Coronary Artery Disease

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Coronary heart disease is the most common cause of death in the general population and in patients with ESRD. The principles of cardiovascular risk assessment and management apply to both populations. Advances in noninvasive coronary artery imaging have improved early detection of subclinical disease. The goals of medical management of coronary disease are to modify the natural history of disease and to improve the symptoms of angina. Coronary revascularization poses a different risk and benefit equation in the ESRD population. In stable ESRD with multivessel coronary artery disease, coronary bypass surgery, despite the upfront risks of stroke, myocardial infarction, and chest wound infection, seems to be a favored approach. In patients with ESRD and acute coronary syndromes, percutaneous coronary intervention on the target vessel has been associated with the most favorable outcomes. This article explores the clinical issues with respect to coronary artery disease in patients with ESRD.

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Coronary heart disease is the leading cause of death in the US general population. With the obesity pandemic and the expected worsening of cardiovascular risk factors in the general population, the incidence and the prevalence of heart disease is expected to rise. Coronary artery disease (CAD) is the leading cause of death in patients with chronic kidney disease (CKD): Of the more than 320,000 patients with ESRD that requires dialysis or kidney transplantation in the United States, half will die from cardiovascular causes, and patients with milder degrees of CKD are more likely to die of CAD than to develop kidney failure that requires renal replacement therapy (1).

The observational studies concerning CAD and ESRD have revealed the following: (*1*) By the time patients reach dialysis, approximately 70% have significant coronary artery calcification indicative of coronary atherosclerosis, (*2*) patients with CAD and ESRD have markedly increased mortality over the general population, (*3*) treatment with disease-modifying therapy that is proved to reduce rates of (MI) or death (*e.g.*, aspirin, β adrenergic receptor blockers [BB], angiotensin-converting enzyme inhibitors [ACEI], 3-methylglutaryl CoA reductase inhibitors or statins) are used less frequently in patients with ESRD than in the general population, (*4*) noninvasive imaging seems to have less precision and accuracy in patients with ESRD, and (*5*) patients who have ESRD and are selected for revascularization with coronary artery bypass graft (CABG) or percutaneous coronary intervention (PCI) have improved survival compared with those who are treated with medical therapy alone (2–6). Table 1 presents a summary of CAD risk reduction, diagnosis, and management principles. Recommendations in Table 1 present a task list for optimal (not minimal) clinical goals and go beyond many current professional society guidelines concerning the individual treatment targets.

ESRD: More Than a Coronary Heart Risk Equivalent

Patients with ESRD have more than a 10-fold increased risk for coronary heart disease (CHD) death per 1000 person-years than a patient with five Framingham risk factors projected over time (7). This is partly due to the observation that patients with ESRD have a cluster of CHD risk factors that most commonly include advanced diabetes; hypertension; low HDL cholesterol; hypertriglyceridemia; and, less commonly, obesity, smoking, and family history of CHD (8). It is also partly due to the unique changes that occur in ESRD and accelerate atherosclerosis, destabilize the atherosclerotic plaque, cause myocardial fibrosis, and create valvular heart disease (9). It is beyond the scope of this article to speculate on the wide range of basic mechanisms (*e.g.*, inflammation, oxidative stress, disordered calcium–phosphorous–parathyroid hormone balance) that have been implicated in this potentially unique and serious form of CHD that occurs in ESRD (9). It should be recognized that, as a result, most patients with ESRD have significant CAD and structural heart disease (left ventricular hypertrophy and cardiac fibrosis) and therefore are at increased risk for sudden death that is triggered by myocardial ischemia, electrolyte shifts, sepsis, and other events (10). In addition, most patients with ESRD have extensive coronary, aortic, and valvular calcification (aortic and mitral), which may influence interventional and medical management (11). Finally, biomarkers of cardiac injury (troponin and creatine kinase myocardial band) are frequently elevated in patients with ESRD in the absence of cardiac symptoms or signs of ischemia (12). Studies of baseline troponin in ESRD suggest that elevations are associated with more extensive coronary

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Table 1. Selected strategies for CAD risk stratification and management in patients with ESRD[a]

