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Coronary Angiography in Patients with Chronic Kidney Disease

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1. Introduction

Cardiovascular disease, mostly coronary artery disease (CAD), remains an important cause of morbidity and mortality in patients with any degree of renal dysfunction. Presently, according to the United States Renal Data System, the size of the population with chronic kidney disease (CKD) stage V reached a new high in 2008, with almost 550,000 patients under treatment (U.S. Renal Data System, 2010). Sadly, it is expected that half of them will die from cardiovascular disease; moreover, it is noteworthy that even patients with less advanced stages of CKD are at greater risk of death from CAD than of reaching the final stages of renal failure that would eventually require renal replacement therapy (Sarnak et al., 2003). In these patients, about 50% to 60% of all cardiovascular deaths are due to acute myocardial infarction, sudden death, or ischemic heart disease (Herzog et al., 2008).

The interplay between CKD and CAD is a very complex one and can only be partly explained by the fact that patients with CKD share many of the so-called traditional risk factors also linked to CAD, such as long-standing diabetes, hypertension, low levels of HDL-cholesterol, and hypertriglyceridemia (McCullough, 2002a). Additionally, a pro-inflammatory state and high oxidative stress levels are usually seen in patients with advanced CKD, which may contribute to accelerated atherosclerosis, plaque instability, acute coronary syndromes, and myocardial fibrosis (Yerkey et al., 2004). The result of this clustering of traditional and nontraditional risk factors may explain the epidemiological observation that patients with CKD stage V have more than a 10-fold increased risk of death from CAD than a patient with 5 Framingham risk scores (McCullough, 2002b).

The prevalence of CAD in CKD patients undergoing dialysis varies with such factors as age, diabetic status, time of follow-up, and the diligence with which CAD is investigated. In a group of 4,024 new dialysis patients, including 44% with diabetes, 32% had a history of CAD (Foley, 2003), which is conceivably more than 3 times the figure corresponding to CAD in the general population (Levey & Eknoyan, 1999). The prevalence of angiographically confirmed CAD, on the other hand, defined as luminal reduction >50%, ranges from 24% in low-risk patients to 85% in older, diabetic patients (Goldsmith & Covic, 2001). Nevertheless, most of these data originated from studies in which coronary angiography was performed only in patients with clinical evidence and/or noninvasive studies suggestive of CAD,

leaving out patients in whom asymptomatic, undiagnosed, significant CAD might be already present; therefore, the actual prevalence of CAD might even be higher.

Adding to the importance of CAD in patients with CKD, observational data also show that CAD remains a significant clinical problem even if a patient undergoes successful kidney transplantation (Aakhus et al., 2004). The annual risk of CVD death is 3.5% to 5% in renal transplant recipients, which is 50-fold higher than that in the general population (Ojo, 2006). Acute coronary syndrome is highly prevalent in the early posttransplant period, and on average, CVD mortality accounts for 30% and 75% of early and late posttransplant deaths, respectively.

Therefore, coronary angiography is a valuable tool, very often used in patients with CKD both in the acute as well as in the chronic setting of CAD, for the purpose of either the diagnosis of, or the treatment of CAD by means of percutaneous coronary intervention. The major challenges with coronary angiography relate to when is it appropriate to perform it as part of the cardiovascular assessment in patients with CKD and what are the risks associated with the procedure. The ultimate management of CAD, should it be found after the diagnostic procedure, is beyond the scope of this chapter.

2. Screening for coronary artery disease in patients with CKD

Based on the above considerations, screening for the presence and severity of CAD in patients with CKD, and especially in those being considered for renal transplantation, is a fundamental step during routine cardiovascular risk assessment; in the setting of kidney transplantation, for instance, numerous studies indicate that not only are the majority of serious cardiovascular events in renal transplant recipients related to CAD but also these events tend to occur during the first few months following transplant (Kahwaji et al., 2011). The last observation strongly suggests that relevant CAD could have been missed before patients were actually included on renal transplant waiting lists, although they had been screened for it during routine pretransplant workups according to current guidelines. According to the American Society of Transplantation recommendations (Kasiske et al., 2001), patients with diabetes, a prior history of ischemic heart disease or an abnormal ECG, or age ≥ 50 years should be considered at high risk for CAD and referred for a cardiac stress test, and only in those with a positive stress test, for coronary angiography.

This strategy, however, is under dispute for the now recognized reduced sensitivity and specificity of noninvasive testing in renal patients compared with that in the general population (Marwick et al., 1990; Schmidt et al., 2001; Welsh et al., 2011), a finding that prompted many investigators to recommend direct diagnostic coronary angiography in high-risk patients. In agreement with the last view, in a previous study, our group showed that significant CAD, as defined by coronary angiography, was the best predictor of cardiovascular events compared with radionuclide myocardial perfusion study or dobutamine-atropine stress echocardiography in renal transplant candidates (De Lima et al., 2003), even taking into account that coronary angiography is an expensive, invasive procedure, not free from complications. However, in the same investigation, we also found that the prevalence of significant CAD was less than 50%, suggesting a need for better screening mechanisms that could identify high-risk individuals in whom coronary angiography could be avoided without incurring an increased risk of future events.

