ELSEVIER

Risk Factors and Impact of Proximal Fixation on Acute and Chronic Renal Dysfunction After Endovascular Aortic Aneurysm Repair Using Glomerular Filtration Rate Criteria

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Background: The incidence of renal impairment relevant to proximal fixation of aortic endograft devices remains unclear.
Methods: Retrospective cohort of 208 consecutive patients that underwent EVAR from 2006 to 2011. Estimated glomerular filtration rate (eGFR) was based on MDRD study equation. Acute kidney injury (AKI) and chronic kidney disease (CKD) were classified with ADIQ/RIFLE criteria and National Kidney Foundation criteria, respectively. Kaplan-Meier curve was applied to evaluate progression to CKD. Multivariate regression model was fit to identify predictors for developing AKI and CKD.
Results: Suprarenal fixation group (SF) included 110 patients and infrarenal fixation group (IF) included 98 patients. Both groups had similar demographics, baseline eGFR, and renal-protection protocols. There was a trend for decreased use of contrast in IF group (median: 93.5 vs. 103 cc, $P = 0.07$). AKI occurred in 15% of patients in SF group and 19% of patients in IF group (RR: 1.24, $P = 0.47$). The freedom from progression to stage 3 or 4 CKD in the SF group was 0.76, 0.72, and 0.49 at 6, 12, and 18 months, respectively, while for IF group was 0.8, 0.73, and 0.68, respectively ($P = 0.4$). Increasing age ($P = 0.07$), lengthy procedures ($P < 0.001$), and baseline renal dysfunction ($P < 0.001$) were significant predictors for developing CKD. Contrast volume ($P < 0.001$) and ace-inhibitors ($P = 0.07$) were predictors for AKI.
Conclusion: Proximal fixation type has no significant effect on both acute and chronic renal function. Identification of modifiable perioperative risk factors may be used to improve renal function outcomes.

INTRODUCTION

Since the introduction of EVAR in 1991 by Parodi,[1] there have been a series of dramatic device improvements, mainly attempting to expand patient eligibility criteria and decrease rates of complications. Renal function outcomes have been correlated with several procedural aspects, mainly contrast, hemodynamic changes, and microemboli to renal parenchyma. Furthermore, the addition of suprarenal fixation (SF) stents to improve proximal fixation and seal[2] in challenging neck cases has been hypothesized to increase risk of microemboli and impede actual renal artery flow[3,4] compared to infrarenal fixation (IF) devices, therefore increasing risk of acute and chronic renal dysfunction. So far, most existing studies looking at renal outcomes after EVAR have either used plain creatinine values with arbitrary cut-off points to define renal dysfunction or outdated eGFR definitions.[5]

In this study, we seek to assess the incidence of acute and chronic renal impairment in relevance

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Table IA. RIFLE criteria for the classification of acute kidney injury

Class	GFR criteria
Risk	GFR decline >25% or serum creatinine increase 1.5 x baseline
Injury	GFR decline >50% or serum creatinine increase 2 x baseline
Failure	GFR decline >75% or serum creatinine increase 3 x baseline or acute serum creatinine >4 mg/dL
Loss	Persistent ARF = complete loss of kidney function requiring dialysis for >4 weeks but <3 months
End stage	End-stage kidney disease requiring dialysis for >3 months

Table IB. National Kidney Foundation classification stages for chronic kidney disease

Stage	Description	GFR
1	Kidney damage with normal or ↑ GFR	>90 (with CKD risk factors)
2	Kidney damage with mild or ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

RIFLE, risk, injury, failure, loss, end-stage kidney disease; GFR, glomerular filtration rate; ARF, acute renal failure; CKD, chronic kidney disease.

to proximal fixation after EVAR, using current eGFR definitions and widely accepted criteria for acute kidney injury (AKI) and chronic kidney disease (CKD). In addition, we aim to identify potential predictors for AKI and CKD based on the above criteria.

METHODS

This is a retrospective study of 208 consecutive patients that underwent EVAR for infrarenal aortic or aortoiliac aneurysms in a single institution from 2006 to 2011.