Clinical Strategy	Rationale
Weight loss/weight maintenance to body mass index ≤25 kg/m^2 in the obese with adequate nutrition	Improvement of the dysmetabolic syndrome and diabetes
Low sodium intake	Reduce BP; make BP more responsive to medications; reduce volume retention between dialysis sessions
Aspirin 81 mg/d orally or clopidogrel 75 mg/d orally if aspirin intolerant	Prevention of MI and stroke
Lipid control (diet, statin, fibrates, niacin, others): LDL cholesterol < 100 mg/dl (consider <70 mg/dl in established CAD); TG <150 mg/dl; HDL cholesterol >50 mg/dl	Prevention of MI, stroke, and possibly CVD death
BP control to target of SBP <130 mmHg	Prevention of MI, stroke, heart failure, and CVD death
Renin-angiotensin system blocking agents	Preserve residual urine volume in peritoneal dialysis patients
β blocking agents	Reduce left ventricular hypertrophy
other add-on agents	Treatment of subclinical cardiac ischemia
Blood glucose control in diabetes	Reduction in risk of MI, stroke, and CVD death
glycohemoglobin reduced to <6.0% (nondiabetic range) is optimal	Reduction in worsened nephropathy/retinopathy/neuropathy
Phosphorus, PTH, and calcium optimization	Reduction in all cause death
order of priority is (*1*) PO_4 (3.5 to 5.5 mg/dl, (*2*) iPTH (150 to 300 pmol/L), and (*3*) Ca (8.4 to 9.5 mg/dl)	Possible reduction in coronary, aortic, and valvular calcification; avoid calciphylaxis; reduce metabolic bone disease
Dobutamine stress echocardiography/comprehensive cardiac computed tomographic angiography	Diagnosis of CAD in very high risk or symptomatic patients + evaluation of left ventricular function and cardiac valves
Treatment of acute coronary syndromes	Reduce rates of MI and death
aspirin	
clopidogrel	
antithrombotics (heparin, low molecular weight heparin, bivalirudin, fondaparinux)	
β blockers	
RAS-blocking drugs	
optimal percutaneous revascularization	
Revascularization (PCI or CABG)	Reduce mortality in stable patients with multivessel CAD with or without left ventricular dysfunction

[a]CABG, coronary artery bypass graft; CAD, coronary artery disease; CVD, cardiovascular disease; iPTH, intact parathyroid hormone; MI, myocardial infarction; PCI, percutaneous coronary intervention; PTH, parathyroid hormone; RAS, renin-angiotensin system; SBP, systolic BP; TG, triglycerides.

disease and worsened long-term survival (13). The immediate interpretation of troponin levels in patients with ESRD is problematic in a patient with symptoms of cardiac ischemia, and a characteristic rise and fall of troponin with another supportive piece of clinical data (chest pain, ischemic electrocardiogram changes, or a culprit lesion found on angiography) are needed for the diagnosis of acute MI (12).

Given the near uniform presence of CAD and heightened risk for cardiac events, recommendations include aspirin 81 to 325 mg/d orally, use of BB as part of the antihypertensive regimen, and lipid modification according to the published guidelines in patients with CKD and ESRD (14–16). Observational studies of patients who present with acute coronary syndromes indicate reduced rates of death over 5 yr in patients who received ASA, BB, and ACEI (17,18). They are less frequently used in patients with ESRD because of increase rates of bleeding with aspirin, bradycardia and conduction system disease with BB, and hyperkalemia with ACEI. Recent data from the general population at very high cardiac risk, particularly those after acute coronary syndromes, suggest an optimal LDL cholesterol may be <70 mg/dl (19). These recommendations, however, do not specifically mention patients with ESRD, who are often excluded from trials of acute or chronic cardiovascular disease. This being considered, the algorithm published by the Kidney Disease Outcomes Quality Initiative (K/DOQI) concerning the management of dyslipidemia in CKD is shown in Figure 1.

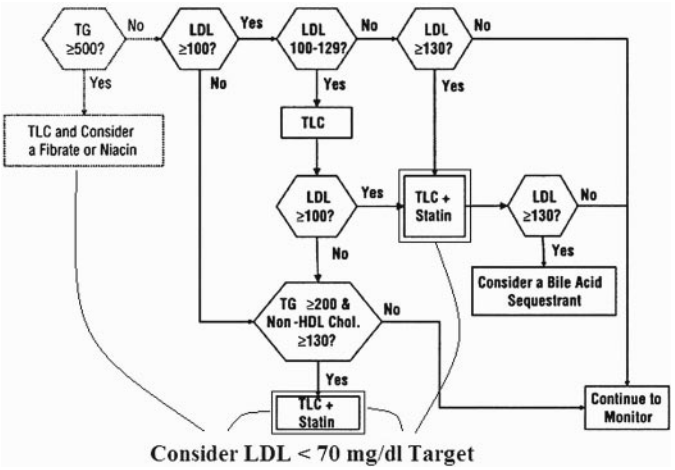

Figure 1. Suggested treatment algorithm for the management of dyslipidemia modified from Kidney Disease Outcomes Quality Initiative (K/DOQI). TG, triglycerides; TLC, therapeutic lifestyle changes.

