina	8 (31)	18 (16)	.98	10 (26)	2 (3)	<.001
Myocardial infarction	9 (35)	36 (32)	.82	6 (15)	9 (16)	1
Stroke	0 (0)	8 (7)	.35	3 (8)	2 (3)	.37
Diabetes	7 (27)	24 (21)	.60	5 (13)	5 (9)	.74
Atrial fibrillation	5 (19)	31 (27)	.62	4 (10)	6 (10)	1
Current smoker	4 (15)	24 (21)	.79	5 (13)	17 (29)	.09
Pack years − n	29 ± 13	33 ± 26	.64	Not available		
ACEi	9 (36)	52 (46)	.38	14 (36)	17 (30)	.66
ARB	5 (20)	10 (9)	.14	8 (21)	6 (10)	.15
Statin	19 (76)	98 (86)	.22	30 (74)	41 (70)	.82
Aspirin	72 (52)	60 (53)	1	15 (38)	36 (62)	1
Clopidogrel	13 (24)	25 (22)	.79	6 (15)	9 (16)	1
Diuretic	7 (28)	17 (15)	.14	8 (21)	7 (12)	.26
NSAIDs	2 (8)	1 (1)	.08	2 (5)	2 (3)	.64
β blocker	10 (40)	47 (41)	1	14 (36)	17 (29)	.51
Baseline SCr − mmol/L	115 ± 37	94 ± 22	<.001	111 ± 27	85 ± 24.4	<.001
eGFR − mL/min/kg^2	62 ± 33	71 ± 21	<.001	70 ± 22	72 ± 17	.09
Urea − mmol/L	7.5 ± 3.2	6.2 ± 2.1	.01	7.3 ± 1.8	5.8 ± 2	<.001
Haemoglobin − g/L	131 ± 19	142 ± 81	.56	132 ± 18	141 ± 17	.06
Proximal neck length − mm	19 ± 4	20 ± 3	.21	15 ± 2	14 ± 4	.84
Proximal neck angulation − degrees	33 ± 7	34 ± 7	.82	Not available		
Suprarenal device fixation	22 (88)	104 (91)	.72	Not available		
Contrast volume − mL	137 ± 81	114 ± 67	.27	Not applicable		
Procedure duration − mins	96 ± 14	99 ± 12	.33	128 ± 22	127 ± 23	1
Vasopressors used	22 (88)	68 (60)	<.001	48 (100)	51 (88)	<.001
Duration of vasopressors − mins	54 ± 35	89 ± 30	.14	90 ± 50	92 ± 66	.41
RBC transfusion	2 (8)	2 (2)	<.001	4 (12)	6 (6)	<.001
Accessory renal covered	1 (5)	3 (3)	.51	1 (3)	2 (4)	1

Data are given as n (%) or mean ± standard deviation. AAA = abdominal aortic aneurysm; ACEi = angiotensin converting enzyme inhibitors; AKI = acute kidney injury; ARB = angiotensin receptor blocker; BP = blood pressure; COPD = chronic obstructive pulmonary disorder; eGFR = estimated glomerular filtration rate; EVAR = endovascular aneurysm repair; NSAIDs = non-steroidal anti-inflammatory drugs; OAR = open aneurysm repair; SCr = serum creatinine; RBC = red blood cells; SD = standard deviation.

the vascular literature to report renal outcomes after the immediate post-operative phase.[12,27]

Most authors so far have SCr as the main renal endpoint in vascular populations.[12] SCr is an insensitive index as it is affected by muscle mass, frailty, body weight, and will not fluctuate outside normal values until at least 50% of the functioning renal mass has been affected.[35] Some vascular studies have occasionally reported creatinine clearance (CrCl) estimated with the Cockroft-Gault formula. This equation carries all the limitations of SCr, significantly overestimating renal function.[35] Inclusion of "weight" in the denominator adds further to the overestimation of true GFR in obese individuals. It also requires computation of body surface area and adjustment to 1.73 m^2. Interestingly, some studies have reported incidence of renal "deterioration" in EVAR or OAR using the proportion of patients requiring renal replacement therapy as a main outcome measure.[36] Need for dialysis is not common after these procedures, especially in the elective setting, hence this leads to further confusion as rates of dialysis or very significant SCr increase will not exceed 1—5% in the medium term.[36]

This current study has addressed some of these issues, using the widely accepted KDIGO criteria and measuring both standardised SCr as well as urine outputs systematically in the post-operative phase. Inclusion of patients from 11 different centres ensures that these figures correspond to real world practice. The present findings have confirmed that AKI is a very common complication in this setting. Even after elective infrarenal EVAR, almost one in five patients developed the KDIGO endpoint within 48 h.