2.1 The challenge of referring patients with CKD for coronary angiography

Tables 1 and 2 show the most recent guidelines for coronary angiography to establish the *diagnosis* in patients with suspected angina and for *risk stratification* in patients with chronic stable angina, respectively (Fraker Jr et al., 2007).

Invasive coronary angiography has received a Class I indication for patients with known or possible angina pectoris who have survived sudden cardiac death. For risk stratification in patients with chronic stable angina, coronary angiography should be performed in: a) patients with disabling (Canadian Cardiovascular Society classes III and IV) stable angina despite medical therapy; b) patients with high-risk criteria on noninvasive testing; c) patients with angina and symptoms and signs of heart failure; d) patients with clinical characteristics indicating a high likelihood of severe CAD.

Coronary angiography is indicated to establish the diagnosis of CAD in patients with suspected angina in those with...	Level of Evidence	Comment for Patients with CKD
Known or possible angina pectoris who have survived sudden cardiac death	B	Survival rates much lower than rates for the general population

Table 1. Class I indication of the AHA/ACC Guidelines for coronary angiography to establish a *diagnosis* in patients with suspected angina in the general population. Modified from Fraker Jr, et al. (2007).

Coronary angiography is indicated for risk stratification in patients with chronic CAD in those with...	Level of Evidence	Comment for Patients with CKD
Disabling chronic stable angina despite medical therapy	B	Uncommon clinical presentation for patients with CKD
High-risk criteria on noninvasive testing regardless of angina severity	B	Lower sensitivity than that in the general population
Angina who have survived sudden cardiac death or serious ventricular arrhythmia	B	Survival rates much lower than those in the general population
Angina and symptoms and signs of congestive heart failure	C	May be confused with hypertensive cardiomyopathy, fluid overload, subdialysis
Clinical characteristics that indicate a high likelihood of severe CAD	C	Lower specificity than that in the general population

Table 2. Class I indications of the AHA/ACC Guidelines for coronary angiography for *risk stratification* in patients with chronic stable angina. Modified from Fraker Jr, et al. (2007).

Although these guidelines have been extensively tested and validated in the general population, for patients with CKD the data supporting that approach is less robust. The main pitfalls of applying the same criteria recommended for patients with normal renal function to patients with CKD are briefly described below.

2.1.1 Clinical presentation

Although angina pectoris is usually a reliable indicator of the presence of CAD in the general population, it is not so among patients with CKD, particularly in the more advanced stages of renal failure. For instance, chest pain is commonly observed in the dialysis population; nevertheless, its characteristics make the discomfort often described as "atypical," and the pain does not correlate with the presence of significant CAD. In fact, fewer than 50% of dialysis patients with acute myocardial infarction present with chest pain, which likely explains why myocardial infarction is infrequently suspected on admission (Herzog et al., 2007). Reasons for the different presentations of ischemia are not well understood but may be related to associated diabetic or uremic neuropathy. Diagnosis of ischemia is further complicated by the fact that other symptoms of ischemia, such as dyspnea on exertion, fatigue, and hypotension, are common in dialysis patients and may be attributed to dialysis-related factors, such as anemia, volume overload, acidosis, non-compliance with fluid intake or dialysis or to ultra-filtration during dialysis (Sosnov et al., 2006). Furthermore, silent ischemia occurs frequently in patients with CKD.

We previously found that, in a group of high-risk renal transplant candidates who underwent coronary angiography *per* protocol, regardless of symptoms, the prevalence of significant CAD was 40% in patients with, and 58% in patients without, angina (De Lima et al., 2003). Disabling symptoms, even in the presence of significant and extensive CAD, are quite uncommon in patients with CKD.

2.1.2 Noninvasive testing

2.1.2.1 Resting and exercise electrocardiogram

The interpretation of resting and exercise electrocardiograms for the diagnosis of CAD in patients on dialysis is often hampered by the presence of concurrent left ventricular hypertrophy, anemia, and electrolyte disturbances that may cause electrocardiographic changes resembling those of ischemia (Sharma et al., 2007). Also, most patients with CKD have poor physical stamina, limiting the utility of exercise electrocardiography as a diagnostic tool. The overall specificity and sensitivity of both tests for the diagnosis of CAD are lower (<50%) in patients with CKD than in the general population.