Chart review included patients with asymptomatic infrarenal aortic aneurysms of >5 cm, or aneurysms 4–5 cm that were symptomatic, eccentric, or rapidly expanding >5 mm in 6 months, and iliac aneurysm >3 cm in diameter. Exclusion criteria were applied to patients with emergent repair due to rupture, baseline dialysis, or stage 5 CKD. In addition, patients with concurrent stenting or of inadvertent coverage of a renal artery were also excluded. Suprarenal fixation grafts included Cook Zenith (Bloomington, IN), Endologix Powerlink (Irvine, CA), Medtronic Talent, and Endurant (Santa Rosa, CA), while infrarenal fixation grafts included Gore Excluder and C3 (Flagstaff, AZ), Medtronic AneuRx (Santa Rosa, CA), and Endologix Powerlink (Irvine, CA). Device selection was based on surgeon's preference.

RENAL FUNCTION DEFINITIONS

Estimated GFR was determined by using the abbreviated Modification of Diet in Renal Disease (MDRD) study equation ([eGFR] [mL $\cdot$ min^{-1} $\cdot$ 1.73 m^{-2}] = 186 $\times$ [serum creatinine]$^{-1.154}$ $\times$ [age]$^{-0.203}$ $\times$ [0.742 if female] $\times$ [1.210 if African American]).[6] Chronic kidney disease (CKD) stage I–V was classified based on the National Kidney Foundation/Kidney Disease Outcome Quality Initiative (NKF/KDOQI) criteria[7] (Table IA). The most recent proposal for a consensus definition for acute kidney injury (AKI) stems from the Acute Dialysis Quality Initiative group,[8] who suggested criteria for 3 grades of increasing severity—risk of acute renal dysfunction (R), injury to the kidney (I), failure of kidney function (F)—and 2 outcome classes—loss of kidney function (L) and end-stage kidney disease (E) (Table IB). The RIFLE classification based on eGFR within 48–72 hours after the procedure was applied to postoperative diagnosis of acute renal dysfunction. The patients' chronic renal function was monitored postoperatively as an outpatient at 3 months and every 6 months thereafter.

Perioperatively, renal protection protocol for patients with baseline CKD stage 3–4 included sodium bicarbonate intravenous (IV) hydration and oral acetylcysteine according to recommended guidelines[9] and all potentially nephrotoxic drugs were held. Intraoperative imaging was acquired with a portable or fixed c-arm unit and nonionic iso-osmolar contrast agent (Visipaque-320, General Electric, Fairfield, CT) was used in all cases with either hand injection or power injection at flow rates of 20 mL/s at 900 psi. The protocol for postoperative imaging included multi-detector CT angiography using 95 mL of nonionic iodinated contrast agent (Isovue-300, Bracco Diagnostics, Princeton, NJ) at a rate of 3 mL/s. Surveillance CT was obtained at 6, 12 months, and annually thereafter.

Table II. Demographics and comorbidities of patients in SF and IF groups

Variable	SF group (%)	IF group (%)	*P*
Gender (M/F)	110/0	98/0	n/a
Age (years)	70 ± 8	69 ± 8	0.2
Race (African American)	15 (14%)	12 (13%)	0.8
Hypertension	100 (91)	91 (93)	0.7
Diabetes mellitus	24 (22)	26 (26)	0.5
Hyperlipidemia	88 (80)	77 (78)	0.4
Coronary artery disease	53 (48)	45 (46)	0.6
Congestive heart failure	11 (10)	10 (10)	0.9
Chronic pulmonary disease	44 (40)	35 (36)	0.4
Smoking history (current or previous)	97 (88)	90 (92)	0.4
BMI	28 ± 5	27 ± 4	0.1

SF, suprarenal fixation; IF, infrarenal fixation; BMI, body mass index.

STATISTICAL ANALYSIS

Descriptive statistics were used to assess demographics, cardiovascular risk factor distribution, preoperative renal function, and intraoperative parameters. A student's t-test and Fisher's exact test were used to compare baseline characteristics for continuous and categorical variables respectively. Kaplan-Meier curve was applied to evaluate progression to CKD stage 3 or 4 during the follow-up period and log-rank test to compare the curves for both groups. Multivariate regression analysis after adjusting for confounders was performed to identify potential risk factors for developing AKI and CKD. A *P*-value of <0.05 was considered significant. The analyses were carried out using STATA version 11.2 (StataCorp, College Station, TX).