3-Hydroxy statins are featured as the primary agents after therapeutic lifestyle change to achieve an LDL cholesterol <100 mg/dl or a non–HDL cholesterol <130 mg/dl. Bile-acid sequestrants are considered a second-line agent to be added to a statin when these targets are not achieved. Niacin and fibrates are considered as primary treatment for persistently elevated triglycerides >500 mg/dl. However, it should be kept in mind that elevated triglycerides in both the general and the CKD population is largely due to excess adiposity and intake of sugars, starches, and saturated fats.

There are two common therapies that reduce LDL cholesterol in patients with ESRD: The noncalcium phosphate binder sevelamer and statins (20). Sevelamer in addition to binding phosphate in the gastrointestinal tract also works as a bile-acid sequestrant and results in a predictable reduction in LDL cholesterol (20). Sevelamer was tested in the Dialysis Clinical Outcomes Revisited (DCOR) trial, the largest outcomes study ever conducted in the hemodialysis population. The 3-yr trial, involving >2100 patients, compared the difference in mortality and morbidity outcomes for patients who received sevelamer hydrochloride *versus* those who used using calcium-based phosphate binders (21). Despite the expected (but not measured) LDL cholesterol reduction with sevelamer, there was no reduction in mortality between the treatment groups (9% relative risk reduction with sevelamer; $P = 0.30$) (21). The latest results with statins have come from the 4D Trial (Deutsche Diabetes Dialyze Studie), in which 1255 patients with type 2 diabetes and new ESRD were randomly assigned to atorvastatin 20 mg/d orally or placebo for a median of 4 yr (22). The statin was effective in reducing the median serum LDL cholesterol by 42% throughout the study period. However, the primary end point—defined as the composite of cardiac death, nonfatal MI, and fatal or nonfatal stroke—was reduced by only 8% with atorvastatin ($P = 0.37$). The 4D investigators concluded that the negative results might have been due to the advanced cardiovascular diseases in the patients who were on long-term HD and because statin therapy was initiated too late. It seems from DCOR and 4D that LDL cholesterol reduction in ESRD may not have an impact on cardiovascular events or mortality because of the advanced disease that is present by the time ESRD develops, competing cardiovascular mechanisms for terminal events in ESRD (*e.g.*, nonischemic arrhythmias, bradycardia), and the high rates competing noncardiovascular sources of mortality (*e.g.*, sepsis, venous thromboembolism).

Diagnosis of CAD in ESRD

The most definitive and increasingly available test to diagnose the presence of CAD in ESRD is cardiac computed tomography (CT). In approximately 70% of patients who start dialysis, coronary artery calcification (expressed as Agatston score, equivalent mass, and volume [mm^3]) is present on cardiac CT (20). A recent study in the general population using intravascular ultrasound with virtual histology confirmed with atherectomy and necropsy of coronary atheroma specimens that coronary artery calcium is found in the necrotic core of atherosclerosis (23). Therefore, it is reasonable to conclude that coronary artery calcium represents anatomic atherosclerosis. Given its high prevalence and uncertain relationship to management, cardiac CT is not recommended for patients with ESRD as a screening test. Cardiac CT angiography (CTA) is an advanced technique that uses approximately 60 to 80 intravenous iodinated contrast and multidetection CT scanners (64, 128, and 256 slice), which can determine the degree of stenosis similar to coronary angiography. Heavy coronary calcium can cause a "bloom" artifact that may make this technique more difficult to use in ESRD. In the United States, Medicare recently approved cardiac CTA as a reimbursable diagnostic test for patients in the general population who present with chest pain or those with indeterminate results from conventional stress testing. There are no published studies at the time of this writing using CTA in ESRD.