Table 5. Predictors of acute kidney injury (AKI), after endovascular aneurysm repair (EVAR) and open aortic repair (OAR) of an infrarenal abdominal aortic aneurysm (AAA)

Predictor of AKI	Infrarenal EVAR			Infrarenal OAR		
	OR	95% CI	*p*	OR	95% CI	*p*
Age − y	1.44	1.02–2.37	.03	1.58	1.21–2.44	.02
Ischaemic heart disease	4.42	2.12–5.89	.01	5.20	2.20–7.79	<.001
Current smoker	1.89	0.91–12.11	.88	2.32	1.00–4.71	.13
eGFR − mL/min/kg^2	0.88	0.56–0.93	<.001	0.74	0.57–0.98	.01
Anaemia	1.47	0.99–4.45	.82	1.83	1.22–5.71	<.001
Contrast volume − mL	2.21	0.44–14.59	.73	NA	NA	NA
Procedure duration − mins	3.21	0.47–11.29	.44	0.92	0.12–2.24	.86
Vasopressors used	2.19	0.98–12.47	.32	2.27	0.99–3.92	.57
RBC transfusion	1.27	0.96–2.27	.33	3.32	0.76–5.46	.09

AAA = abdominal aortic aneurysm; AKI = acute kidney injury; CI = confidence interval; eGFR = estimated glomerular filtration rate; EVAR = endovascular aneurysm repair; NA = data not available; OAR = open aneurysm repair; OR = odds ratio; RBC = red blood cells.

Besides SCr based criteria to define peri-operative renal injury, in the form of AKI, subclinical markers of renal dysfunction may be used in this setting. Abdelhamid *et al.*, for instance, showed that following aortic repair, cystatin C, a marker of tubular damage, rises early and that the rise is sustained even over a twelve month period.[37] Besides cystatin C (either in the serum or the urine), a plethora of other biomarkers have been proposed as early indicators of renal injury in the context of surgery or critical care.[38,39] These markers, however, have not been evaluated in clinical practice in prospective cohorts among patients having aortic procedures and therefore cannot yet be applied in clinical practice. Future research should focus on assessing such markers as potential early indicators of AKI or even predictors of future renal decline. This was beyond the scope of this study.

Developing AKI after any procedure may indeed be a reflection of poor pre-operative fitness. This is more so an issue in patients with vascular disease, who will often have multiple co-morbidities or established chronic kidney disease. Previous research, however, has shown significant and strong associations between post-operative AKI in vascular patients and medium or even long term outcomes. More specifically, post-operative AKI after major endovascular procedures is independently associated with a threefold increase in morbidity and twofold increase in mortality at five years.[12,40] In a meta-analysis of 41 709 individuals undergoing endovascular intervention, post-operative AKI was associated with more cardiovascular events over a five year follow up.[40] Finally, developing post-procedural AKI is independently associated with a more rapid renal long term decline.[28]

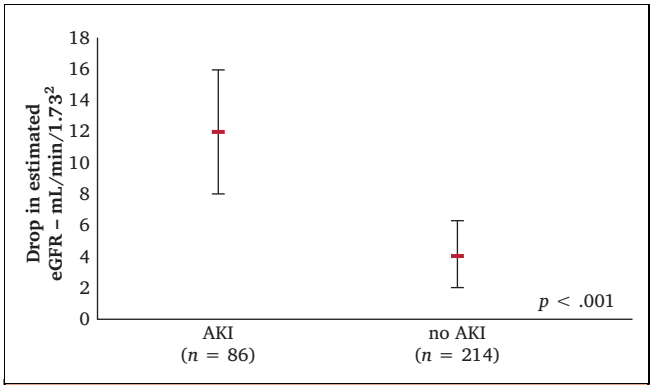

Figure 1. Drop in estimated glomerular filtration rate (eGFR) one year after the index aortic procedure for patients who did and did not develop acute kidney injury (AKI) peri-operatively (presented as mean drop per group with 95% confidence interval).

