Review Article

Coronary Revascularisation in Patients with Chronic Renal Disease

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Patients with chronic renal disease make up a steadily growing population. This is especially significant from a clinical point of view, since these patients are affected to a disproportionate degree by cardiovascular disease, including coronary artery disease.[1,2] A creatinine clearance rate below 60 ml/min/m^2 is a marker of poor prognosis for a number of cardiovascular outcomes.[3] This is especially true for patients with end-stage renal disease (ESRD), where cardiovascular mortality accounts for around 50% of the total.[2] Acute coronary syndromes (ACS) are very common in these patients[4] and have an extremely unfavourable long-term prognosis, with two-year survival being just 30%.[5] This disproportionate impact of cardiovascular events on patients with renal disease is due to the exceptionally atherogenic environment created by a combination of classical risk factors,[6] such as diabetes mellitus, arterial hypertension and dyslipidaemia, with other, novel factors, such as increased oxidative burden,[7] subclinical inflammation[8,9] and malnutrition.

However, part of the responsibility for the cardiovascular morbidity and mortality of patients with renal disease rests with the limitations on their use of established medications, such as β-blockers, statins, acetylsalicylic acid,[4,10] and thrombolytic agents.[5] In particular, the likelihood of administration of acetylsalicylic acid and β-blockers decreases in parallel with the patient's creatinine clearance on admission for myocardial infarction, while in-hospital mortality increases proportionally.[11] As regards angiotensin-converting enzyme inhibitors, one study showed that their use improved the long-term prognosis of patients who were admitted for ACS, provided that creatinine clearance on admission was <64 ml/min/m^2. Other medications used in patients with ACS (aspirin, standard and low molecular weight heparin, statins and β-blockers) showed no statistically significant interaction with creatinine clearance as regards clinical outcome. The authors commented that dose-titration strategies are still unclear as concerns thrombolytic therapy and platelet glycoprotein IIb/IIIa inhibitors.[12] Other researchers consider that thrombolysis has been tested sufficiently in patients with compromised renal function, while IIb/IIIa inhibitors do not increase the rate of haemorrhage in these patients.[13]

Apart from drug treatment, clinicians have proved to be extremely reluctant to refer the ACS patient with renal failure to the catheterisation laboratory, and even more so with respect to revascularisation, using either percutaneous transluminal coronary angioplasty or coronary artery bypass grafting (CABG).[14] One major

contribution to this phenomenon has been the absence of guidelines for patients with renal disease, who have tended to be excluded from large clinical studies in the field of ACS.[15] In consequence, there is confusion regarding the choice of revascularisation method—i.e. angioplasty with or without stenting, or CABG—in patients with renal disease and coronary artery disease (chronic or after ACS). A review of the available literature would be useful, given the frequency with which this clinical dilemma arises in everyday clinical practice.

Percutaneous transluminal coronary angioplasty (PTCA)

The results of PTCA should be presented in chronological order, starting with conventional angioplasty, i.e. without stenting (Table 1). It became apparent early on (1997) that the method was associated with a very high restenosis rate, above 50%, and therefore a frequent need for further revascularisation, with either PTCA or CABG.[16] Similarly, in a retrospective study of 362 patients with renal failure (serum creatinine >1.5 mg/dL), compared with 2972 patients with normal renal function, PTCA demonstrated a relatively lower success rate in opening the culprit lesion (89.5% versus 92.9%), a higher incidence of in-hospital major cardiovascular events (10.8% versus 1.8%), and much poorer long-term survival (6.1% versus 27.7%).[17] It is interesting to note that there were no significant differences between patients who were under haemodialysis and those who were not. The same group of investigators confirmed in a later study that in patients with chronic renal disease serum creatinine was not a prognostic factor for an adverse outcome after conventional PTCA.[18] Another study that included patients with the entire spectrum of renal failure found not only a smaller success rate for vessel opening and lower two-year event-free survival (54% versus 69%) compared with controls, but also greater cardiac and all-cause mortality.[19] A later case-control study found, in contrast to earlier ones, a similar success rate for opening coronary vessels in patients with impaired renal function, although in-hospital complications and long-term mortality were again worse in patients with renal disease (12.1% versus 0% and 27.3% versus 10.6%, respectively).[20] However, that study did not include patients with ESRD. To conclude, studies of conventional PTCA showed clearly worse outcomes in patients with renal disease compared to controls, mainly because of the high restenosis rate.

