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Evidence that Statins Protect Renal Function During Endovascular Repair of AAAs☆

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Abstract *Objectives:* Several studies have documented a slight but significant deterioration of renal function after endovascular repair of abdominal aortic aneurysm (AAA) (EVAR). The aim of this retrospective study was therefore to investigate whether medication with statins may favourably affect perioperative renal function.
Material and Methods: From January 2000 to January 2008, out of a total cohort of 287 elective patients receiving endovascular repair of their AAA or aortoiliac aneurysm, 127 patients were included in the present study, as their medication was reliably retrievable. Patients were divided according to whether their medication included statins (>3 months). Second, they were subdivided according to their supra- (SR) or infrarenal (IR) endograft fixation. Serum creatinine (SCr) and creatinine (CrCl) clearance were determined preoperatively, postoperatively, at 6 and 12 months. Patients with known pre-existing renal disease, with incorrect placement of the stent graft resulting in severe renal artery stenosis, and with occlusion or renal parenchymal infarction were excluded from the study.
Results: Patients receiving an infrarenal fixation of their graft had no change in the renal function, regardless whether they were on statins or not. In patients with SR fixation not receiving statins, a deterioration in renal function was observed in the early postoperative period ((SCr) preoperative vs. SCr postoperative: 1.02 ± 0.2 vs. 1.11 ± 0.28, $p < 0.001$ and (Cr.Cl) preoperative vs. Cr.Cl postoperative: 74.1 ± 21.4 vs. 68.0 ± 21.4, $p < 0.001$), whereas patients on statins experienced no change in renal function (SCr preoperative vs. SCr postoperative: 0.99 ± 0.24 vs. 1.02 ± 0.20 n.s. and Cr.Cl preop vs. Cr.Clpostop.: 76.4 ± 19.1 vs. 74.28 ± 20.50, n.s.). During follow-up, a constant worsening of renal function at 6 and 12 months was observed, irrespective of the medication with statins.

Abbreviations: AAA, Abdominal aortic aneurysm; EVAR, Endovascular aortic repair; SR, Suprarenal; IR, Infrarenal; SCr, Serum creatinine; CrCl, Creatinine clearance.

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Conclusions: The present study suggests a slight immediate deterioration of the renal function using (SR) fixation, and this could be prevented by the use of statins. During follow-up, statins did not protect from further renal deterioration. Broader studies are needed to confirm a definitive relation between statin use and renal protection during the endovascular repair of AAA.

Although endovascular repair of abdominal aortic aneurysms (AAA) has been proven to reduce systemic ischaemia—reperfusion injury effects,[1] renal impairment occurs in a subset of patients.[2,3] The impairment is multifactorial, and various mechanisms — such as nephrotoxicity of the administered contrast medium,[4–6] micro- or macroembolisation into the renal arteries due to aortic manipulation of endovascular materials,[6,7] and an induced inflammatory response — are potentially harmful. The role of suprarenal (SR) stent fixation, as an additional factor of renal function worsening, remains unclear and debatable.[8–10]

There is accumulating evidence that statins have beneficial effects on the vascular tree, independent of their classical actions on lipoproteins. They induce an improvement of endothelial dysfunction, and an increase in nitric oxide bioavailability and antioxidant effect. Furthermore, the anti-thrombotic effect, its anti-inflammatory properties and the stabilisation of atherosclerotic plaques contribute to fewer vascular 'accidents'.[11,12] In addition, a putative immunosuppressive activity and an indirect effect, through a combination of modulating mechanisms in renal function, may play a role in patients undergoing AAA repair.[11,12]

Based on these beneficial effects of statins, we investigated the effect of statin medication on renal function of patients undergoing endovascular aortic aneurysm repair (EVAR).

Patients and Methods

Study design and population study

A retrospective, single-centre, non-randomised study was conducted to evaluate an eventual protective role of statins in renal response after EVAR in infrarenal (IR) aortic aneurysms. From January 2000 to January 2008, 287 elective patients were treated with endovascular procedures for pathology of the aorta, in our tertiary care university hospital, out of which 127 patients could be included in the study. The indication was an aneurysm larger than 5 cm, and the type of stent-graft device was selected according to the anatomic appearance of the aneurysm and in accordance with published guidelines.[13]

The database for the 127 patients was prospectively recorded, whereas the statin use was retrospectively analysed. Risk factors and clinical data were classified according to the Society for Vascular Surgery/International Society for Cardiovascular Surgery reporting standards[14] (Table 1).

