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Review

Impact of Chronic Kidney Disease on Decision Making and Management in Transcatheter Aortic Valve Interventions

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ABSTRACT

The coexistence of chronic kidney disease (CKD) and severe aortic stenosis (AS) is common, and the prevalence of both is rising. The 2 conditions are inherently linked in that significant CKD may accelerate the development of AS and severe AS may result in deteriorating kidney function. The volume of and indications for transcatheter aortic valve implantation (TAVI) procedures are ever-increasing, and there are many challenges that need to be considered in patients with concomitant severe AS and CKD being assessed for TAVI. Throughout the process of working these patients up for definitive management of their valvular heart disease, the presence of CKD impacts on diagnostic investigations, treatment decisions, and therapeutic interventions. Herein we review the current literature regarding TAVI in patients with CKD focusing on the decision-making process and specific risks involved in TAVI and CKD. We also provide specific practical strategies to best manage this challenging patient cohort.

RÉSUMÉ

La coexistence de la maladie rénale chronique (MRC) et de la sténose aortique (SA) est fréquente, et la prévalence de ces 2 maladies augmente. Les 2 maladies sont intrinsèquement liées, en ce sens qu'une MRC importante peut accélérer le développement de la SA, et qu'une SA grave peut entraîner la détérioration de la fonction rénale. Le nombre d'interventions de remplacement valvulaire aortique par cathéter (TAVI) et leurs indications ne cessent d'augmenter, mais de nombreux enjeux doivent être considérés chez les patients qui sont atteints de façon concomitante de SA grave et de MRC et qui sont évalués pour subir une intervention TAVI. Durant tout le processus du bilan de santé pour la prise en charge définitive de la valvulopathie des patients, la présence de la MRC a des répercussions sur les examens diagnostiques, les décisions de traitements et les interventions thérapeutiques. Dans le présent article, nous passons en revue la littérature actuelle sur le TAVI chez les patients atteints de MRC, et nous nous penchons sur le processus décisionnel et les risques particuliers inhérents au TAVI et à la MRC. Nous proposons également des stratégies pratiques particulières pour mieux prendre en charge cette cohorte de patients complexes.

Transcatheter aortic valve implantation (TAVI) is now a well-established treatment strategy for severe aortic stenosis (AS). Initially reserved for patients deemed high risk for surgical aortic valve replacement (SAVR), TAVI has now been shown to be comparable with SAVR in intermediate- and low-risk patients in terms of clinical outcomes.[1-3] Chronic kidney disease (CKD) commonly coexists in patients with severe AS, and this provides many challenges in their management strategy. In this review, we aim to explore the relevance of CKD in modern TAVI practice and offer practical guidance to the management of patients with CKD being assessed and undergoing TAVI.

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Chronic Kidney Disease and Aortic Stenosis

Patients with severe AS are often elderly and possess multiple comorbidities, CKD being one of the more prevalent.[4] In a recent analysis of 28,716 patients who underwent TAVI in Germany, 31.5% of patients were found to have CKD $\geq$ stage 3.[5] The prevalence of CKD in the western world has risen because of increasing rates of hypertension, diabetes, and obesity and is associated with poor clinical outcomes. Likewise, because of an aging population, the prevalence of severe AS is also rising. CKD is associated with increased rates of cardiovascular disease, stroke, and death with a graded inverse relationship between baseline glomerular filtration rate (GFR) and poor clinical outcomes.[6-8]

CKD is inherently linked to AS in that it both increases aortic valve disease prevalence and accelerates progression.[4,9] Reduced kidney function results in abnormal bone and mineral metabolism, which in turn leads to calcification within the cardiovascular system, inclusive of the aortic valve. The prevalence of severe AS is higher in patients undergoing haemodialysis than that of the general population.[9] Severe AS

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Table 1. Definition of stages of CKD

	Definition
GFR staging	
G1	Kidney damage with GFR $\geq$ 90 mL/min/1.73 m^2
G2	Kidney damage with GFR of 60-89 mL/min/1.73 m^2
G3a	GFR of 45-59 mL/min/1.73 m^2
G3b	GFR of 30-44 mL/min/1.73 m^2
G4	GFR of 15-29 mL/min/1.73 m^2
G5	GFR $<$ 15 mL/min/1.73 m^2 or kidney failure treated by dialysis or transplantation
Albuminuria staging	
A1	$<$ 30 mg/g
A2	30-300 mg/g
A3	$>$ 300 mg/g

CKD, chronic kidney disease; GFR, glomerular filtration rate.

in turn has a bidirectional relationship with kidney function; AS results in a low cardiac output state and hence reduced kidney perfusion contributing to worsening function.