Few studies have examined exercise electrocardiography and its association with outcomes in ESRD. A report of 95 patients with ESRD found that exercise electrocardiogram testing was problematic in that 44% did not achieve the target heart rate (85% of maximum predicted heart rate) and no CAD that required intervention was identified by exercise testing (24). Furthermore, the symptom of angina pectoris on stress testing has a sensitivity and a specificity of 65 and 66%, respectively. Therefore, stress imaging with an assessment of left ventricular function is advised when evaluating patients with ESRD for CAD. In a review of CAD diagnostic testing in ESRD, Hedayati and Szczech (25) reported that most studies of stress testing were in patients who had ESRD and were undergoing renal transplant evaluation. In general, the diagnostic accuracy for significant CAD with pharmacologic stress testing with nuclear scintigraphy or echocardiography is approximately 80% at best. Dobutamine-echocardiography is considered to be of particular value in ESRD because it evaluates for significant CAD and can give left ventricular ejection fraction, valvular structure and function, and inferences on diastolic function, which cannot be obtained from nuclear scintigraphy. In all cases of ESRD, a low index of suspicion is required for underlying CAD, and in

selected patients, consideration for coronary angiography and possible revascularization is warranted (26). As a practical point, for patients who have ESRD and are undergoing contrast procedures (CTA, catheterization and angiography, and PCI), iso-osmolar iodixanol contrast media recommended by K/DOQI and dialysis, which removes the contrast, is advised shortly after the procedure or the next day to limit adverse effects, including delayed contrast reactions (nausea, vomiting, rash) (27). Furthermore, the risk for contrast-induced nephropathy and cessation of residual renal function in peritoneal dialysis patients should be carefully considered before contrast procedures.

Coronary Revascularization in ESRD

The available data regarding CABG and PCI in patients with CKD consist of observational studies that are limited by small populations; difficulty in controlling for confounders, including comorbidities and degree of coronary artery disease; and, most important, selection bias in patients who are referred for invasive procedures (28). No randomized, controlled trials have specifically evaluated the indications for coronary revascularization in patients with CKD, and the treatment of these patients is extrapolated from studies of patients with CAD and normal renal function. Coronary revascularization for significant left main CAD and three-vessel CAD with depressed left ventricular systolic function, primary PCI for ST segment elevation MI, and early revascularization for non–ST segment elevation MI all have been shown to confer survival benefit to patients with normal renal function; therefore, these indications, as well as medically refractory angina, are considered indications for coronary revascularization in patients with ESRD (29,30). Such extrapolations are complicated by the increased risk and decreased durability of revascularization procedures in patients with ESRD compared with patients with normal renal function.

The Northern New England Cardiovascular Disease Study analyzed clinical outcomes of 279 dialysis patients and 15,271 patients who had normal renal function and were undergoing CABG (31,32). The patients with ESRD were older and had significantly more comorbid conditions, including diabetes, peripheral vascular disease, and chronic obstructive pulmonary disease. Patients with ESRD were 4.4 times more likely to experience in-hospital mortality than were the other CABG patients (odds ratio 4.4; 95% confidence interval 3.0 to 6.4; $P < 0.001$), a three-fold higher risk for mediastinitis, and a two-fold higher risk for stroke compared with patients who were undergoing CABG and were not on dialysis. Another study from the same database reported 5-yr survival after CABG as 83.5% for patients with normal renal function and 55.8% for all patients with ESRD (32). Data on coronary revascularization from the United States Renal Data System database have been reported in 14,306 dialysis patients who underwent their first coronary revascularization procedure after initiation of dialysis (33). The in-hospital death rate was more than doubled in patients who underwent CABG compared with those who underwent balloon angioplasty (12.5 *versus* 5.4%). Despite the greater in-hospital mortality, CABG was associated with improved long-term clinical outcomes. Because this analysis did not assess the effect of stents, which have been shown to improve procedural outcomes in patients with ESRD, a subsequent study that analyzed the effect of routine stenting was reported from the same database: The patient population consisted of 4836 dialysis patients who were treated with balloon angioplasty, 4280 who were treated with stenting, and 6668 who were treated with CABG (34). Patients who underwent balloon angioplasty alone required repeat revascularization procedures more frequently than patients who underwent coronary artery stenting: Stents decrease restenosis rates in patients who underwent PCI. Patients who underwent CABG, however, had the lowest rate of repeat revascularization procedures (Figure 2). In-hospital mortality rate was highest for those who underwent CABG (8.6%) when compared with those who underwent balloon angioplasty (6.4%) and stenting (4.1%). Long-term survival was significantly better in dialysis patients who underwent CABG (56.4 ± 1.4%) compared with those who were treated with either balloon angioplasty (48.2 ± 1.5%) or stenting (48.4 ± 2.0%; $P < 0.0001$).