The introduction of the new technology of stents to PTCA brought hope of a reduction in restenosis rates in patients with renal failure. Indeed, in a study of patients with renal disease who underwent PTCA with or without stenting (i.e. bare metal stents, BMS), there was a clear reduction in major cardiac events and mortality, from 71% to 30%, in patients in whom a stent was implanted.[21] An interesting study by Stigant et al evaluated the outcome in patients with renal disease who underwent PTCA during a time when stents were little-known, compared with a period when they were widely used. The study included 1879 patients with renal failure of various stages and found fewer cardiac events in those who underwent the procedure when the use of stents was prevalent.[22]

A similar success in reducing restenosis following PTCA was achieved with intracoronary irradiation, since the restenosis rate and need for target vessel revascularisation were reduced from 53.8% to 22.6%, and from 78.6% to 23.7%, respectively, namely to levels comparable with those of patients with normal renal function.[23] However, the total morbidity and mortality remained higher in patients with renal failure (7.6% versus 1.9%), in spite of the reduction in restenosis.

In recent years, the use of drug-eluting stents (DES) in patients with renal dysfunction has brought satisfactory results, comparable with those seen in the general population. Specifically, a recent study that compared patients on haemodialysis with coronary patients who had normal renal function found a better perioperative result and lower in-hospital and later mortality in the latter group—i.e. the same as in the conventional PTCA era—with the difference, however, that the restenosis rate was the same in the two groups. Thus, the observed difference in outcomes was probably due to the higher comorbidity of the renal disease patients and not to factors associated with stent implantation.[24] Indeed, other investigators found comparable mortality and incidence of cardiovascular events, specifically new acute myocardial infarction and restenosis, in patients with renal disease (though not end-stage) and controls. However, this finding was not confirmed in other patient series.[26] In view of the above, it seems that DES are a worthwhile development for patients with renal disease who undergo PTCA. Although they do not produce the same survival rate as in patients with normal renal func-

Table 1. Clinical studies of the use of percutaneous angioplasty in patients with different degrees of renal dysfunction.

Author/ Study year	Number/Type of patients	Revascularisation method	Successful opening of vessel (%)	Survival(%)	MACE (%)	Other outcome	Other findings
Rubenstein 2000[17]	362 CRF (mild to ESRD) vs. Ctrl	PTCA	89.5 vs. 92.9*	6.1 vs. 27.7*	51 vs. 33*		ESRD = nonESRD
Asinger 2001[19]	77 CRF (mild to ESRD) vs. Ctrl	PTCA	89 vs. 97*			One-year freedom from cardiac events 54 vs. 69*	ESRD = nonESRD
Reinecke 2003[20]	66 CRF (not ESRD) vs. Ctrl	PTCA	85 vs. 83	72.7 vs. 89.4*		In-hospital complications 12.1 vs. 0	Increased creatinine, reduced survival
Malanuk 2001[21]	19 ESRD vs. Ctrl	PTCA with BMS	-	71 vs. 30*†		-	-
Stigant 2005[22]	780 CRF (era when stents little known) vs. 1099 CRF (era when stents widely used)	PTCA with or without stent	-	-	HR 0.61* in favour of era when stents widely used	-	Benefit at all levels of renal function
Hassani 2006[24]	72 ESRD vs. 3370 Ctrl	PTCA with DES	96 vs. 98	16 vs. 3.8*	23 vs. 8.1*	Similar restenosis rates	Increased comorbidity of ESRD
Halkin 2005[25]	223 non-ESRD vs. 419 mild CRF vs. 658 Ctrl	Randomisation to BMS or DES	-	99.6 vs. 100 vs. 99.8	3.6 vs. 1.4 vs. 3.6	Similar stent thrombosis	BMS = DES as regards survival and MACE, but DES had less restenosis
Zhang 2006[26]	410 non-ESRD vs. 602 Ctrl Non-ESRD: 146 BMS vs. 264 DES	Randomisation to BMS or DES	- -	92.7 vs. 97.7* 89.1 vs. 94.7*	18.3 vs. 13.8* 24.6 vs. 15.1*	- -	- -
Das 2006[27]	89 ESRD	PTCA with DES or BMS	-	-	-	TVR: 4 vs. 26* Death+AMI+TVR: 33 vs. 60*	-