SAVR vs TAVI in CKD

CKD, although traditionally defined by GFR alone, is now defined as abnormalities of kidney structure or function, present for $>$ 3 months with implications for health, and can be classified into stages based on cause, GFR, and albuminuria[10] (Table 1). The presence of CKD may influence the treatment decision-making process for patients with severe AS. Up to one-third of patients with severe valvular heart disease are refused for operation based on the severity of their kidney disease.[11] There are specific risks associated with both SAVR (periods of hypotension, bleeding requiring blood transfusions, and cardiopulmonary bypass) and TAVI (nephrotoxic contrast loads, hypotension, and bleeding) that may result in worsening of kidney function. Outcomes after both procedures are known to be worse in patients with CKD than in those with normal kidney function.[12-14] However, both have improved outcomes as compared with medical management of severe AS and may be protective to deteriorating kidney function; hence, the presence of significant kidney disease alone should not serve as a reason for turndown for definitive intervention.[15-17] Patients with severe CKD were excluded from the large randomized-controlled trials examining TAVI vs SAVR,[18,19] and so we must rely on observational data to help guide decision making between the 2 treatment options for this cohort.

In the largest observational study to date examining TAVI vs SAVR in CKD, Kumar et al.[20] examined data from the National Inpatient Sample in more than 6500 patients who underwent intervention for severe AS in the United States. The TAVI group, as expected, was older (82.5 $\pm$ 0.2 years vs 76.3 $\pm$ 0.3 years, $P < 0.001$) with a greater burden of comorbidities. In multivariate analysis, it was found that patients who underwent TAVI had lower rates of acute kidney injury (AKI) (odds ratio [OR] 0.18, 95% confidence interval [CI] 0.14-0.22, $P < 0.001$) and lower rates of dialysis-requiring AKI (OR 0.30, 95% CI 0.2-0.44, $P < 0.001$), both of which were associated with increased in-hospital mortality. TAVI was also associated with a significant reduction in in-hospital mortality compared with SAVR (OR 0.47, 95% CI 0.32-0.69, $P < 0.001$) and a reduction in post-operative stroke (OR 0.27, 95% CI 0.13-0.53, $P < 0.001$). Furthermore, TAVI resulted in a shorter length of stay with a similar cost of hospitalization. A limitation of this study is that AKI was defined by a coding of "acute renal failure" as per the International Classification of Diseases, Ninth Revision, Clinical Modification rather than by a standardized definition. This may have led to errors due to miscoding as well as changes in coding practices. However, other smaller studies support this data with lower mortality rates associated with TAVI as compared with SAVR in patients with CKD at the expense of higher pacemaker insertion rates.[21,22] In the absence of randomized-controlled data, these studies suggest that TAVI may be a preferable approach to the treatment of patients with severe AS and CKD.

CKD and TAVI

Contemporary management of severe AS is complex requiring a multidisciplinary heart team approach. The involvement of cardiologists, cardiothoracic surgeons, anaesthetists, radiologists, nursing staff, and other clinical teams concerned with the patient's care is imperative in optimizing patient outcomes. For patients with CKD, this approach is especially important as there is a risk of worsening kidney function before, during, and after TAVI; hence, the involvement of the nephrology service is especially required throughout the decision-making process.

The first management decision required in patients with significant CKD being considered for TAVI is whether or not to proceed at all. Steinmetz et al.[17] examined outcomes of patients with CKD stage 3-5 and severe AS undergoing TAVI as compared with those managed conservatively. They included 360 patients and found that after a mean follow-up of 1.9 years, mortality was greater in the conservatively managed group with a hazard ratio (HR) of 3.95 (95% CI 2.59-6.02) although mortality rates were high in both groups (32.7% with TAVI and 49.5% with conservative management). At 1 year, there was a significant decrease in kidney function in the control group (39.6 $\pm$ 13.9 mL/min to 34.4 $\pm$ 15.3 mL/min), but not in the TAVI group (41.7 $\pm$ 13 mL/min to 42.9 $\pm$ 14.5 mL/min) ($P = 0.001$). Although this is a single-centre retrospective analysis and mortality in both groups was high, it suggests that significant CKD should not be prohibitive to TAVI.

