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Title page

Kidney injury as postinterventional complication of TAVI

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Short running title: Kidney injury and TAVI

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Abstract

Transcatheter aortic valve implantation (TAVI) is an accepted treatment approach of aortic stenosis. In the beginning, this technique was executed in high risk patients only. Today, intermediate risk patients are also amenable for TAVI, as long as the transfemoral approach is chosen. Numerous predictors have been identified that could lead to periprocedural complications and are defined by patient comorbidities as well as being inherent to the technical approach. Although vascular complications and postinterventional paravalvular regurgitation have been minimized over the past years by revised technologies and techniques, there is a prevailing individual risk brought about by the specific pathophysiology of the cardiorenal syndrome.

Key words: transcatheter aortic valve replacement -·aortic valve stenosis -·renal insufficiency -·contrast media - forced diuresis

Abbreviations: Contrast media: CM; Transcatheter aortic valve implantation: TAVI; estimated glomerular filtration rate: eGFR; chronic kidney disease: CKD; acute kidney failure: AKI; EuroSCORE: European System for Cardiac Operative Risk Evaluation

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Michael Morcos received honoraria from NovoNordisk, BerlinChemie, Sanofi Aventis, Bayer, Astra Zeneca and Amgen

Christof Burgdorf Andrijana Vukadinivikj and Joerg Latus have no conflicts of interest to declare

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Consent for publication

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Introduction

Nowadays, interventional therapies are of crucial importance in treating structural heart diseases. In particular, the therapy of aortic valve stenosis underwent fundamental changes over the past 10 years with the establishment of the catheter-based aortic valve implantation ("transcatheter aortic valve implantation", TAVI). Initially, TAVI was recommended for treatment of high-risk patients only [1]. Due to positive results from several studies [2-5], TAVI now qualifies as a first line therapy in patients with medium operational risk, at least when a transfemoral approach is feasible: A recent study displayed lower rates of in-hospital mortality and acute kidney injury using a transfemoral rather than a transapical approach [6]. In patients with pre-existing kidney disease, TAVI was associated with a significantly lower incidence of AKI when compared to surgery [7]. Other studies showed that TAVI is associated with an increased survival throughout 2 years of follow-up when compared to surgical aortic valve replacement in intermediate and high-risk patients [8]. The benefit was even greater among females and patients undergoing transfemoral TAVI. Similar results were found in a study by Gaede et al. [9]: While in the low risk group in-hospital mortality was similar, all other risk groups displayed lower in-hospital mortality after TAVI than after surgery. Importantly, a recent metaanalysis confirmed superiority of TAVI even in low risk patients [10]. A transapical approach of TAVI might however still be more favorable as compared to surgical valve replacement if a transfemoral approach is technically not possible [11].

Several studies employing new-generation valve types underline these favorable outcomes [12-17]. Finkelstein et al. showed good outcomes concerning safety and efficacy, while some procedural and post-procedural outcomes differed significantly between valve types [16]. A recent study (SOLVE TAVI) in high-risk patients compared new generation self-expanding valves with balloon expanding valves as to the composite of all-cause mortality, stroke, permanent pacemaker implantation, and leakage and found similar outcome results [17]. The SCOPE 1 study compared the self-expanding ACURATE neo TAVR system with the balloon-expandable SAPIEN 3 TAVR system and showed that the employment of the Accurate Neo Valve was correlated with a higher amount of injected CM and thus a higher incidence of kidney injury [18]. Taken together, new generation valves are safe and efficient; nevertheless, some differences can be seen concerning periprocedural complications. Therefore, valve types should be chosen with respect to the patients individual conditions.

In general, TAVI induced kidney injury occurs in up to more than a third of patients, being associated with a longer in-hospital stay and an increased mortality [19,20]. For periprocedural complications numerous predictors have been identified, on the one hand defined by the patients` individual comorbidities, and on the other hand represented by procedural

complications. But while vascular complications and post-interventional paravalvular insufficiency have been significantly decreased in recent years through technical innovations, there are still considerable individual predisposing risk factors caused by complex cardiorenal interactions [21] as defined by the so called cardiorenal syndrome, which is defined as 'disorders of the heart and kidneys whereby acute or chronic dysfunction in one organ may induce acute or chronic dysfunction of the other'. Five subtypes of the cardiorenal syndrome have been identified [22]. These predisposing risk factors in TAVI require a closer look.

Main text

Chronic renal failure before TAVI

Based on the estimated glomerular filtration rate (eGFR), a pre-existing chronic kidney disease ("Chronic kidney disease", CKD) stage 3b or higher (eGFR <45 ml/min/1.73 m^2; [23], is found in about 30% of TAVI patients in the FRANCE-2 register (n = 2929). The cumulative 30-day and 1-year mortality after TAVI in this group was significantly higher than in patients with CKD 1-3a (eGFR ≥45 ml/min/1.73 m^2). Accordingly, the UK-TAVI register (n = 3980) demonstrates, that a decrease in eGFR by 10 ml/min/1.73 m2 is correlated with an increase in hospital mortality and cumulative mortality after TAVI amounting to 8.2% and 4.4%, respectively [24]. Moreover, a meta-analysis (n = 5266) reported an incremental all-cause mortality with a stepwise increase of 19 % with every mg/dl creatinine [25]. Importantly, the procedural success rate for TAVI is lower in patients with CKD 5 (92.7%) than in patients with CKD 1–4 (97.9%; [23]). Dialysis itself is an independent predictor of mortality using TAVI. Interestingly, in dialysis patients, pulmonary hypertension >60 mmHg appears to be the only significant risk factor for 1-year mortality [26].