2.1.2.2 Myocardial perfusion assessment

Dipyridamole radionuclide stress testing with single-photon emission tomography (SPECT) has been widely used as a screening test for CAD in patients with a variety of clinical conditions. The reported specificity and sensitivity of the test are close to 70% and 80%, respectively. Among patients with CKD, however, the results are highly variable with sensitivity often <60% compared to that of coronary angiography (Holley et al., 1991; Sharma et al., 2007). The reasons for the unexpected low sensitivity and great variability of cardiac scintigraphy in patients with CKD are unclear but may reflect the increased levels of circulating adenosine, reduced coronary flow reserve, or left ventricular hypertrophy. Combined dipyridamole-exercise thallium imaging reportedly increases the sensitivity and specificity of the test.

Dobutamine-atropine stress echocardiography has been associated with better and more consistent results for detecting significant (>70%) coronary stenosis in patients with CKD,

with a sensitivity and negative predictive value between 70% and 80% (Herzog et al., 1999; Ferreira et al., 2007). However, the utility of this test is limited, because dobutamine often increases blood pressure, causing interruption of the test in about 15% of patients.

Until a few years ago, magnetic resonance imaging (MRI) had been used for assessment of myocardial function and detection of myocardial ischemia and scar tissue in patients with CKD (Andrade et al., 2009). Myocardial stress perfusion studies performed with MRI adequately detected CAD, and there was a good correlation between these results and those from coronary angiography and radionuclide studies (Andrade et al., 2004). Unfortunately, further development of this imaging modality was halted, because of the description of a serious complication related to the use of gadolinium-based contrast media in patients with CKD, called nephrogenic systemic fibrosis (Grobner, 2006). Thus, starting in 2007, the United States Food and Drug Administration issued a boxed warning about this progressive, debilitating and, occasionally fatal condition recommending that the use of gadolinium-based contrast agents should be avoided in patients with acute or chronic renal insufficiency with a glomerular filtration rate $< 30\text{mL}/\text{min}/1.73\text{m}^2$.

2.1.2.3 Coronary angiotomography

Multidetector-row computed tomography (MDCT) has been recently applied to the detection of CAD in patients with ESRD (Rosário et al., 2010). This method allows not only the detection of coronary calcifications but also the detailed visualization of the arterial wall and lumen, and therefore, may become an alternative to coronary angiography. Experience with this method in patients with CKD is still very limited.

2.1.2.4 Clinical implications

Thus, referring patients with CKD for coronary angiography is not an easy task (De Lima et al., 2010; Lentine et al., 2010). One could be very “conservative,” only referring patients with overt, unequivocal symptoms of angina or extensive myocardial ischemia on noninvasive testing to undergo coronary angiography.

This approach would most likely yield a prohibitively large number of “false negatives,” meaning that many patients with significant CAD will be deemed free of disease. It is quite easy to see the clinical implications of such an approach, especially for renal transplant candidates. Alternatively, one could be very “aggressive” and refer all patients with CKD stages IV or V for coronary angiography. We would be able to diagnose every single patient with CAD in adopting this strategy. The downside is that coronary angiography is a somewhat risky, costly procedure and as many as 50% to 60% of all patients will have no significant CAD at all and, therefore, will not be further referred for coronary interventions (percutaneous or surgical). Finally, patients still not on renal replacement therapy might not only not benefit from this strategy but might also have worsening of their residual renal function because of the contrast-induced nephropathy.

As a matter of fact, it is still unclear whether the benefits outweigh the harms of routine screening and prophylactic revascularization to prevent coronary artery disease (CAD) in asymptomatic kidney transplant candidates. To help clarify that issue, an ongoing randomized clinical trial is being performed to study the effect of CAD screening on major adverse cardiac events (Kasiske et al., 2011).

3. In whom should coronary angiography be performed?

The same indications presented in Tables 1 and 2 should be applied to patients with CKD. The greatest challenge facing the clinical cardiologist called upon to assess a patient with

CKD regarding the presence and severity of CAD is, based on the previous considerations, which additional patients should be referred for coronary angiography, especially those asymptomatic patients being assessed before renal transplantation.

In a previous study, we sought to determine the clinical predictors more closely related to CAD in 301 renal transplant candidates treated by hemodialysis (Gowdak et al, 2007). CAD (> 70% stenosis) was found in 45% of patients, and the clinical variables significantly associated with CAD were diabetes, peripheral vascular disease, and previous myocardial infarction. More important, the prevalence of CAD increased with the number of clinical predictors from 26% (none) to 100% (all present), while the incidence of events increased 2-, 4-, and 6-fold in those with diabetes, peripheral vascular disease, or previous myocardial infarction, respectively ($p < .0001$). This approach would allow reducing the prevalence of unnecessary angiography from 55% (when all patients undergo angiography) to 26%. However, we believe that missing the diagnosis of CAD in one-fourth of patients is still not totally satisfactory.