CKD has been shown consistently to result in poor outcomes after TAVI. In a large analysis of the UK TAVI registry including 3980 patients with CKD who underwent TAVI, Ferro et al.[13] showed a graded inverse relationship between estimated GFR and mortality after TAVI. For every 10 mL/min/1.73 m^2 decrease in estimated GFR, the in-hospital mortality increased by 8.2% (95% CI 1.1% to 14.7%; $P = 0.03$) and the long-term mortality (median follow-up 543 days) increased by 4.4% (95% CI 1.2% to 7.5%; $P = 0.007$). CKD stages 3b, 4, and 5 were all independently associated with cumulative mortality. The subsequent need for dialysis was 12.7% and 33% in patients with stage 4 and stage 5 CKD, respectively.

The presence of significant CKD does not only increase mortality after TAVI, but also increases complication rates. In a multicentre analysis of 2075 patients who underwent TAVI, Allende et al.[23] demonstrated that the presence of CKD stage 4 was associated with a higher rate of 30-day stroke ($P = 0.01$)

and major/life-threatening bleeding ($P = 0.001$). There was no difference in rates of pacemaker insertion or significant aortic regurgitation. AKI occurred in 18.4% of patients with a significant association between the severity of CKD before the procedure and the occurrence of AKI and the need for dialysis after TAVI. Patients who suffered AKI had higher 30-day and cumulative overall mortality. Multivariate analysis of this cohort demonstrated that the presence of atrial fibrillation (AF) (HR 2.29, 95% CI 1.47-3.58; $P = 0.001$) and dialysis therapy (HR 1.86, 95% CI 1.17-2.97; $P = 0.009$) determined a higher risk of cumulative late death. Patients with CKD, on dialysis, and with a history of AF had very high mortality rates of 70.8% at 1 year (vs 20.1% in patients without these 2 risk factors; $P < 0.001$) and 100% at 2 years (vs 28.4% without these 2 risk factors; $P < 0.001$); this would lead one to question the utility of TAVI in this specific subgroup.

The potential need for dialysis after TAVI is an important consideration. Dialysis is a considerable strain on both a health care system and patients and results in a significant reduction in quality of life for those of who require it. It predicts poor outcomes and an increase in cardiovascular disease and death. An analysis of the Society of Thoracic Surgeons/American College of Cardiology Transcatheter Valve Therapy registry demonstrated that 14.6% of patients undergoing TAVI with CKD stage 4 will require dialysis at 1 year. In patients with CKD stage 5, more than one-third (35.3%) will require dialysis within 30 days and nearly two-thirds (60.1%) at 1 year. Given these high rates, one should question the appropriateness of TAVI in this group on a case-by-case basis with an emphasis on quality of life. If this risk is deemed unacceptable to the patient, cardiologist, or nephrologist, then TAVI should be reconsidered and a palliative approach may be adopted.

AKI After TAVI

The impact of AKI after TAVI is known to be significant, and even small reductions in GFR have been shown to be associated with increased mortality.[23,24] The definition of AKI is varied resulting in a wide range of reported prevalence of AKI after TAVI (8.3% to 57%); this is mainly due to varying definitions of AKI.[25] The currently accepted and recommended definition within the TAVI literature is the Acute Kidney Injury Network classification used by the Valve Academic Research Consortium-2[26] (Table 2).

The pathogenesis of AKI after TAVI is multifactorial. The most important etiologic factors are the use of nephrotoxic contrast, transient haemodynamic instability, and atheroembolization of cholesterol deposits to the renal vasculature.[27,28] There are several factors that have been identified as predictors for AKI in TAVI. These can be categorized as preoperative, intraoperative, and postoperative (Table 3). In addition, the age, creatinine clearance, ejection fraction score has been validated to predict the risk of AKI in patients undergoing TAVI[29] and can be useful to identify patients at high risk of AKI.