= – equivalent outcomes; AMI – acute myocardial infarction; BMS – bare metal stent; CRF – chronic renal failure; Ctrl – control group (normal renal function); DES – drug-eluting stent; ESRD – end-stage renal disease; HR – hazard ratio; MACE: major adverse coronary events; nonESRD – non-end-stage CRF; PTCA percutaneous transluminal coronary angioplasty; TVR – target vessel revascularisation.
All outcomes as percentages (%). *$p<0.05$. †Combined endpoint of death and MACE.

tion, they do reduce the restenosis rate and thus the risk of ACS and need for revascularisation. In any case, DES have proved clearly more effective than BMS in all relevant studies. In a subanalysis of the TAXUS-IV trial, which showed the superiority of paclitaxel-eluting stents over BMS in the general population, patients with renal dysfunction showed lower restenosis rates (2.1% versus 20.5%) and less need for revascularisation (3.3% versus 12.2%) at 9 and 12 months.[25] In the study of Zhang et al, involving 410 patients with a moderate degree of renal failure who underwent PTCA with DES or BMS, both total mortality (5.3% versus 10.9%) and cardiovascular morbidity (15.1% versus 24.6%) were lower in the DES group after a mean period of 17 months.[26] Finally, in another study, DES compared to BMS reduced the need for target vessel revascularisation at 9 months in 89 patients who were under haemodialysis, despite the fact that ischaemic events remained at high levels.[27] In conclusion, stents, and particularly DES, have clearly reduced post-PTCA restenosis rates in patients with renal disease, without, however, reducing cardiovascular mortality and morbidity to levels comparable with those of coronary patients who have normal renal function.

As far as primary PTCA is concerned, only limited data are available. However, they leave no doubt concerning the less favourable outcome of patients with impaired renal function after this procedure. A systematic Japanese registry of 1359 patients who underwent primary PTCA and were divided into three categories of renal function (normal: creatinine <1.2 mg/dL, moderately impaired: 1.2-2 mg/dL, and severely impaired: >2 mg/dL) found a steady progression in in-hospital mortality from the first group to the third (3.9%, 17.1% and 34.5%, respectively).[28] However, this observation does not decrease the value of primary PTCA in patients with chronic renal disease: in fact, it is believed that the benefit is greater in this group compared to patients with normal renal function.[13]

At this point, special mention should be made of nephropathy caused by contrast media, a significant complication of percutaneous interventions that is encountered more and more frequently. This concerns a deterioration in renal function by 0.5 mg/dL serum creatinine, or by 25% during the first 24 hours after the administration of contrast medium (peaking at 3 days and disappearing at 10).[29] The risk factors for contrast-induced nephropathy include diabetes mellitus, hypovolaemia, heart failure (with use of inotropes or intraaortic balloon pump), infusion of large quantities of contrast medium, and finally pre-existing renal dysfunction, which is a fundamental predictive factor for the appearance of this form of nephropathy[30] and is also a marker for unfavourable in-hospital and long-term outcome.[31] Specifically, it was found that patients with initial serum creatinine between 1.6 and 2 mg/dL developed nephropathy requiring haemodialysis in about 1% of cases; for creatinine levels between 2.1 and 3 mg/dL the rate was 5%, and for creatinine >3 mg/dL 11%.[32] The equivalent percentages after CABG have not been elucidated in the literature.