Another approach should be to restrict the use of coronary angiography as a predictive tool of cardiovascular events in patients with CKD in which this strategy is powerful enough to discern between patients at high- and low-risk, comparatively to either clinical stratification alone or to noninvasive testing. We compared the efficacy of clinical stratification, noninvasive testing, and coronary angiography in predicting cardiovascular events in patients with CKD being considered as potential renal transplant candidates (De Lima et al., 2006; Gowdak et al., 2008). We were able to show that, for patients considered at low-risk for CAD (age < 50 years, with no diabetes, and no history of cardiovascular disease), additional noninvasive or invasive testing, added no capability in predicting cardiovascular events beyond that given by clinical stratification. On the other hand, for patients at high-risk for CAD, defined by the presence of any 2 risk factors combined (age \geq 50 years *or* diabetes *or* cardiovascular disease), noninvasive testing failed to identify patients at higher risk for cardiovascular events; indeed, the finding of significant CAD by angiography (luminal stenosis \geq 70%) was the strongest predictor of events. For patients at intermediate-risk for CAD, defined by the presence of any single risk factor alone, noninvasive testing was a good tool in identifying patients at higher risk of events, should an abnormal myocardial perfusion scan be found. Figure 1 shows the proposed algorithm for risk stratification of CAD in renal transplant candidates.

4. Contrast-induced Acute Kidney Injury

Very often, the highlighted importance of coronary angiography for the diagnosis and/or management of CAD in patients with CKD is offset by the possibility of contrast-induced acute kidney injury (AKI) that may follow the use of any iodinated contrast media. Contrast-induced AKI is a common and potentially serious complication after the administration of contrast media in patients at risk for acute renal injury (McCullough, 2008). Unfortunately, the most important risk factor for contrast-induced AKI is an already compromised baseline renal function. The knowledge that not only contrast-induced AKI adversely affects the short- and long-term prognosis but also implies greater health costs prompted cardiologists to be particularly careful whenever coronary angiography is recommended to patients with any degree of CKD.

Many definitions currently exist in practice regarding contrast-induced AKI; it can be simply defined as an increase in serum creatinine (SCr) occurring within the first 24h after contrast exposure and peaking up to 5 days afterwards. The rise in SCr can be expressed

either in absolute terms (0.5 to 1.0mg/dL) or as a proportional rise in SCr of 25% to 50% above the baseline value (Gleeson & Bulugahapitiya, 2004).