End points of study

The primary end point was to investigate the hypothesis that medication with statins for more than 3 months preoperatively may positively affect postoperative renal function, compared with cases in which this medication was not taken. The secondary goal was to examine whether the type of fixation would have a negative influence on renal function and the role of statins in such a case.

Patient selection, grouping and inclusion and exclusion criteria

Of the 287 patients, there were not sufficient and available computerised data for 101 patients during the period 2000—03, for the medication, the biochemical markers and the follow-up. These 101 patients were excluded from the study. The rest of the patients were grouped into two main groups: patients receiving chronic (>3 months) medication with statins, and patients not receiving statins. Patients in these two groups were then subgrouped according to the endograft fixation type (SR or infrarenal (IR)). Patients with pre-existing vasculitis taking cortisone and patients with a history of a known preoperative renal disease or a serum creatinine (SCr) concentration >1.5 mg dl^{-1} or >132 μmol were eliminated from the study. Patients with thoracoabdominal, juxtarenal aortic aneurysms, dissecting aneurysms, aortic para-anastomotic pseudo-aneurysms and ruptured aneurysms were also excluded from this study. Finally, patients in whom an incorrect placement of the stent graft was documented, resulting in severe renal artery stenosis, occlusion and renal parenchymal infarction, were also eliminated from the study. Exclusion criteria and numbers of patients excluded are analytically described in Table 2.

Endovascular technique and contrast infused

A team of vascular surgeons performed all EVARs in an operating room equipped with a portable C-arm (Philips BV 300), with angiographic and road-mapping capabilities. During the past 2 years, the procedures were performed in an Angio-Suite operating theatre with a ceiling mounted C-Arm (Philips, Allura Xper FD20). A variety of endovascular type stent grafts were used (Table 3). Contrast medium was infused for the angiogram just before the deployment of the stent graft main body, then before deployment of the contralateral and ipsilateral iliac leg extension, to visualise the hypogastric artery, and finally, at the end of the procedure (completion angiography). The protocol of contrast medium infusion during EVAR was identical for all patients.

Renal function assessment and follow-up

The renal function was assessed by SCr concentration and its clearance preoperatively, on day 3 postoperatively at 6

Table 1 Patient demographics and preoperative risk factors. Values are expressed as mean ± S.D. and percentages. n.s.: Not significant, ASA: American Society of Anesthesiologists, BMI: Body mass index. Values in bold are statistically significant.

	Statin users ($n = 58$)	Statin no users ($n = 69$)	p Value
Male Gender	51/58 (88%)	58/69 (88.5%)	>.05
Age (Years)	70.6 ± 8.8	72.3 ± 7	>.05
BMI	27.9 ± 3.2	26.4 ± 4.4	**.03**
AAA Diameter	5.74 ± 1.1	5.9 ± 1.2	>.05
ASA Classification	2.85 ± 0.6	2.91 ± 0.4	>.05
Type I and II	25%	34%	
Type III	62%	62%	
Type IV	11%	4%	
Type V	2%	—	
Active smokers	62%	59%	>.05
Hyperlipidemia	38%	12%	**<.001**
Diabetes Mellitus	18.5%	11 %	>.05
Pulmonary Disease	29%	33%	>.05
Cardiac status			
Asymptomatic	34%	40%	>.05
Hypertension	48%	56%	>.05
Coronary Disease	55%	53%	>.05
Arrhythmic Disorders	5%	9%	>.05
Valve Insufficiency	10%	6%	>.05
Cerebrovascular disease	21%	19.5%	>.05
Type of statin			
Atorvastatin (-40 mg)	10/58 (17.2%)	—	
Fluvastatin (40 mg)	1/58 (1.7%)	—	
Lovastatin (40 mg)	2/58 (3.4%)	—	
Pravastatin (-40 mg)	6/58 (10.3%)	—	
Simvastatin (-40 mg)	39/58 (67.2%)	—	
Medications			
Antiplatelet Drugs (Asp ± Clop)	78%	77%	>.05
Calcium Antagonists	16%	13%	>.05
ACE Inhibitors	35%	32%	>.05
β-Blockers	42%	32%	>.05
Nitrates	12%	10%	>.05

and 12 months before the scheduled computed tomography (CT) control. Creatinine clearance (CrCl) was then calculated according to the Cockcroft–Gault formula.[15]

Helical CT scans were performed on each patient postoperatively before admission, at 6 and 12 months, and then annually thereafter. CT at shorter intervals or angiography was performed when indicated. The follow-up CT angiograms were used not only to identify the presence of endoleaks or stent migration and to confirm the patency of the stent graft and renal arteries, but also to detect the presence of any renal parenchymal perfusion defects.