RESULTS

The study included 208 patients with 110 in the SF group and 98 in the IF group. The key demographic characteristics for the MDRD formula were evenly distributed (Table II). All patients were male with mean age of 70 years. No difference was found in the African American population between SF and IF groups (14% vs. 13% respectively, $P = 0.8$) Cardiovascular morbidities had similar distribution for both groups, with the most common being hypertension and hyperlipidemia, followed by chronic obstructive pulmonary disorders and coronary artery disease. Diabetes mellitus and chronic heart failure were diagnosed in less that a quarter of patients, as summarized in Table II.

Baseline eGFR had no difference between SF and IF groups (73.1 vs. 69.4 ml/min x 1.73 m^2, $P = 0.2$) and was similarly distributed for individual CKD stages between the two groups. Specifically, most patients had normal renal function or early stage CKD (53% vs. 58%, $P = 0.2$), with CKD stage 3 (41% vs. 34%, $P = 0.3$) being far more common than 4 (6% vs. 8%, $P = 0.6$), as shown in Table III.

Table III. Preoperative renal function stratification for SF and IF groups

Variable	SF group (%)	IF group (%)	*P*
Normal and CKD 1−2	58 (53)	57 (58)	0.2
CKD 3	45 (41)	33 (34)	0.3
CKD 4	7 (6)	8 (8)	0.6
eGFR (ml/min x 1.73 m^2)	73.1 ± 23	69.4 ± 24	0.2

SF, suprarenal fixation; IF, infrarenal fixation; eGFR, estimated glomerular filtration rate (mL/min/1.73 m^2).

Table IV. Procedure-related parameters for both groups

Variable	SF group (mean ± SD)	IF group (mean ± SD)	*P*
Contrast (ml)	112 ± 49	101 ± 54	0.12
Operative IVF (ml)	1864 ± 1052	1620 ± 834	0.07
Operative time (min)	172 ± 88	150 ± 78	0.05
Operative blood loss (ml)	350 ± 382	242 ± 291	0.02
Perioperative PRBC (u)	0.9 ± 1.5	0.6 ± 1.4	0.08

SF, suprarenal fixation; IF, infrarenal fixation; IVF, intravenous fluids; PRBC, packed red blood cells.

Procedural contrast volume was similar between the groups (112 cc vs. 101 cc, $P = 0.1$). There was a trend in the SF group towards increased intraoperative IV fluids (1864 cc vs. 1620 cc, $P = 0.07$), operative time (172 min vs. 150 min, $P = 0.05$), operative blood loss (350 cc vs. 242 cc, $P = 0.02$), and perioperative blood transfusion (0.9 units vs. 0.6 units, $P = 0.08$) (Table IV).

AKI Findings

We applied the RIFLE criteria to assess the incidence of AKI after EVAR for each group (Fig. 1). There were 21 patients in the SF group (19%) and 15 patients in the IF group (15%) that developed >25% eGFR decrease compared to baseline values within the 72-hour postoperative period. The risk ratio was low (1.24) and the difference in the observed rates was not significant ($P = 0.47$). No patients went on dialysis during the immediate postoperative period. With respect to more advanced eGFR declines, there were 10 (9.1%)