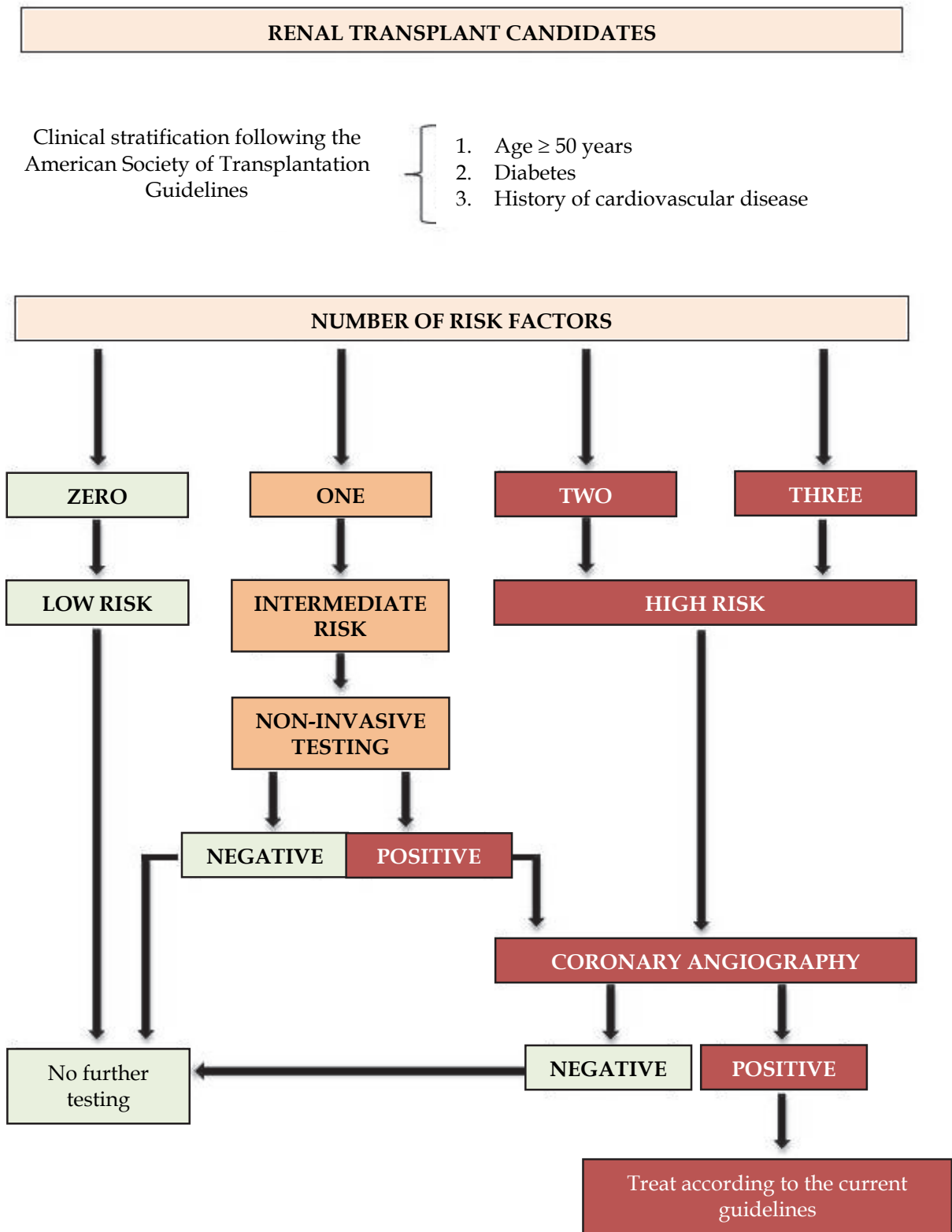


Fig. 1. Proposed algorithm for coronary angiography in patients with CKD awaiting renal transplantation.

The increase in SCr seen in patients with contrast-induced AKI is associated with a significant increase in the risk of death. In a large retrospective study comprising over 16,000 patients undergoing procedures, those who developed contrast-induced AKI had a 5.5-fold increased risk of death during hospitalization (Levy et al., 1996). Another study with more than 7,000 patients confirmed the short-term worse prognosis in patients with AKI but extended the greater risk of death up to 5 years after the procedure: the mortality rates at 1 year after development of contrast-induced AKI (12.1%) and at 5 years (44.6%) were higher compared with rates of 3.7% and 14.5%, respectively, in patients who did not develop contrast-induced AKI, indicating that the increased risk of death persisted in the long term (Rihal et al., 2002).

Besides the increased mortality, contrast-induced AKI also poses a high morbidity risk. Its occurrence has been linked to late cardiovascular events after percutaneous coronary intervention (such as myocardial infarction), bleeding requiring transfusion, and vascular complications. As a consequence of more serious adverse events, the in-hospital stay is longer with an estimated US\$10,000 to US\$12,000 added to the final bill during the first year (Subramanian et al., 2007).

4.1 Pathophysiology of contrast-induced Acute Kidney Injury

Centrally to the pathophysiology of contrast-induced AKI is the presence of a reduced nephron mass, a hallmark of chronic kidney disease. It has already been demonstrated that in patients with CKD, defined by an estimated glomerular filtration rate < 60mL/min/1.73m², there is a considerable loss of nephron units, making the residual renal function prone to further declines with renal insults (Figure 2). When serum creatinine or eGFR is unavailable, then a survey may be used to identify patients at higher risk than the general population for contrast-induced AKI. Iodinated contrast, after causing a brief (minutes) period of vasodilation, causes sustained (hours to days) intrarenal vasoconstriction and ischemic injury. The ischemic injury sets off a cascade of events largely driven by oxidative injury causing death of renal tubular cells. If a sufficient mass of nephron units is affected, then a recognizable rise in serum creatinine will occur. Any superimposed insult, such as sustained hypotension in the catheterization laboratory, micro-showers of atheroembolic material from catheter exchanges or the use of intraaortic balloon counterpulsation, or a bleeding complication can amplify the injury processes occurring in the kidney (McCullough et al., 2006a).

Renal function must be assessed before administration of contrast medium so appropriate caution may be exercised to avoid additional risks. The National Kidney Foundation Kidney Disease Outcome Quality Initiative recommends that clinicians use an eGFR calculated from the SCr as an index of renal function rather than using SCr in stable patients. In such a situation when an eGFR cannot be obtained prior to the procedure, clinicians should inquire about risk markers for AKI after iodinated contrast (Table 3).

The presence of multiple contrast-induced AKI risk factors in the same patient or high-risk clinical scenarios can create a very high risk (~50%) for contrast-induced AKI and (~15%) acute renal failure requiring dialysis after contrast exposure.

In the setting of emergency procedures, where the benefit of very early imaging outweighs the risk of waiting, the procedure can be performed without knowledge of serum creatinine or eGFR.

Risk markers for contrast-induced acute kidney injury	
previous renal disease	diabetes mellitus
prior renal surgery	hypertension
proteinuria	gout
use of nephrotoxic drugs, such as nonsteroidal anti-inflammatory agents	

Table 3. A brief 7-item survey to identify patients at higher risk for AKI.