Drug medication

All prescriptions and medications were noted by confirmed intake of the patient and the referral letter from the general practitioner. Drugs listed were as follows: statins,

Table 2 Exclusion criteria. In parenthesis the number of patients excluded.

Patients with known preexisting renal disease, SCr > 1.5 mg/dL or >132 micromol (21)
Patients with preexisted vasculitis affecting the kidneys, and patients receiving cortisone (2)
History of nephrectomy, or cancer of the urologic system (3)
Thoracoabdominal, dissecting, juxtarenal aortic and para-anastomotic aortic aneurysms (23)
Symptomatic and ruptured aneurysms (5)
Procedures complicated by peripheral thromboembolism, limb ischemia and myoglobinuria (2)
Patients with inappropriate endostent placement, and patients with covered polar (accessory) artery (3)
Patients in which medication with or without statins could not be determined (101)

Table 3 Operation characteristics and endostent type. n.d.: not determined.

	Statin users ($n = 58$)	Statin not users ($n = 69$)	p Value
Suprarenal fixation	31/58 (53%)	42/69 (61%)	>.05
Endostent type			n.d.
Anaconda	7 (12%)	8(11.6%)	
AneuRX	—	1(1.45%)	
Endofit	1(1.75%)	—	
Endologix	1(1.75%)	1(1.45%)	
Endurant	2 (3.5%)	—	
Excluder	18 (31%)	15(21.7%)	
Fortron	—	2(2.9%)	
Lifepath	—	2(2.9%)	
Talent	3(5%)	3(4.3%)	
Zenith	26(45%)	37(53.6%)	
Operative Time (min)	136 ± 33 (63—189)	144 ± 42 (85—320)	>.05
Blood loss (ml)	447 ± 362 (100—2400)	385 ± 242 (0—1000)	>.05
Postoperative Discharge	6.7 ± 2.4(4—15)	7.7 ± 3.6 (3—21)	>.05
30-d Mortality	0%	0%	—

anti-platelets, β-blockers, angiotensin-converting enzyme inhibitors, calcium channel blockers, nitrates and digoxin. The medication was not discontinued before or after the operation. Patients unable to take medications orally perioperatively were switched to intravenous formulas, or the oral medications were restarted as soon as possible after surgery — normally, the following day.

Statistical analysis

Descriptive statistics were completed with a mean ± S.D. The comparison of continuous variables was made by using the Mann—Whitney test for independent variables and the Wilcoxon signed-rank test for dependent variables. Categorical variables were compared with the Fisher's exact test. Differences were considered significant if the p value was <0.05. All evaluations were performed using the statistical package SPSS for MS-Windows, release 17.0 (SPSS, Chicago, IL, USA).

Results

All 127 patients underwent successful placements of a stent graft. Patients' characteristics and risk factors are analytically described in Table 1, while operation characteristics and stent-graft type-related data are included in Table 3. Fifty-eight (46%) of the 127 patients received a chronic medication with statins. Seventy-three patients (57%) had a SR attachment of the graft.

No episodes of acute renal failure requiring haemodialysis occurred during the perioperative period. An increase in SCr > 30% was observed in 7% (9/127) of patients, while a rise in SCr < 30% was found in 55% (70/127) of all patients.

When considering the entire group of EVAR patients, a statistically significant increase of SCr (1.04 ± 0.22 vs. 1.07 ± 0.26; $p = 0.04$) and a decrease of CrCl (74.7 ± 20.7 vs. 64.16 ± 19.1, $p = 0.019$) were observed between the preoperative and postoperative periods. During follow-up, further deterioration of the renal function after 6 months and 12 months was also observed (Table 6 available online).