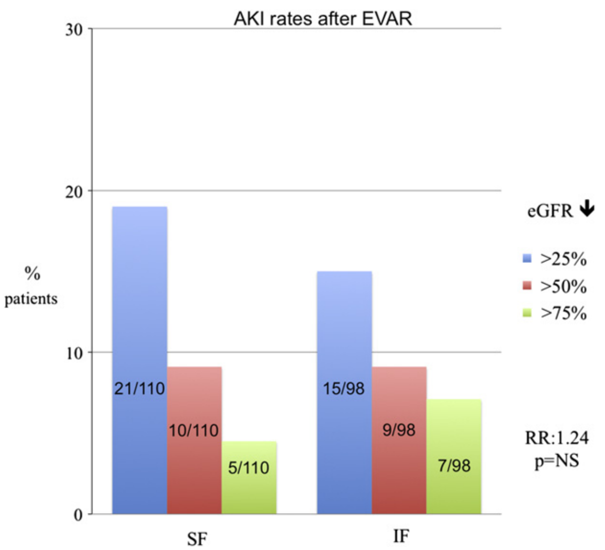

Fig. 1. Rates of AKI following EVAR for SF and IF groups using RIFLE criteria. SF, suprarenal fixation; IF, infrarenal fixation; AKI, acute kidney injury; RR, risk ratio.

patients in the SF group vs. 9 (9.1%) patients in the IF group who developed GFR decline by more than 50% ($P = 0.638$); and 5 (4.5%) patients in the SF group vs. 7 (7.1%) of patients in the IF group whose GFR declined by more than 75% ($P = 0.77$).

CKD Findings

Patients' renal function was followed for up to 24 months and checked routinely prior to CT angiography for graft surveillance. The mean follow-up for the cohort was 6.4 months with a standard deviation of 7.8 months. The freedom from progression to stage 3 or 4 CKD in the SF group was 0.76, 0.72, and 0.49 at 6, 12, and 18 months, respectively. In the IF group, freedom from progression was 0.8, 0.73, and 0.68, respectively (Fig. 2). The difference between the two groups was not significant throughout the follow-up period ($P = 0.4$). Two patients in stage IV went on to HD in the follow-up period, one from each group for a total incidence of 0.5% per group.

Risk Factors

Multivariate analysis was performed to identify potential risk factors of AKI and progressing CKD post-EVAR after adjusting for comorbidities and perioperative confounding variables (Table V). For AKI, there was a significant association with operative contrast volume) ($P < 0.001$) and a trend towards patients with perioperative angiotensin converting enzyme-inhibitors ($P = 0.07$).

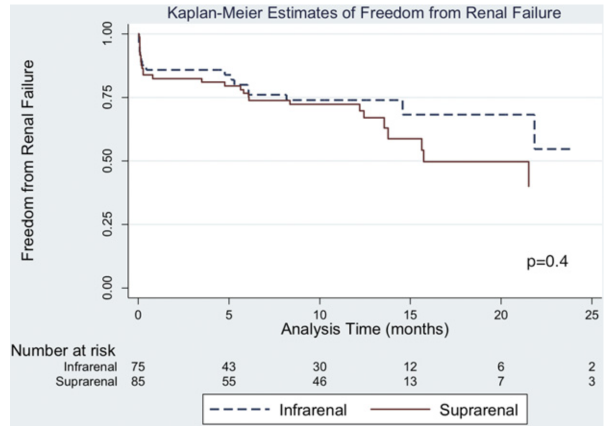

Fig. 2. Kaplan-Meier estimates of freedom from progression to CKD 3 and 4 following EVAR for both groups. CKD, chronic kidney disease.

Table V. Risk factors associated with progression to AKI and CKD after EVAR

Morbidity	Predictor	*P* value
AKI	Contrast volume (ml)	<0.001
	Perioperative Ace-i	0.07
CKD	Operative time (min)	<0.001
	Baseline renal function (eGFR < 60)	<0.001
	Age (years)	0.07

AKI, acute kidney injury; CKD, chronic kidney disease; Ace-I, angiotensin converting enzyme inhibitor.

Compromised baseline renal function was significant predictor of postoperative worsening CKD ($P < 0.001$), as well as lengthy procedures ($P < 0.001$). Increasing age was also associated with declining long-term renal function ($P = 0.07$).