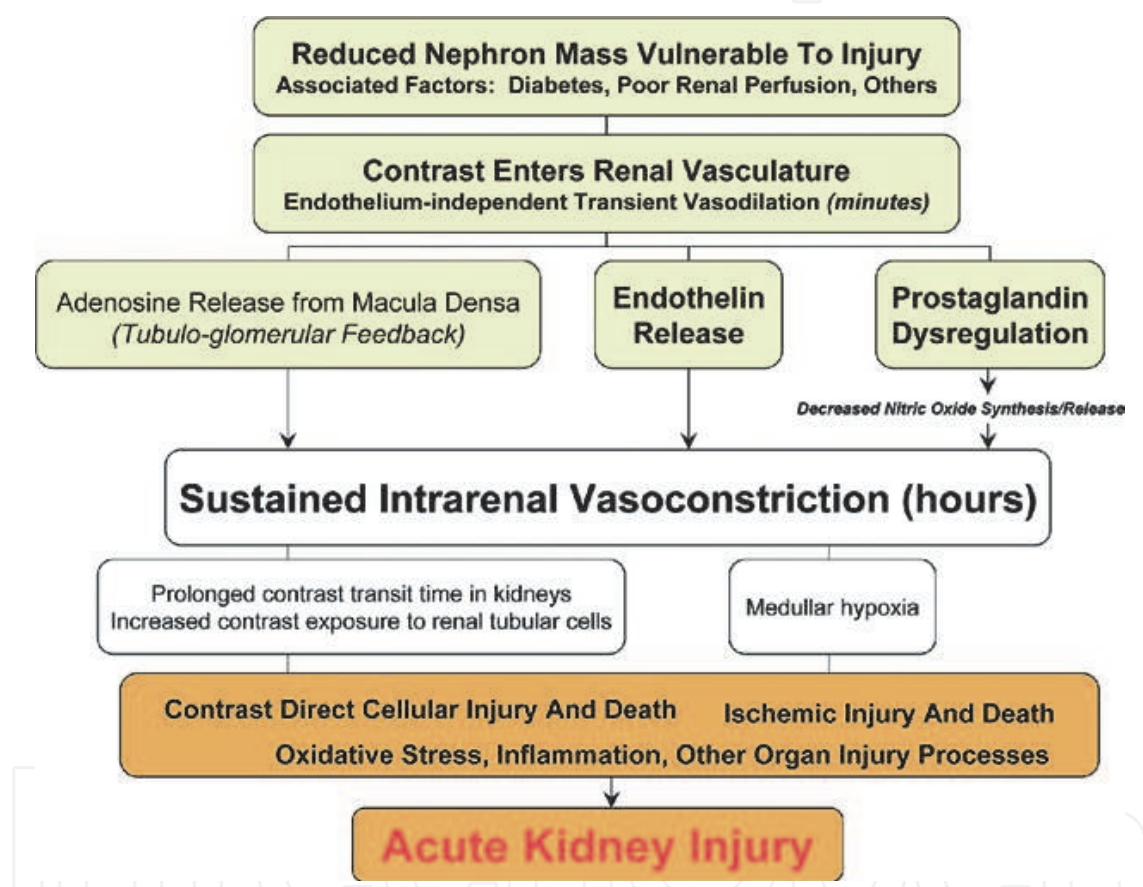


Fig. 2. Postulated pathophysiology of contrast-induced AKI (McCullough, 2008).

4.2 Contrast medium use

In patients at increased risk for contrast-induced AKI undergoing intraarterial administration of contrast, ionic high-osmolality agents pose a greater risk for contrast-induced AKI than low-osmolality agents (Barrett & Carlisle, 1993). Red blood cell deformation, systemic vasodilation, intrarenal vasoconstriction, as well as direct renal tubular toxicity are all more common in contrast agents with osmolality greater than that of blood.

Current evidence suggests that for intraarterial administration in high-risk patients with CKD, particularly those with diabetes mellitus, nonionic, iso-osmolar contrast is associated

with the lowest risk of contrast-induced AKI. Iodixanol (an iso-osmolar contrast media) has been shown to have the lowest risk for contrast-induced AKI in patients with CKD and diabetes mellitus compared with low-osmolar contrast media (McCullough et al. 2006b). The *American College of Cardiology/American Heart Association* guidelines for the management of acute coronary syndromes in patients with CKD listed the use of iso-osmolar contrast media as a class I, Level of Evidence: A recommendation. The *National Kidney Foundation Kidney Disease Outcome Quality Initiative* guidelines have also recommended use of iso-osmolar contrast media in renal dialysis patients to minimize the chances of volume overload and complications before the next dialysis session.

Higher contrast volumes (~100 mL) are associated with higher rates of contrast-induced AKI in patients at risk. However, even small (~30 mL) volumes of iodinated contrast in very high-risk patients can cause contrast-induced AKI and acute renal failure requiring dialysis, suggesting the absence of a threshold effect (Manske et al., 1990). As a general rule, the volume of contrast received should not exceed twice the baseline level of eGFR in mL.

Finally, intraarterial administration of iodinated contrast appears to pose a greater risk of contrast-induced AKI above that with intravenous administration.