Keeley *et al.* (35) demonstrated that in patients who had ESRD and presented with an acute coronary syndromes, patients who were selected for PCI had an improved survival over those who were selected for CABG or medical therapy alone (Figure 3). Importantly, this study was published before the use of drug-eluting coronary stents (DES). It is now understood that the use of drug-eluting coronary stents results in lower restenosis rates in patients with ESRD with the consequence of requiring a prolonged period of dual platelet inhibition with aspirin and clopidogrel after implantation (36). Although there are no studies of the optimal duration of therapy for stented patients with ESRD, it is reasonable to plan on at least 1 yr of aspirin and clopidogrel and, if tolerated from a bleeding perspective, consideration for lifelong dual therapy. Otherwise, a change to aspirin alone can be considered.

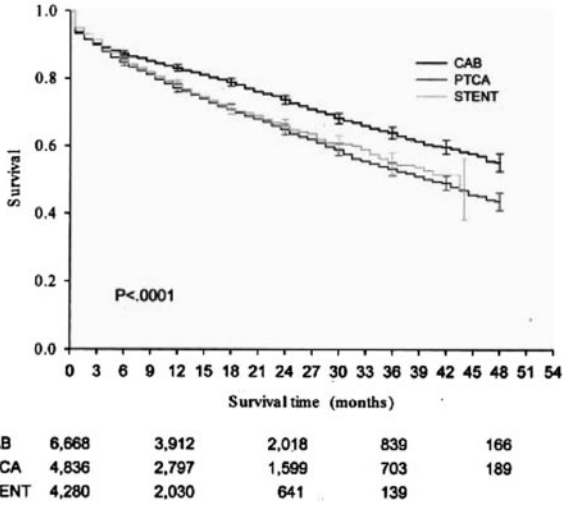

Figure 2. Survival after coronary revascularization from the US Renal Data System. Reprinted from Herzog *et al.* (34) with permission.

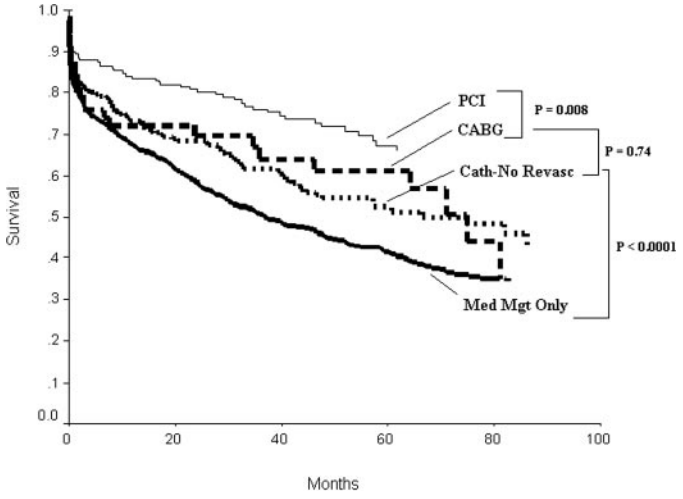

Figure 3. Long-term survival in patients with severe chronic kidney disease (estimated GFR <60 ml/min per 1.73 m^2 including $n = 199$ ESRD) by revascularization (Revasc) or management strategy used, adjusted for the propensity for revascularization, type of acute coronary syndrome, medical therapy received, and other significant baseline variables. CABG, coronary artery bypass graft; Cath, catheterization; Med Mgt, medical management; PCI, percutaneous coronary intervention. Reprinted from Keeley *et al.* (35) with permission.

Conclusions

The majority of patients with ESRD have anatomic CAD, and the major clinical goals are to reduce the future risk for MI and death. Standard CAD risk reduction and management principles in the general population apply to patients with ESRD. When suitable, CABG despite its upfront risks is associated with improved survival in patients with ESRD and multivessel disease. Conversely, in patients with acute coronary syndromes, a targeted approach with PCI is reasonable. There is a need for large trials of CAD risk reduction and management in patients with ESRD given the unique balance of risk and benefit and overall high event rates in this population.

Disclosures

None.

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