DISCUSSION

The adverse impact of EVAR on renal function has been studied before and related to various possible mechanisms that may have short- or long-term effects. One of the most extensively studied pathways is contrast-induced nephropathy (CIN).[10] The use of nonionic iso-osmolar contrast, lower contrast volume (<100 ml), and perioperative IV hydration with antioxidants, all decrease the risk of CIN after endovascular interventions.[9] Adjunct techniques that may further minimize contrast load such as intravascular ultrasound (IVUS) also play a role in decreasing incidence of CIN as shown in thoracic endovascular aneurysm repairs.[11] As an alternate mechanism, coverage of small accessory renal arteries[12] and generation of microemboli during wire manipulations and stent-graft deployment may result in <10% parenchymal loss with no significant impact in renal function.[13,14] Although coverage of large (>3 mm) accessory renal arteries may result in segmental renal infarction[12] and is generally discouraged,[15] several retrospective studies report minimal adverse impact on renal function.[16] In our cohort, there was no significant renal parenchymal loss (>10%) identified on completion angiogram or follow-up CT, compared to baseline.

Furthermore, the extent of proximal fixation through and above renal artery level has raised concerns for potential further kidney injury.[17] In addition to possible microembolism from deployment of suprarenal stent, flow impedance through struts may have long-term sequelae in renal function. Lau and colleagues[17] discuss that several small series have reported parenchymal loss without actual change in renal parameters, which could be partially due to insensitive methods of monitoring renal outcomes.

In the present study, the incidence of AKI 15−19% was comparable between the groups. Other studies using similar eGFR criteria report high incidence of acute renal dysfunction with increasing complexity of the procedure.[18] Taking into consideration pathologic mechanisms of AKI, it is possible that the increased rates we observed are partially due to transient dysfunction as several of these patients seem to recover with only a fraction progressing to CKD over the long term.

The freedom from developing CKD was very similar for both groups. It seems that a continuous low risk of worsening renal function persists after EVAR throughout the follow-up period of nearly two years without statistical difference in relevance to proximal fixation. We did not identify any transient fluctuations in chronic renal function during the follow-up time-points, although temporary decline in renal function has been reported within the first year after EVAR.[18−20]

It is interesting that after adjusting for intraoperative parameters and comorbidities as confounding factors, different predictors were identified for AKI and CKD. This is expected to some extent, as these two entities occur via different underlying pathophysiologic mechanisms as discussed above. Perioperative angiotensin-converting enzyme inhibitors showed a trend towards AKI and may have a deleterious effect on short-term renal function in patients with CHF as shown by Cruz et al.[21] This adds to the importance of maintaining hemodynamic stability, especially in the presence of perioperative fluctuations in intravascular volume. Of the predictors identified for long-term renal dysfunction, aging was significant and it is a known parameter for naturally declining GFR.[7,22] Patients with baseline stage 3 or 4 CKD (eGFR $\leq$ 60 mL/min/1.73 m^2) had significantly worse renal outcomes after EVAR, which correlates with findings from other studies using similar criteria.[18] Chronic renal dysfunction patients are expected to have less reserve and be prone to deterioration from any procedure involving the pararenal aorta, surveillance contrast-dependent studies, or comorbid cardiovascular conditions alone. It is unclear in which mechanism lengthy procedures impact long-term renal function, but has been identified as risk factor in previous work[11] as well and could be explained by increasing micro-emboli due to prolonged exposure to wire and device manipulations.

The increased incident of baseline CKD stage 3 and 4 in our study (42−47%, eGFR $\leq$ 60 mL/min/1.73 m^2) is in part due to applying more sensitive criteria than creatinine cut-off points to address renal insufficiency. This correlates with increased baseline CKD rates in reports using GFR criteria (18.5−32%)[18,23] compared to lower rates (7.6−15%) reported in studies using serum creatinine alone (1.3−1.5 mg/dL) as parameter.[19,24−27]

Renal function after EVAR in relevance to proximal fixation has been studied mostly in retrospective small series. In a meta-analysis by Sun et al.,[5] no significant difference in renal outcomes was found between the groups. On the contrary, Walsh and colleagues[23] report in a more recent review that suprarenal fixation increases the risk for renal impairment in the medium term. The authors conclude that the results should be considered with caution, since the available studies use inconsistent definitions for renal impairment and most are based on retrospective analysis of non-consecutive patients. This is an important observation since the diversity in definitions and criteria for AKI and CKD do not allow for comparisons among studies and set significant limitations to the reliability of potential meta-analyses.