Some quite simple clinical algorithms have been developed for the prediction of the risk of development of contrast-induced nephropathy after percutaneous coronary intervention,[33] in which the major components are the volume of contrast infused and pre-existing renal failure, defined either as serum creatinine >1.5 mg/dL or as creatinine clearance <60 ml/min/1.73m^2, with greater weight being given as the latter reduces to 40, 20 and <20 ml/min/1.73m^2. As regards the quantity of contrast medium administered, when this divided by serum creatinine in mg/dL exceeds 5 ml/kg body weight there is a high possibility of contrast-induced nephropathy requiring haemodialysis.[32] For this reason, staged procedures should be preferred in patients with renal disease, so as to use the smallest possible quantity of contrast medium per session.[32] In addition, all practical means should be used for the minimisation of contrast use, such as the avoidance of test injections and the creation of landmarks with the use of marker wires.[34] Other means of protecting against contrast-induced nephropathy[29,35] include the use of agents with osmolality less than or equal to that of plasma, hydration with normal saline at a rate of 1 ml/kg body weight per hour for 12 hours before and 12 hours after the session, and the discontinuation of metformin, diuretics and non-steroid anti-inflammatory drugs during the procedure. Measures such as the preventive administration of N-acetylcysteine, forced diuresis, or alkalinisation of urine, have not proved their efficacy and are not recommended as standard practice.[29]

CABG in patients with kidney failure

The prognosis after surgical intervention, whether it be CABG or valve replacement, or both, is less favourable

in patients with ESRD.[36] Even a moderate degree of renal failure (serum creatinine <2.5 mg/dL) is associated with an increase in the short-term and long-term morbidity and mortality after CABG in comparison with patients who have normal renal function.[37] In the world literature there are reports of small series of patients under haemodialysis who underwent classical "on-pump" CABG.[38-42] The perioperative mortality in those studies reached 5-12%, a level that is generally considered acceptable,[38,39,42] while the incidence of haemorrhagic complications and the days of hospitalisation were greater than in the control group.[41,42] The five-year survival ranged from 60-70% and was higher than in the general population of patients with ESRD.[40] Some researchers reported an improvement of symptoms, for example angina, which was not, however, necessarily accompanied by an improvement in quality of life.[39] It should be noted that in such patients, CABG is usually used to treat multi-vessel disease (three-vessel in 50%, main stem in 20%) and the vessels are also extremely calcified, making the surgical manipulations considerably more difficult. Furthermore, surgical manipulation of a similarly calcified aorta involves high risk, while in addition the extracorporeal circulation places a further burden on the already stimulated coagulatory system of the patient with renal disease.

In recent years, "off-pump" CABG, a reliable alternative to the classical technique in the general population, has also been used in quite a large number of patients with renal disease and the safety of the method has been confirmed in this patient population.[43-47] Tabata et al, in a study of 402 patients (68 with renal dysfunction) who underwent off-pump CABG, found that the degree of renal dysfunction did not affect the early outcome, which was similar in patients with renal disease and in those with normal kidney function.[44] Excellent results from off-pump CABG and comparable outcomes in patients under haemodialysis or not were reported in another study.[45] However, it is too early to draw firm conclusions concerning the superiority of the off-pump over the on-pump technique. Data from the USA show that during the two years 2000-2001 only one in six patients with ESRD underwent off-pump CABG, which was, however, associated with a 16% reduction in total mortality compared to the classical method.[46] In contrast, other investigators reported better long-term survival in haemodialysis patients who underwent on-pump CABG (annual mortality 19% versus 38.1%), despite the fact that the off-pump group had clearly better perioperative mortality (1.7% versus 17.2%).[47] The authors attributed this to the fact that the number of revascularised vessels was on average lower in the off-pump group, meaning that revascularisation was more complete in the group undergoing the classical procedure. To summarise, CABG is a satisfactory method of revascularisation in patients with renal failure, with outcomes worse than in the general population, but with the possibility of better results as the off-pump technique finds wider application.