4.3 Strategies to further reduce the risk of contrast-induced Acute Kidney Injury

4.3.1 Volume expansion

Adequate intravenous volume expansion with isotonic crystalloid (1.0–1.5 mL/kg/h) for 3–12 h before the procedure and continued for 6–24 h afterwards can lessen the probability of contrast-induced AKI in patients at risk (Mueller et al., 2002). Achieving a good urine output (~150 mL/h) in the 6h after the procedure has been associated with reduced rates of AKI in one study. The data on oral as opposed to intravenous volume expansion as a contrast-induced AKI prevention measure are insufficient.

4.3.2 Pharmacologic agents

No adjunctive medical or mechanical treatment has been proven to be efficacious reducing the risk of AKI after exposure to iodinated contrast. With iodinated contrast, the pharmacologic agents tested in small trials that deserve further evaluation include the antioxidants ascorbic acid and N-acetylcysteine (NAC), aminophylline/theophylline, statins, and prostaglandin E1.

4.3.2.1 Ascorbic acid

Only ascorbic acid has been tested in a multicenter, blinded, placebo-controlled trial (n=231) and been shown to reduce rates of contrast-induced AKI. The dose of ascorbic acid (vitamin C over the counter) used in this trial was 3g orally the night before and 2g orally twice a day after the procedure (Spargias et al., 2004).

4.3.2.2 N-acetylcysteine

Although popular, NAC has not been consistently shown to be effective. Nine meta-analyses have been published, all documenting the significant heterogeneity between studies and pooled odds ratios for NAC approaching unity (Stacul et al., 2006). Importantly, only in those trials where NAC reduced SCr below baseline values because of decreased skeletal muscle production did renal injury rates appear to be reduced.

However, NAC as an antioxidant has been shown to lower rates of AKI and mortality after primary PCI in 1 trial. The recently published REMEDIAL (Renal Insufficiency Following Contrast Media Administration) trial suggested that the use of volume supplementation with sodium bicarbonate together with NAC was more effective than NAC alone in reducing the risk of AKI (Briguori et al., 2007; Brown et al., 2009). Dosing of NAC has varied in the trials; however, the most successful approach has been with 1,200 mg orally twice a day on the day before and after the procedure.

4.3.2.3 Other agents

Fenoldopam, dopamine, calcium-channel blockers, atrial natriuretic peptide, and L-arginine have not been shown to be effective in the prevention of contrast-induced AKI. Furosemide, mannitol, and an endothelin receptor antagonist are potentially detrimental (Stacul et al., 2006).

In general, cardiovascular patients undergoing procedures with iodinated contrast have either high risk for atherosclerosis or have the anatomic presence of disease. Therefore, the vast majority of patients should be on statin therapy with a common low-density lipoprotein cholesterol target of <70 mg/dL. Several studies have demonstrated that patients continuing on statins during cardiovascular procedures including PCI and coronary artery bypass grafting have lower rates of AKI (Khanal et al., 2005). All small, randomized trials published to date support this concept as well. Preservation of endothelial function at the level of the glomerulus and reductions in systemic inflammatory factors are postulated mechanisms by which statins may have renoprotective effects.

4.3.2.4 Dialysis and hemofiltration

Prophylactic hemodialysis or hemofiltration has not been validated as an effective strategy, even when carried out within 1 h or simultaneously with contrast administration.

Hemofiltration, however, performed 6 h before and 12 to 18 h after contrast deserves consideration, given reports of reduced mortality and need for hemodialysis in the postprocedure period in very high-risk patients (Marenzi et al., 2003).

4.3.2.5 Withdrawal of additional nephrotoxic drugs

Despite the lack of more robust data in this area, it is a reasonable practice to withhold nonsteroidal anti-inflammatory drugs, calcineurin inhibitors, high-dose loop diuretics, aminoglycosides, and other nephrotoxic agents if possible for several days before contrast exposure (McCullough, 2008). It is routine practice to withhold metformin before all contrast procedures, not because metformin itself is nephrotoxic but because in the setting of AKI if metformin is continued, lactic acidosis can develop leading to systemic complications and death. As a general rule, metformin should not be restarted until the clinician is confident that the patient has not developed AKI.

4.4 Management of patients receiving iodinated contrast media

An integrated proposed algorithm for the management of patients receiving iodinated contrast media is presented in Figure 3. Since there are no approved pharmaceutical agents for the prevention of this complication, the practitioner should be cautious with the use of any of the drugs suggested. We also recommend that all patients at risk for contrast-induced AKI should have follow-up serum creatinine and electrolyte monitoring daily while in the hospital, and then at 48 to 96h after discharge.