Statins vs. no statins

Both groups showed similarities, especially in their demographics, and also revealed certain differences, particularly in terms of their risk profiles (Table 1). Baseline preoperative SCr and glomerular filtration rate (GFR) were similar between the two groups (Table 4). In patients receiving statins, SCr remained unchanged (SCrpreop vs. SCrpostop.: 1.03 ± 0.23 vs. 1.02 ± 0.21, $p > 0.05$), as did CrCl (CrClpreoperative vs. CrClpostoperative :75.9 ± 20.2 vs. 76.79 ± 20.8, $p > 0.05$), in contrast to non-statin users (SCrpreop vs. SCrpostop.: 1.02 ± 0.2 vs. 1.11 ± 0.28, $p < 0.001$ and CrClpreop vs. CrClpostop.: 74.1 ± 21.4 vs. 68.0 ± 21.4, $p < .001$). An increase in SCr > 30% was observed in 5.1% of patients receiving statins, and in 8.7% of patients who were not under statin prescriptions ($p < 0.05$). A less than 30% rise in SCr, associated with a decrease in CrCl, was found in 43.1% of statin users and in 65.3% of no users ($p < 0.05$). A stable creatinine value was documented in 51.8% of statin users and in 26% of non-statin users ($p < 0.05$) (Table 5).

During follow-up, a statistically significant rise in renal function markers was observed at 6 and 12 months in both groups.

IR vs. SR fixation

There was no difference in procedure time, estimated blood loss or contrast volume between the IR and SR fixation groups. Baseline preoperative levels of SCr were also similar between the two groups. Although the preoperative median CrCl value was numerically slightly higher in the SR fixation group, this was not statistically different to the value in the IR fixation group (Table 7 available online).

There were no relevant changes in the SCr and the CrCl in the IR fixation group after EVAR. On the contrary, the postoperative SCr value, as well as the CrCl, differed significantly from the preoperative values in patients with

Table 4 Subgroup analysis between statin users and non-statin users. Mean SCr, CrCl in statin users and no users. p value[1] preop. compared with postop, p value[2] preop. compared with 6 months, p value[3] preop. compared with 12 months. Postop. (3): Postoperatively on day 3. Values in bold are statistically significant.

	Statin users ($n = 58$)	Statin no users ($n = 69$)	p Value
Preoperative baseline values			
SCr	1.03 ± 0.23	1.04 ± 0.2	>.05
CrCl	75.9 ± 20.2	73.7 ± 21.2	>.05
Postoperatively on day 3			
>30 % ↑SCr	5.1% (3/58)	8.7% (6/69)	**<.05**
<30 % ↑SCr	43.1% (25/58)	65.3% (45/69)	**<.05**
Unchanged SCr	51.8% (30/58)	26% (18/69)	**<.05**
SCr	1.02 ± 0.21	1.11 ± 0.28	**<.05**
CrCl	76.79 ± 20.8	68.0 ± 21.4	**.023**
At 6 months			
SCr	1.10 ± 0.13	1.15 ± 0.24	>.05
CrCl	68.0 ± 14.0	61.3 ± 21.3	>.05
At 12 months			
(SCr)	1.12 ± 0.16	1.21 ± 0.23	>.05
CrCl	66.06 ± 16.0	56.8 ± 19.0	>.05

Patients receiving statins ($n = 58$)

	Preop	Postop	6 months	12 months	p Value[1]	p Value[2]	p Value[3]
SCr	1.03 ± 0.23	1.02 ± 0.21	1.10 ± 0.13	1.12 ± 0.16	>.05	.017	<.001
CrCl	75.9 ± 20.2	76.79 ± 20.8	68.0 ± 14.0	66.06 ± 16.0	>.05	<.001	<.001

Patients not receiving statins ($n = 69$)

	Preop	Postop (3)	6 months	12 months	p Value[1]	p Value[2]	p Value[3]
SCr	1.02 ± 0.2	1.11 ± 0.28	1.15 ± 0.24	1.21 ± 0.23	<.001	<.001	<.001
CrCl	74.1 ± 21.4	68.0 ± 21.4	61.3 ± 21.3	56.8 ± 19.0	<.001	<.001	<.001

SR fixation, indicating an influence over the type of fixation in renal function.

During follow-up, both types of patients experienced steady worsening of renal function groups at 6 and 12 months.

Statin and SR fixation

A relatively low number of patients were included in the subgroup analysis correlating medication with or without statins, in patients with SR fixation. Patients taking statins had no deterioration of renal function during the immediate postoperative period (SCrpreop vs. SCrpostop.: 0.99 ± 0.24 vs. 1.02 ± 0.20 $p > .05$ and CrClpreop vs. CrClpostop.: 76.4 ± 19.1 vs. 74.28 ± 20.50, $p > .05$), whereas there was a steady decline in renal function during follow-up, even among patients taking statins (Table 5).