To address this issue, we elected to use eGFR that represents a more sensitive indicator of kidney function than serum creatinine.[6] Specifically, we elected to use the MDRD study equation to estimate GFR according to National Kidney Foundation clinical practice guidelines[7] that has been validated on a larger population and is more accurate than the older Cockcroft-Gault formula used in former studies.[28,29] We stratified our patients based on the NKF/KDOQI criteria in an attempt to address CKD in a widely accepted manner and allow for future study comparisons.

For similar reasons, AKI was classified using the RIFLE criteria that have been shown to be sensitive and an independent predictor of hospital mortality in intensive care unit and cardiovascular patients.[30,31]

We need to acknowledge specific limitations in our study. It is retrospective review and subject to sampling error. Due to short length of stay of 2−3 days with endovascular aneurysm repair, we may have missed a small percentage of acute postoperative eGFR changes that are known to occur even up to 7−10 days. Despite using sensitive parameters to monitor renal function, there are studies reporting even better accuracy with specific markers such as cystatin-C.[32,33] No female patients met the inclusion criteria, therefore the outcomes may relate only to male patients. Re-interventions to treat endoleaks or other contrast-related interventions may have introduced an additional unknown risk of renal dysfunction, but the incidence was low (4%) and equally distributed between the groups. Also, MDRD formula has decreased accuracy in patients with BMI extremes, which did not significantly affect our cohort (BMI 28 ± 5).

In conclusion, proximal fixation type for EVAR does not appear to have significant impact on either acute or chronic renal function outcomes. There are different predictors identified for AKI and CKD. Non-modifiable risk factors may be considered to better risk-stratify patients prior to an EVAR procedure regarding their anticipated renal outcomes, while identified modifiable factors could be used to further decrease risk of renal dysfunction. Prospective randomized studies are needed to validate our results, using widely accepted criteria and sensitive renal function parameters.