Further to establishing the incidence of AKI in these patients, this study sought to understand the risk factors of this important complication. Previously, baseline renal function and co-morbid conditions, especially relating to cardiovascular risk, have been identified as the major predictors of AKI in vascular patients.[24,26] This study confirmed this association, both for baseline eGFR as well as co-existing ischaemic heart disease. Certainly patients with either of those risk factors should receive close targeted follow up to ensure their renal function is not affected after aortic intervention.

The mechanisms that lead to AKI in these patients are complex and have not been fully explored in well designed studies. For those having open procedures, blood loss, dehydration, suprarenal clamping, and ischaemia reperfusion injury are the main proposed pathophysiological mechanisms. Pre- and post-operative hydration and transfusion when necessary are therefore paramount. In those having endovascular procedures, the proposed mechanisms include: contrast induced nephropathy,[41] ischaemic nephropathy from renal micro-emboli during stent graft deployment,[42] dissection or coverage of the renal arteries,[43] ischaemia reperfusion syndrome caused by lower limb ischaemia during the procedure,[44] and the presence of a pro-inflammatory milieu secondary to the systemic inflammation associated with AAA,[45] patients' pre-existing chronic kidney disease and poor cardiovascular reserve.[24] Interactions between all these factors mean that offering renoprotection in these patients is not a simple task.

Previous studies exploring AKI prevention in this setting have assessed all of the following interventions: intravenous fluids with or without N-acetylcysteine,[46] ischaemic preconditioning,[47] targeted renal therapy, which involves administering vasodilatory agents directly into the renal artery,[48] intravenous fluids with sodium bicarbonate,[49] administration of antioxidants (e.g. vitamin C),[50] and bolus doses of sodium bicarbonate intra-operatively.[23] Unfortunately, most of these studies were under powered and some have not used a consistent AKI definition. As a result, the optimal form of renoprotection in aortic intervention remains unknown.[12,23]

The findings of this study confirm that aortic intervention can be associated with high rates of AKI, especially in complex anatomies. The 2019 European Society for Vascular Surgery (ESVS) Guidelines on the Management of Abdominal Aorto-iliac Artery Aneurysms suggest that patients with renal impairment should be adequately hydrated before aortic repair, and eGFR, fluid input, and urine output should be monitored post-operatively.[51] These action points are even more crucial in patients undergoing fEVAR/bEVAR or complex OAR, as very high rates of AKI have been documented in these subgroups. Besides peri-operative monitoring, the fact that 72 patients (24%) in this series had persistent renal decline after 30 days means that those patients should have at least one assessment of their renal function during the first few weeks or months of follow up.

Limitations

This study has some limitations that need to be mentioned. The follow up of most patients was limited to one year, hence long term outcomes/associations cannot be assessed. The plethora of data that had to be collected, including precise urine outputs, intra-operative information, and standardised SCr measurements, means that not all patients undergoing aortic intervention in these centres could be recruited (97 patients were not recruited in the study). Furthermore, after removal of the urinary catheter, urine output was recorded using urinary bottles — this may have meant that in some cases the volumes were imprecise. Finally, associations between parameters such as proximal aneurysm neck balloon moulding (data not collected) and use of overlay techniques or suprarenal EVAR fixation (the vast majority of patients had a device with suprarenal fixation) could not be explored.

CONCLUSION

This prospective multicentre study has confirmed that AKI after open or endovascular aortic intervention is a common complication; renal deterioration at 30 days was also common. Age, baseline renal function, and pre-existing cardiovascular disease appear to be the main risk factors. Research should now focus on prevention of this important complication.

CONFLICT OF INTEREST

None.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2019.09.508.