Discussion

The present study confirms previous reported studies, emphasising a slight but significant postoperative deterioration of renal function after endovascular reconstruction in AAA patients ranging from 3% to 7%.[16,17] In this study, a 3% change was observed in SCr, and a 12% change in CrCl. The subgroup analysis of the study revealed that this deterioration was observed in patients experiencing suprarenal fixation. Patients receiving statins did not suffer from a decrease in renal function in the postoperative period, as opposed to non-statin users. The subgroup of individuals with SR fixation prescribed to statins preoperatively did not experience any worsening in renal function. Deterioration of renal function during the 12 months of follow-up was observed independently of the medication with statins.

Today, for the treatment of vascular patients, the use of statins is widely applied in clinical practice. As 3-hydroxy 3-methylglutaryl (HMG)-CoA reductase inhibitors became more widely used, their effects beyond lipid lowering began to emerge. Several trials have documented that among patients undergoing major vascular surgery, statins exert cardioprotective effects and reduce postoperative mortality rates.[18–20]. Only indirect data exist for the role of statins in protecting renal function in patients undergoing EVAR. Several studies indicate that statins improve renal function in the general population of patients with peripheral artery disease (PAD) and lead to a lower progression of kidney failure.[21,22] Another clinical study assessing the effects of statins in patients undergoing SR, aortic cross-clamping for aortic surgery suggested an association between statins and a preserved renal function, by reducing reperfusion injury effects.[23]

Table 5 Subgroup analysis correlating medication with or without statins in patients with suprarenal endostent fixation. Mean serum creatinine concentration, creatinine clearance and incidence of >30% increase in serum creatinine levels, within a determined period of time. Postop. (3): Postoperatively on day 3. Values in bold are statistically significant.

	Statin users and SR ($n = 31$)	Statin no users and SR ($n = 42$)	p Value
Preoperative baseline values			
Serum creatinine (SCr)	0.99 ± 0.24	1.03 ± 0.22	>.05
Creatinine clearance	76.4 ± 19.1	78.7 ± 22.3	>.05
Postoperatively on day 3			
Serum creatinine	1.02 ± 0.23	1.1 ± 0.28	>.05
Creatinine Clearance	74.47 ± 20.5	71.59 ± 22.2	>.05
At 6 months			
Serum creatinine	1.09 ± 0.14	1.15 ± 0.24	>.05
Creatinine Clearance	67.73 ± 17.44	68.68 ± 21.76	>.05
At 12 months			
Serum creatinine (SCr)	1.11 ± 0.12	1.23 ± 0.27	>.05
Creatinine Clearance	62.79 ± 18.68	63.41 ± 20.98	>.05

SF and Statin users Group ($n = 31$)

	Preop	Postop	6 months	12 months	p value[1]	p value[2]	p value[3]
SCr	0.99 ± 0.24	1.02 ± 0.23	1.09 ± 0.14	1.11 ± 0.12	>.05	>.05	**<.001**
CrCl	76.4 ± 19.1	74.47 ± 20.5	67.7 ± 17.4	62.79 ± 18.6	>.05	**.007**	**<.001**

SF and no Statin users Group ($n = 42$)

	Preop	Postop. (3)	6 months	12 months	p value[1]	p value[2]	p value[3]
SCr	1.03 ± 0.22	1.1 ± 0.28	1.15 ± 0.24	1.23 ± 0.27	**.003**	**.002**	**.003**
CrCl	78.7 ± 22.3	71.59 ± 22.2	68.6 ± 21.7	63.41 ± 20.9	**.001**	**.001**	**.001**

Attempting to interpret the results of the present study, which indicates an early protective role of statins in renal function after EVAR, emphasis should be given to five important and relevant pleiotropic effects of statins: (1) improved endothelial function, (2) modulated inflammatory response, (3) decreased oxidative stress, (4) maintained plaque stability[24,25] and (5) prevention of thrombus formation.[26,27] These effects could positively interfere with various mechanisms involved in renal dysfunction after EVAR. The renal dysfunction after EVAR seems to be a multifactorial process. Intra-aortic manipulation with guide wires, catheters and introducer sheaths may lead to the disruption of arterial plaques and subsequent embolisation into the renal arteries.[7,8] There is evidence that microembolisation is more common in patients undergoing EVAR, compared with conventional open surgery.[28] Statins may induce plaque stability[24,25] and potentially prevent thrombus[26,27] and microemboli, leading to a preservation of kidney parenchyma integrity.