Preoperative risk factors

One of the strongest predictors for AKI after TAVI is pre-existing CKD with an inverse relationship between baseline GFR and rates of AKI.[23,30,31] Other risk factors include high Society of Thoracic Surgeons score, diabetes mellitus, increasing age, peripheral vascular disease, and chronic obstructive pulmonary disease.[25] Many of these variables are not modifiable, but identification of patients at an increased risk of AKI may allow adaptation of a procedural technique to attempt to reduce this risk.

Table 2. VARC-2 definition of acute kidney injury

Acute Kidney Injury Network classification
Stage 1
Increase in serum creatinine to 150% to 199% (1.5-1.99 × increase compared with baseline*) **OR**
Increase in serum creatinine of ≥ 0.3 mg/dL (≥ 26.4 mmol/L) **OR**
Urine output < 0.5 mL/kg/h for > 6 but < 12 h
Stage 2
Increase in serum creatinine to 200% to 299% (2.0-2.99 × increase compared with baseline) **OR**
Urine output < 0.5 mL/kg/h for > 12 but < 24 h
Stage 3
Increase in serum creatinine to ≥ 300% (≥ 3 × increase compared with baseline) **OR**
Serum creatinine of ≥ 4.0 mg/dL (≥ 354 mmol/L) with an increase of at least 0.5 mg/dL (44 mmol/L) **OR**
Urine output < 0.3 mL/kg/h for ≥ 24 h **OR**
Anuria for ≥ 12 h **OR**
Need for dialysis

VARC-2, valve academic research consortium-2.
*The increase in creatinine must occur within 48 h.

Intraoperative risk factors

Intraoperative risk factors are the most amenable to targeted approaches to reduce rates of AKI. A transfemoral (TF) approach is associated with lower rates of AKI than a transapical (TA) approach and so is preferable if feasible.[32] The use of larger doses of contrast has been shown in one study to result in higher rates of AKI in TAVI, but not confirmed in others.[15,25,33] Despite this inconsistent evidence, increased contrast dose is a well-established risk factor for AKI in patients undergoing angiography and percutaneous coronary intervention[34] so efforts to keep contrast use to a minimum during TAVI is advisable. Blood transfusion is strongly associated with AKI after SAVR and TAVI with rates of AKI in patients having received transfusion more than 3 times that of patients who have not.[35,36] This may reflect not only associated hypotension related to acute blood loss but also the presence of nephrotoxic-free haemoglobin and iron due to transfusion and a secondary inflammatory response resulting in AKI.[37] Higher transfusion targets have not been shown to improve outcomes or reduce AKI in cardiac surgery.[38,39] Intraoperative hypotension due to blood loss, anaesthesia, or decreased cardiac output secondary to aortic valve obstruction during TAVI valve positioning and/or rapid pacing is a further important intraoperative cause of AKI.

Postoperative risk factors

After TAVI, risk factors associated with AKI generally reflect preoperative state and intraoperative complications. A prolonged stay in the cardiac intensive care unit or intensive care unit is associated with increased rates of AKI and poor outcomes.[40] This however is clearly confounded by the fact that patients remaining in the cardiac intensive care unit or

Table 3. Risk factors and management strategies for AKI in TAVI

Risk factors	Management strategies
Preoperative	
• Pre-existing CKD—GFR inversely proportional to risk • Hypertension • High STS/EuroSCORE • Diabetes mellitus • Advanced age • Congestive cardiac failure • Peripheral vascular disease • Chronic obstructive pulmonary disease	• Heart team assessment of suitability/role for TAVI on a case-by-case bases • Consider preoperative risk—ACEF score, risk factors • Preoperative optimization of kidney function ○ Nephrology input ○ Hold nephrotoxic agents ○ Consider holding ACEi/ARB pre-TAVI • Reduce risk of CI-AKI ○ Low/isomolar contrast—limited dose ○ Consider CT or angiography alone ○ Dilute contrast ○ Low-dose CT protocols ○ Consider use of NAC ○ Optimization of fluid status before contrast administration ○ CO^2 angiography for peripheral access ○ Consider TEE/balloon sizing • Dialysis on day before TAVI if already dialyzed
Intraoperative	
• Blood transfusion • Transapical approach • Contrast—agent and dose • Intraoperative hypotension	• Use of ultrasound for vascular access • Adequate vascular closure • High threshold for blood transfusion • Favour transfemoral approach • Reduce risk of CI-AKI (see Preoperative) • Dedicated cardiac anaesthetist for procedure
Postoperative	
• Anaemia • Prolonged ICU/CICU stay • Reduced left ventricular ejection fraction	• High threshold for transfusion • Careful management of fluid status after TAVI ○ May need to reduce diuretics • Ongoing nephrology input • Check kidney function 3-5 d after TAVI