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COUP D'OEIL

Endovascular Repair of a Complicated Persistent Sciatic Artery Aneurysm

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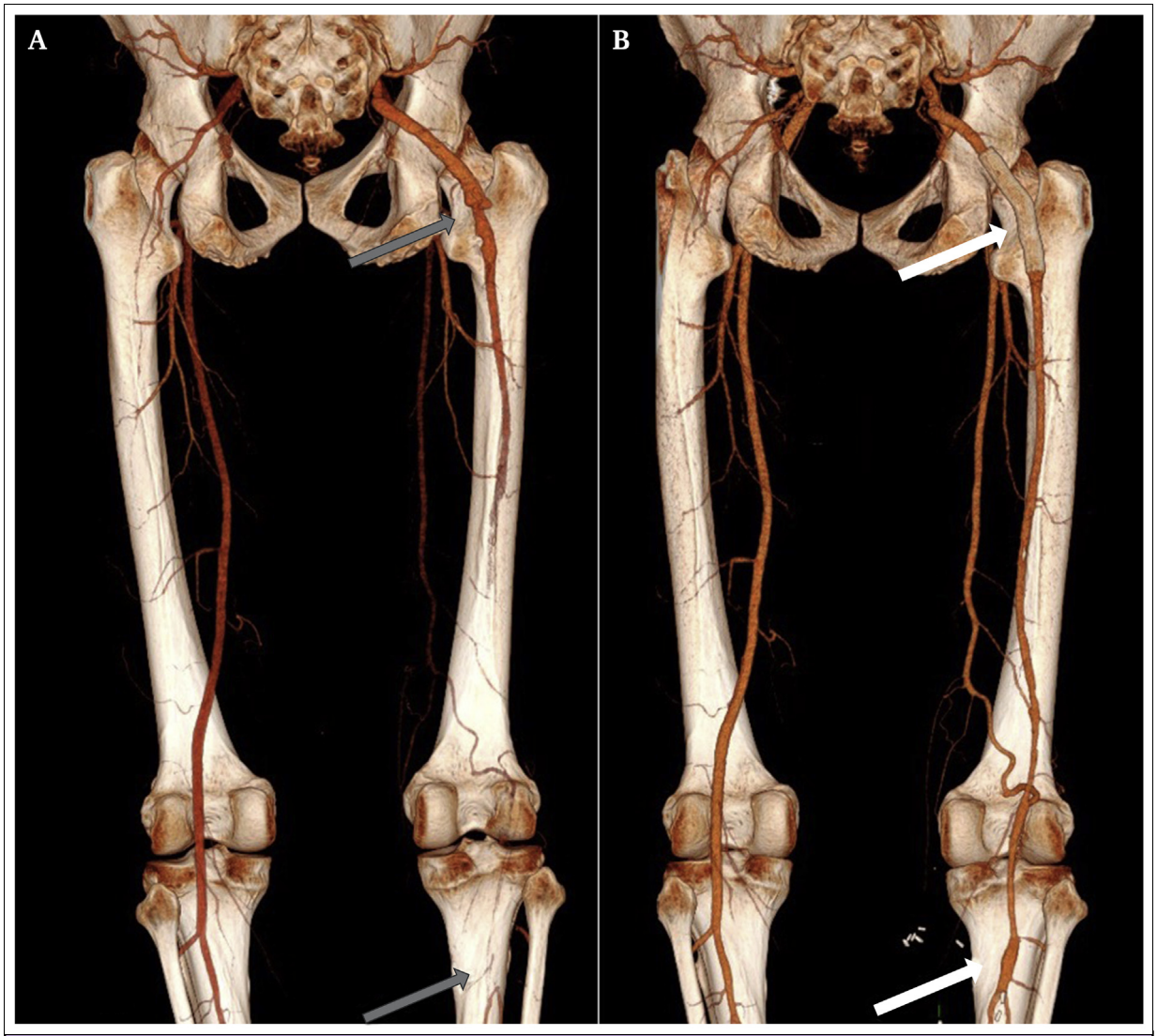

Persistent sciatic artery (PSA) is a rare congenital vascular abnormality, with an incidence of 0.04%. Symptomatic presentation occurs in 60% of cases, mainly due to sciatic nerve compression, or aneurysm formation with thrombosis and/or distal embolism. The endovascular exclusion of a complicated PSA aneurysm (PSAA; A, upper grey arrow) using a covered stent graft is reported (11 × 100 mm [Viabahn; WL Gore, Flagstaff, AZ, USA]). The procedure, in a 43 year old, was performed via a percutaneous contralateral approach after prior popliteal thrombectomy for distal embolism from the PSAA (A, lower grey arrow). Post-operative computed tomography angiography after 26 months revealed a patent stentgraft and below knee outflow (B, white arrows).

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