Comparison of outcomes between PTCA and CABG

Only a limited number of studies have compared PTCA with CABG in patients with chronic renal disease (Table 2). They were almost all retrospective studies, in which the investigators searched registries covering many years, either national or from their own centres, looking for patients suffering from renal failure who underwent PTCA or CABG in order to compare the outcomes of the two methods. Of course, such studies have limitations, the main one being the fact that the respective patient groups may not be entirely comparable.

In the first studies of this kind, CABG was proved to be superior to conventional PTCA. In 1995, Rinehart et al compared 24 dialysis patients who underwent conventional PTCA with 60 who underwent CABG and found a higher incidence of cardiovascular events (including cardiovascular death) in the former group over a two-year period, although survival was similar.[48] A similar superiority for CABG was found in another retrospective study, where there was an absence of cardiac morbidity at five years in 70% of the CABG patients compared with only 18% in the PTCA group.[49] Simsir et al demonstrated comparable periprocedural mortality and complication rates for patients with ESRD undergoing CABG or PTCA, although the incidence of new cardiovascular episodes was significantly higher in the latter group, mainly because of restenosis and the consequent need for revascularisation.[50] A German study found similar 12- and 24-month survival for CABG (93% and 86%, respectively) and PTCA (95% and 82%, respectively), with a greater need for a further revascularisation procedure in the PTCA group.[51] In contrast to these small-scale studies, where

Table 2. Comparison of outcomes of percutaneous transluminal coronary angioplasty (PTCA), with or without stenting, and coronary artery bypass grafting (CABG) in patients with different degrees of renal dysfunction.

Author/ Study year	Study groups	Survival (%)	Other endpoint (%)	Other outcome	Other finding
Rinehart 1995[48]	ESRD: 24 PTCA vs. 60 CABG	51 vs. 66	Angina, AMI, cardiovascular death more frequent after PTCA	-	More severe CAD in CABG group
Koyanagi 1996 [49]	ESRD: 20 PTCA vs. 23 CABG	-	5-year event free 18 vs. 70*	-	Restenosis rate 70% after PTCA
Simsir 1998[50]	ESRD: 19 PTCA vs. 22 CABG	69 vs. 67	1.5-year event free 40 vs. 87*	-	Similar perioperative mortality/complications
Ivens 2001[51]	ESRD: 40 PTCA vs. 65 CABG	1-year: 95 vs. 93 2-year: 82 vs.86	Repeat procedure rate 57.5 vs. 3.1*	Angina rarer after PTCA	More severe CAD in CABG group
Herzog 1999[52]	ESRD: 6887 PTCA vs. 7419 CABG	RR=0.91* in favour of CABG	AMI+cardiac death RR=0.69* in favour of CABG	In-hospital mortality 5.4 vs. 12.5*	More severe CAD not ruled out in PTCA group
Szczech 2001[53]	ESRD: 163 PTCA vs. 244 CABG NonESRD: 270 PTCA vs. 570 CABG	ESRD: RR=0.39* in favour of CABG NonESRD: RR=0.86	-	-	Outcomes in ESRD generally worse than in nonESRD
Herzog 2002 54	ESRD: 4836 PTCA vs. 4280 BMS vs. 6668 CABG	CABG vs. PTCA: RR=0.8* BMS vs. PTCA: RR=0.94*	-	In-hospital mortality 6.4 vs. 4.1 vs. 8.6*	PTCA = BMS in diabetics
Ix 2005[56]	CRF: 151 BMS vs. 139 CABG	-	Death+AMI+stroke HR=0.93	New revascularisation: HR=0.28* in favour of CABG	-

CAD – coronary artery disease. Other abbreviations as in Table 1.
All outcomes as percentages (%). *p<0.05.