ACEF, age, creatinine, ejection fraction score; ACEi, angiotensin-converting enzyme inhibitor; AKI, acute kidney injury; ARB, angiotensin receptor blocker; CI-AKI, contrast induced acute kidney injury; CICU, cardiac intensive care unit; CKD, chronic kidney disease; CT, computed tomography; EuroSCORE, European System for Cardiac Operative Risk Evaluation; GFR, glomerular filtration rate; ICU, intensive care unit; NAC, n-acetylcysteine; STS, Society of Thoracic Surgeons; TAVI, transcatheter aortic valve intervention; TEE, transesophageal echocardiography.

intensive care unit have likely suffered a complication of the procedure. Likewise, postoperative anaemia predicts increased rates of AKI that may reflect haemorrhage, hypotension, and need for blood transfusions, all risk factors in themselves.[40]

Practical Guidance for Management of Patients With Severe AS and CKD

Having established the issues and risks involved in undertaking TAVI in patients with CKD and severe AS, we hope to offer some practical guidance as to the management of these patients (Table 3). As many of the strategies to reduce poor outcomes in this cohort are aimed at reducing the risk of AKI, we have split the management strategy into preoperative, intraoperative, and postoperative.

Preoperative management

The most important preoperative management decision in patients with CKD is whether to proceed with TAVI or not. As in all patients being assessed for TAVI, this requires a patient-centred multidisciplinary heart team approach and all patient factors and comorbidities need to be considered of which age and frailty are of particular importance. It is worth noting that TAVI has improved outcomes over conservative management in patients with CKD and severe AS, but as in all therapeutic interventions, one must consider the overall benefit that would be gained by the patient and the impact on both longevity and quality of life. As stated previously, patients with CKD stage 4 or 5 who are not already on dialysis are at high risk for needing dialysis after TAVI, and hence significant consideration should be given before proceeding with TAVI, particularly in the case of concomitant AF given the documented poor outcomes in this group.[23]

Optimization of the patient's kidney function before TAVI is imperative and should be carried out under the guidance of nephrology colleagues. This will centre on the management of blood pressure, volume overload, anaemia, glycemic control in diabetics, metabolic acidosis, and the complications of long-standing CKD such as mineral and bone disorders. The use of angiotensin-converting enzyme inhibitors and angiotensin-receptor blockers is central to managing hypertension and preventing progression of CKD. Although their chronic use results in improved outcomes in CKD, they may exacerbate intraoperative hypotension and so it is reasonable to hold them 24 hours before the procedure, particularly in patients with low blood pressure at baseline.[41] In noncardiac operations, this strategy has been shown to reduce perioperative hypotension, mortality, and postoperative cardiovascular events.[42] Stopping other nephrotoxic agents before TAVI is also advised (eg, nonsteroidal anti-inflammatory drugs). Ideally patients on dialysis should be dialyzed on the day before TAVI to optimize fluid status. Although often requested, the need for dialysis after contrast administration is not advised and patients can safely wait for their next scheduled session.

The prevention of contrast-induced AKI (CI-AKI) is a topic that has been studied extensively and has much

conflicting evidence. There are no specific pharmacologic therapies that have been consistently shown to reduce CI-AKI; however, the use of oral n-acetylcysteine is cheap, safe, and has shown some benefit so should be considered.[43,44] The administration of intravenous fluids (IVF) for patients with CKD is a common strategy employed in cardiac catheterization laboratories to attempt to reduce the rate CI-AKI in patients with CKD with evidence that this can be both harmful and beneficial.[45,46] It is likely that both hypovolemia and hypervolemia increase the risk of CI-AKI and so euvolemia is the ideal status for patients to undergo cardiac procedures involving contrast administration. This is especially true for patients undergoing TAVI who are at risk of intraoperative hypotension, which is known to worsen outcomes. The decision to use IVF, along with the decision to give or hold diuretics preoperatively, should be individualized to each patient aiming for a euvolemic state. There is no consistent evidence that sodium bicarbonate is superior to normal saline for this purpose.[47]