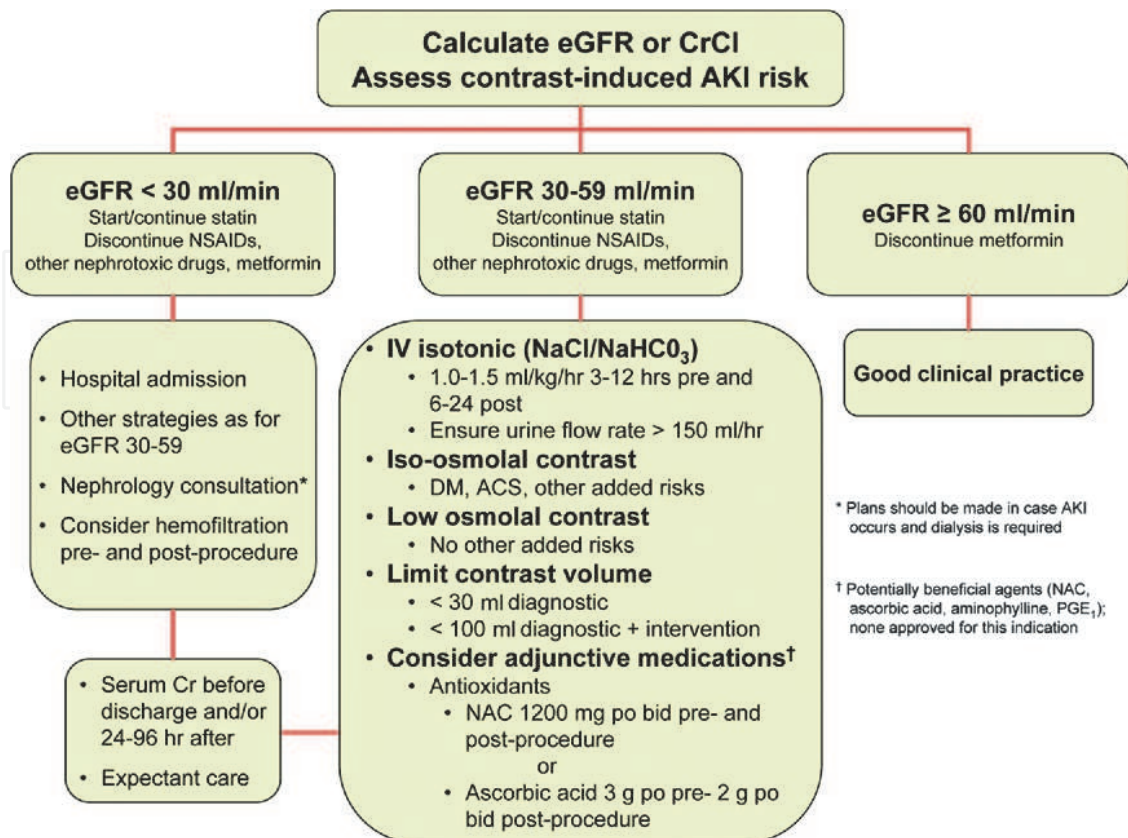


Fig. 3. Proposed algorithm for management of contrast-induced AKI (McCullough, 2008).

5. Conclusion

Patients with chronic kidney disease are at high-risk for CAD and, therefore, coronary angiography is very often needed to establish the diagnosis and severity of CAD. The major challenges in this regard are how to select those patients more likely to have significant CAD, in the absence of typical symptoms suggestive of CAD or myocardial perfusion defects disclosed by noninvasive testing. Moreover, the possibility of contrast-induced acute kidney injury should signal the need for a careful and more judicious decision-making approach by the cardiologist, working in tandem with the nephrologist, when trying to establish the risk-benefit ratio for each patient being considered for invasive assessment. Having decided that an invasive procedure is clearly needed, all preventive and therapeutic measurements should be applied to minimize the risk of worsening residual renal function.

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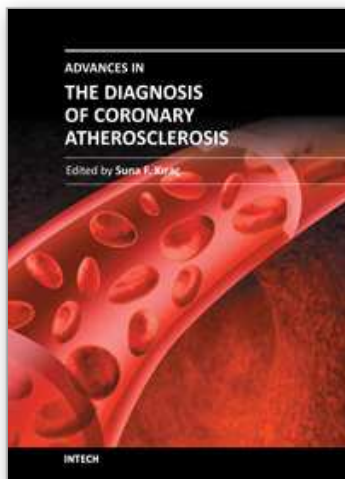
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Coronary artery disease (CAD) and its consequences are most important morbidity and mortality reasons in the developed and developing countries. To prevent hard end-points, early definitive diagnosis and optimum therapy play significant role. Novel advanced diagnostic tests which are biomarkers of inflammation, cell adhesion, cell activation and imaging techniques provide to get the best result in the detection and characterization of calcified or uncalcified atherosclerotic plaques. In spite of last developments in the imaging methods, coronary catheterization is still frequently performed. Following the first cardiac catheterization performed in 1844, date by date historical developments and the mechanics of cardiac catheterization techniques, risks associated with coronary angiography, and also, preventions and treatments of possible complications have been presented in this book. Other important issue is radiation exposure of patients and staff during coronary angiography and scintigraphy. Radiation dose reduction techniques, general radiation protection principles have been discussed in related chapters.

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