REFERENCES

1. Parodi J, Palmaz J, Barone H. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg 1991;5:491−9.
2. Marin M, Parsons R, Hollier L, et al. Impact of transrenal aortic endograft placement on endovascular graft repair of abdominal aortic aneurysms. J Vasc Surg 1998;28:638−46.
3. Sun Z, Zheng H. Effect of suprarenal stent struts on the renal artery with ostial calcification observed on CT virtual intravascular endoscopy. Eur J Vasc Endovasc Surg 2004;28:534−42.
4. Liffman K, Lawrence-Brown M, Semmens J, et al. Suprarenal fixation: effect on blood flow of an endoluminal stent wire across an arterial orifice. J Endovasc Ther 2003;10:260−74.
5. Sun Z, Stevenson G. Transrenal fixation of aortic stent-grafts: short- to midterm effects on renal function—a systematic review. Radiol 2006;240:65−72.
6. Jin R, Grunkemeier G, Brown J, et al. Estimated glomerular filtration rate and renal function. Ann Thorac Surg 2008;86:1−3.
7. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 2002;39(2 Suppl 1): S1−266.
8. Bellomo R, Kellum J, Ronco C. Defining and classifying acute renal failure: from advocacy to consensus and validation of the RIFLE criteria. Intensive Care Med 2007;33: 409−13.
9. Kagan A, Sheikh-Hamad D. Contrast-induced kidney injury: focus on modifiable risk factors and prophylactic strategies. Clin Cardiol 2010;33:62−6.
10. Quintavalle C, Brenca M, De Micco F, et al. In vivo and in vitro assessment of pathways involved in contrast media-induced renal cells apoptosis. Cell Death Dis 2011;2:e155.
11. Pisimisis G, Khoynezhad A, Bashir K, et al. Incidence and risk factors of renal dysfunction after thoracic endovascular aortic repair. J Thorac Cardiovasc Surg 2010;140(6 Suppl):S161−7.
12. Dorffner R, Thurnher S, Prokesch R, et al. Spiral CT during selective accessory renal artery angiography: assessment of vascular territory before aortic stent-grafting. Cardiovasc Intervent Radiol 1998;21:179−82.
13. Krämer S, Seifarth H, Pamler R, et al. Renal infarction following endovascular aortic aneurysm repair: incidence and clinical consequences. J Endovasc Ther 2002;9:98−102.
14. Mehta M, Cayne N, Veith F, et al. Relationship of proximal fixation to renal dysfunction in patients undergoing endovascular aneurysm repair. J Cardiovasc Surg 2004;45:367−74.
15. Chaikof E, Brewster D, Dalman R, et al. SVS practice guidelines for the care of patients with an abdominal aortic aneurysm: executive summary. J Vasc Surg 2009;50:880−96.
16. Greenberg J, Dorsey C, Dalman R, et al. Long-term results after accessory renal artery coverage during endovascular aortic aneurysm repair. J Vasc Surg Epub 2012 Apr 4.
17. Lau L, Hakaim A, Oldenburg W, et al. Effect of suprarenal versus infrarenal aortic endograft fixation on renal function and renal artery patency: a comparative study with intermediate follow-up. J Vasc Surg 2003;37:1162−8.
18. Haddad F, Greenberg R, Walker E, et al. Fenestrated endovascular grafting: the renal side of the story. J Vasc Surg 2005;41(2):181−90.
19. Surowiec S, Davies M, Fegley A, et al. Relationship of proximal fixation to postoperative renal dysfunction in patients with normal serum creatinine concentration. J Vasc Surg 2004;39(4):804−10.
20. O'Donnell M, Sun Z, Winder R, et al. Suprarenal fixation of endovascular aortic stent grafts: assessment of medium-term to long-term renal function by analysis of juxtarenal stent morphology. J Vasc Surg 2007;45(4):694−700.
21. Cruz C, Cruz L, Silva G, et al. Incidence and predictors of development of acute renal failure related to treatment of congestive heart failure with ACE inhibitors. Nephron Clin Pract 2007;105:c77−83.
22. Tiao J-H, Semmens J, Masarei J, et al. The effect of age on serum creatinine levels in an aging population: relevance to vascular surgery. Cardiovasc Surg 2002;10:445−51.
23. Walsh S, Boyle J, Lynch A, et al. Suprarenal endograft fixation and medium-term renal function: systematic review and meta-analysis. J Vasc Surg 2008;47:1364−70.
24. Prinssen M, Verhoeven E, Buth J, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. New Eng J Med 2004;351:1607−18.
25. Parmer S, Fairman R, Karmacharya J, et al. A comparison of renal function between open and endovascular aneurysm repair in patients with baseline chronic renal insufficiency. J Vasc Surg 2006;44:706−11.
26. Waasdorp E, van't Hullenaar C, van Herwaarden J, et al. Renal function after endovascular aortic aneurysm repair: a single-center experience with transrenal versus infrarenal fixation. J Endovasc Ther 2007;14:130−7.

27. Cayne N, Rhee S, Veith F, et al. Does transrenal fixation of aortic endografts impair renal function? J Vasc Surg 2003;38:639—44.
28. Levey A, Bosch J, Lewis J, et al. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of diet in renal disease study group. Ann Intern Med 1999;130:461—70.
29. Forbes T, Harding G, Lawlor D, et al. Comparison of renal function after endovascular aneurysm repair with different transrenally fixated endografts. J Vasc Surg 2006;44:938—42.
30. Bagshaw S, George C, Dinu I, et al. A multi-centre evaluation of the RIFLE criteria for early acute kidney injury in critically ill patients. Nephrol Dial Transplant 2008;23: 1203—10.
31. Arnaoutakis G, Bihorac A, Martin T, et al. RIFLE criteria for acute kidney injury in aortic arch surgery. J Thorac Cardiovasc Surg 2007;134:1554—60. discussion 1560—1.
32. Coll E, Botey A, Alvarez L, et al. Serum cystatin C as a new marker for noninvasive estimation of glomerular filtration rate and as a marker for early renal impairment. Am J Kidney Dis 2000;36:29—34.
33. Nilsson-Ehle P, Grubb A. New markers for the determination of GFR: iohexol clearance and cystatin C serum concentration. Kidney Int Suppl 1994;47:S17—9.