The workup for TAVI in many centres may involve cardiac catheterization with aortic and iliofemoral angiography along with computed tomography (CT) of the aortic root and CT angiography of the descending aorta and iliofemoral vessels for transcatheter valve sizing and assessing vascular access for TAVI. Both investigations involve contrast administration and increase the risk of worsening kidney function before TAVI. Patients may not require both tests and so this decision should be individualized based on their prior investigations. If both investigations are to be preformed, adequate time between the two, and before proceeding with TAVI, should be allowed to ensure kidney recovery. In preoperative coronary assessment, limiting the number of views taken of each coronary artery and use of dilute contrast to assess vascular access may reduce contrast load. CT alone can be used to assess for concomitant coronary disease, valve sizing, and access route negating the need for cardiac catheterization. With modern imaging technology, ultralow contrast protocols have been shown to be effective in screening patients for suitability for TAVI, with doses delivered as low as 20 mL.[48,49] Likewise, if there has been a recent cardiac catheterization carried out and there are concerns with contrast load from an additional CT, then valve sizing can be performed with transesophageal echocardiography and/or balloon sizing.

Intraoperative

The intraoperative management of patients with CKD is centred on the avoidance of the risk factors for AKI, namely CI-AKI, hypotension, bleeding, and a TA approach.

Low-osmolar or isomolar contrast agents should be used to lower the risk of CI-AKI and administration should be kept to a minimum. There is no consistent evidence that either low or isomolar contrast agents are superior to the other.[50] Use of ultrasound to guide common femoral puncture may allow the avoidance of contrast use to both guide and confirm puncture site. It has also been shown to reduce vascular complications and bleeding that can contribute to AKI.[51] If aortoiliac angiography is required, carbon dioxide gas can be used in conjunction with digital subtraction angiography without the risk of AKI.[52] This should not be used above the diaphragm because of the risk of coronary, cerebral, or spinal gas embolism. Aortic root angiography during TAVI in patients with CKD should be kept to a minimum and may even be avoided for valve-in-valve cases where transcatheter heart valve positioning can be achieved without the need for aortography. If required, dilute contrast can be used to minimize dose delivered. Intraoperative transesophageal echocardiography may also assist in valve placement and valve sizing to limit contrast use.

TA-TAVI is associated with higher risk of AKI as compared with TF-TAVI; however, whether this is a direct consequence of the TA procedure or a reflection of a different patient group is unclear. Patients who undergo TA-TAVI are generally not suitable for TF-TAVI due to peripheral vascular disease that itself is known to be a risk factor for AKI, and these patients typically have more comorbidities and predispositions to AKI. TA-TAVI has several procedural steps that may lead to AKI. Use of general anaesthesia can lead to hypotension, which in turn is a risk factor for AKI. Bleeding and the need for blood transfusions are also more likely in TA-TAVI and are strong predictors for AKI. It is unclear whether alternative access routes such as transcarotid or transaxillary TAVI are similarly associated with higher rates of AKI; this requires further investigation.

A dedicated cardiac anaesthetist is advised for all TAVI procedures.[53] Patients undergoing TAVI are prone to large swings in haemodynamics, and rapid and appropriate response is required for control and limitation of complications, including that of AKI.

Postoperative

Management of a patient's fluid status is again imperative in prevention of AKI after TAVI. Many patients are on diuretic therapy to treat heart failure secondary to severe AS. After TAVI there should be a significant improvement in haemodynamics and diuretic therapy may need to be reduced or stopped altogether. This needs to be assessed on a case-by-case basis. In general, IVF are not required after TAVI as patients should be able to take oral fluids almost immediately after the procedure if local anaesthesia has been used. Nephrotoxic agents, particularly nonsteroidal anti-inflammatory drugs for analgesia, should be avoided in patients with established CKD.

In modern TAVI practice, next day discharge in uncomplicated cases is common and has been shown to be safe.[54] The declaration of AKI in patients after TAVI, however, may take over 72 hours so we would advise testing of kidney function at 3 to 5 days after TAVI in patients with CKD to assess for AKI.

Conclusions

The prevalence of CKD in the general population is rising, as are the indications for and volume of TAVI. The issues involved in undertaking TAVI in patients with CKD need to be recognized and considered throughout the process of the TAVI procedure, from planning to postoperative care.

Disclosures

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