mortality did not differ significantly, a registry of coronary revascularisation procedures in dialysis patients in the USA from 1978 to 1995 (US Renal Data System, USRDS) showed that CABG offered a survival benefit compared with PTCA, with a relative risk for total cardiovascular mortality of 0.9, despite the lower in-hospital mortality of PTCA (5.4% versus 12.5% for CABG).[52] Similarly, in another series of patients from the New York area who suffered from renal disease of varying severity, there was a high relative risk of death in the patients who underwent angioplasty compared to those who underwent CABG (relative risk 0.39 in favour of CABG for total mortality). However, this difference mainly concerned patients with ESRD and not those with impaired renal function (creatinine >2.5 mg/dL), in which subgroup the outcomes were equivalent.[53] Overall, PTCA without stenting was proved to be inferior to CABG in all the relevant studies, in terms of both new cardiovascular events and survival, with the exception of the subgroup with milder renal disease.

The first comparison of CABG with PTCA followed by stent implantation was in the continuation of the above-mentioned USRDS registry during the period 1995-1998. More PTCA procedures (4836 conventional, 4280 with BMS) than CABG (6668) were recorded, and CABG continued to be superior to PTCA, but this superiority arose mainly from the comparison with conventional angioplasty procedures, to which PTCA with stenting also proved superior.[54] The second attempt at a direct comparison between CABG and PTCA with BMS was made during the ARTS trial,[55] the only prospective, randomised study in this area.[56] During 1997-1998, 200 patients with impaired renal function, expressed as a glomerular filtration rate <60 ml/min/m^2, were randomised to CABG or to PTCA with stenting (BMS at that time) and were followed for three years. No patient had ESRD, while a prerequisite in every case was agreement between a cardiac surgeon and a cardiologist that the two methods were equivalent for the same degree of coronary revascularisation. No difference was seen in the primary endpoints (death, myocardial infarction, stroke) between the two groups. However, CABG was associated with a lower need for new revascularisation (relative risk 0.28). Compared to the patients with normal renal function who were enrolled in ARTS (1205 patients in total), those with impaired renal function showed around double the risk for adverse clinical events.

Before completing this review with a comparison between DES and CABG, it is worth making special mention of patients who have undergone kidney transplantation. Coronary artery disease in those patients is a major cause of mortality, with more frequent occurrence of three-vessel disease compared to the general population[57] and with one third of cardiac deaths being attributed to acute myocardial infarction.[58] Coronary angiography has been proved to be a safe diagnostic procedure as regards graft function and the probability of rejection,[57,59] while in one study[57] serum creatinine was found not to deteriorate following the coronary angiographic examination. As regards PTCA in patients with a transplanted kidney, it appears that the long-term outcome is clearly worse in comparison with controls (four-year relative risk 9.9 for infarction and cardiac death), while it does not differ significantly from patients under haemodialysis.[60] CABG in transplant patients is generally safe for the kidney graft,[59] while two-year survival has been found to be comparable with that of angioplasty: CABG with internal mammary artery 82.7%; CABG without internal mammary 74.4%; conventional PTCA 81.6%; PTCA with BMS 82.5% (significant difference only between CABG with internal mammary and conventional PTCA).[61] Comparisons between CABG and PTCA with DES in this subcategory of patients with a renal graft are awaited.

Prospective, randomised studies comparing angioplasty with DES and CABG, or even retrospective studies, are not yet available and research efforts should be aimed in that direction. Only after such studies have been completed will we be able to say with certainty whether CABG will continue to offer lower rates of cardiovascular events (including new revascularisation) compared with PTCA. As regards survival, surgical intervention is not superior to PTCA with stenting, even with BMS. Some researchers already suggest that DES should be the method of choice for symptomatic single-vessel coronary disease and should be preferred in focal or multifocal disease, leaving CABG only for unprotected lesions in the coronary trunk or for lesions that are mechanically not correctible by angioplasty.[62] Finally, however, despite the promise of DES and off-pump CABG, it is doubtful whether the outcomes in patients with renal disease will ever be equivalent to those in patients with satisfactory renal function. All our therapeutic efforts should be more aggressive, with optimal use of available medication and coronary angiography early

during hospitalisation for ACS, regardless of whether PTCA or CABG will follow.

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