Renal dysfunction may also be induced by the potential nephrotoxicity of the relatively high dose of administered contrast medium during endovascular procedures.[4–6] Patients during endovascular reconstruction may potentially receive a large amount of contrast medium. The pathways for contrast medium-induced nephrotoxicity include haemodynamic effects, molecule toxicity and endogenous biochemical disturbances, resulting in tubular cell injury.[29] An increase in oxygen-free radicals and a decrease in antioxidant enzyme activity, triggered by contrast medium administration, have been documented as cellular mechanisms of damage.[30,31] The levels of advanced inflammatory response end products appear to increase as GFR decreases. Furthermore, on the basis that the reactive oxygen species could be involved in the pathophysiology of contrast-induced renal dysfunction, a potential prophylactic effect of preoperative *N*-acetylcysteine administration has been suggested.[32] Attenuation of the inflammatory response is considered a possible explanation for the renoprotective actions of statin therapy. Statins may promote potent systemic antioxidant effects through the suppression of distinct oxidation and anti-inflammatory pathways.[33,34] In addition, they reduce endothelial dysfunction, primarily through their ability to enhance endothelial nitric oxide bioavailability.[33,34]

The SR fixation is potentially involved in micro-embolic processes and chronic arterial trauma in renal ostia, causing an additional aggravation in renal function,[28,35] although a recent meta-analysis study concluded that the role of transrenal fixation in renal function remains unclear and debatable, due to the absence of convincing data.[8] In the present study, there was a clear impairment of renal function in patients with SR fixation, who were not statin users. However, patients with SR fixation and taking statins did not experience deterioration in renal function. This indicates the possible role of statins in promoting plaque stability around renal ostia and preventing micro-embolism and thrombus formation.[24–27]

An important finding in our study was continuous renal deterioration through the 12 months of follow-up, which was witnessed independently of the use of statins and the type of fixation. A tendency towards decelerated deterioration at 6 and 12 months of follow-up in patients receiving

statins was observed, but this was not statistically significant. A possible explanation is that statins may play a stabilising role in the postoperative period, during which complex immune, inflammatory and thrombo-embolic mechanisms are involved. Whether statins have potential long-term reno-protective effects is unclear due to relatively controversial data and the heterogeneity of results.[36–38] In addition, risk factors that present in cardiovascular patients – diabetes mellitus (DM), uncontrolled hypertension, progressive atherosclerosis, smoking and advancing age – can explain further gradual impairment in renal function. There is evidence that statins may protect kidneys via cholesterol reduction,[39] as well as through their pleiotropic effects upon patients with chronic kidney disease.[40,41] However, it is also believed that statins may induce proteinuria, impeding albumin uptake by receptor-mediated endocytosis.[42] Although subgroup analyses of major clinical studies and meta-analyses of smaller trials tend to indicate that statin therapy may stabilise the GFR, further studies are required to confirm the benefit of statins in specific populations of patients.[36,40,41] The gold standard for monitoring renal function is the measurement of GFRs; however, patient compliance for GFR is often poor. Serum cystatin C, as a more accurate marker of GFR, is well documented and may be appropriate for further studies.[43]

There are limitations to the present study. A direct effect of contrast media on the kidney and toxic effects on the tubular cells are involved in the pathophysiology of contrast medium-induced nephropathy. The amount of contrast medium administration in each endovascular procedure was not measured due to absence of available data. However, as was previously described, an identical protocol of contrast medium infusion was applied in all patients (just before the deployment of the stent graft main body, then before deployment of the contralateral and ipsilateral iliac leg extension, in order to visualize the hypogastric artery, and finally, at the end of the procedure) and the operative time was not different between the two groups. A second limitation of the study is that the sample size is relatively small, and larger, perhaps even multicentre trials are needed to define the role of statin use for postoperative renal protection in EVAR. The most important drawback is the retrospective analysis, but it is questionable if it would be possible to perform a randomised controlled study, as most patients with AAA are in need of statins. The final question is whether all statins are uniformly effective, or whether type and dosage amount impact effectiveness.

Conclusions

Statins significantly reduce cardiovascular 'accidents.' The present study provides evidence that renal function may be preserved, due to statins, in patients receiving SR fixation for AAA during the postoperative period, compared to non-statin users. Broader studies are needed to definitely confirm this relation.

This is, to our knowledge, the first report attempting to investigate the role of statins in renal function protection after EVAR.

Conflict of Interest/Funding

None.

Appendix
Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi 10.1016/j.ejvs.2010.05.006.

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