On the contrary, it was found that in more than half of the patients (55.7–64%) TAVI can lead to an improvement in kidney function [7,27,28]. Especially in patients with previously severely impaired eGFR, TAVI significantly increased eGFR from 20 to 28 ml/min/1.73 m2 (n = 54; [28]). Recovery of kidney function can be seen in 25% of TAVI patients with CKD thus being more frequent than acute renal injury caused by TAVI [29]. Impaired lung function, previous aortic valve surgery and a severe impairment of renal function at baseline predicted a higher likelihood of renal recovery, while diabetes mellitus, gender, age, anemia and a higher *Society of thoracic risk score* reduced the likelihood of a recovery [29-31]. Moreover, a recent meta-analysis showed improved renal outcomes using TAVI rather than surgical valve replacement despite the exposure to contrast media (CM) and a higher incidence of pacemaker implantations [32]. It is conceivable that improved hemodynamics after TAVI may have led to improvements in kidney perfusion as improved eGFR was accompanied by an increased

cardiac output (from 4.25 ± 1.29 l / min to 4.52 ± 1.4 l / min, n = 301). Thus, considering a patient`s individual risk of acute renal failure after TAVI, one would have to weigh the intra-interventional CM exposure against postinterventionally improved hemodynamics.

Acute kidney injury after TAVI

The procedure of TAVI includes the administration of CM and episodes of pronounced hypotensions ("rapid pacing") for balloon valvuloplasty and valve implantation both of which imply an increased risk for acute kidney injury (AKI). Significant hypotension is common even during uncomplicated TAVI due to the potential need for rapid pacing during pre-dilatation and valve implantation, as well as a transient loss of cardiac output during valve deployment. It thus would be favorable to minimize duration of rapid pacing, to abstain form predilating the aortic annulus if possible, to closely monitor intraprocedural hypotension, and to minimize perioperative inotropic support to avoid periprocedural complications including kidney injury [33,7,34-36].

As there is a high prevalence of comorbid generalized atherosclerosis in these patients, the manipulation with large-lumen aortic TAVI catheters additionally carries the risk of renal cholesterol embolism representing a potential risk factor for AKI [7,41-45,33,46-50]. For instance scraping of aortic plaques by catheters has been observed in >50% of subjects receiving percutaneous coronary intervention and has been defined as a major risk factor for renal injury, which varies between 8-17% after cardiac intervention [37-40].

The risk of embolization is even higher using the transapical approach. The latter has to be chosen, if advanced arterial occlusive disease prevents from using the transfemoral approach [51,50,52]. Massive embolization can cause AKI shortly after the intervention (within 1 week), or progressive kidney dysfunction within several weeks. In a study, mean duration between vascular intervention and diagnosis of AKI was found to be 5.3 weeks [41,53].

The transapical approach in TAVI usually requires less CM, when compared to a transfemoral approach. It is, however, associated with a significantly increased risk of acute kidney injury, even when adjusted for baseline estimated glomerular filtration rate [33,54]. Several hypotheses try to address this point: The transapical approach requires general anesthesia, thus increasing the risk for hypotensive episodes which again predispose to AKI [55], while the surgical trauma might impact kidney function by triggering an inflammatory reaction. But whatever the triggers are, all studies consistently showed a reduced rate of kidney injury using the transfemoral approach for TAVI.

A decrease in renal blood flow with worsening kidney function can also be due to relevant post-procedural paravalvular regurgitation [14,56-58]. The acute volume load of a hypertrophied left

ventricle may entail a critical increase in left ventricular wall tension, that finally leads to sudden cardiac decompensation which again would aggravate "arterial underfilling" of the kidneys. To date larger systematic studies investigating the influence of acute alterations in hemodynamics on the occurrence of acute kidney injury are lacking. A recent trial found an increase in GFR with improved cardiac output, while patients with acute kidney injury did not experience an increase in cardiac output [28]. Another small retrospective study documented that the volume of applied CM correlated with the occurrence of AKI on short-term. It moreover correlated with the CMVxSCr/BW value, a predictor of acute kidney injury with a cut off value of 2,99 [59], actually ranging lower than the cutoff for CM induced nephropathy after percutaneous coronary intervention [60].

Usually, standard TAVI techniques require the use of contrast media (CM) for aortic root angiography and assessment of the function of the implanted valve. Under certain circumstances, a higher amount of CM must be used to confirm hemostasis and correct vascular access. Since the use of CM is an important factor for kidney injury in various interventions, one should apply as little CM as reasonably achievable [61]. It has been demonstrated that calculating the ratio of CM volume /GFR ratio might be helpful in preventing kidney injury: a ratio of 3.9 was specified to predict kidney injury with 71% sensitivity and 80% specificity [62]. Other authors observed a possible association between a higher incidence of AKI and 30-day mortality with regard to the extensive use of CM during TAVI among high-risk patients with preexisting renal impairment [33,63]. This is in line with findings of a study in 270 TAVI patients, showing that postoperative AKI depend on the amount of contrast agent used intra-interventionally. In this study, a higher amount of CM was an independent risk factor for AKI [33,64]. These findings again are supported by findings of Yamamoto et al. who found a correlation between the CM-dosed applied and the incidence of AKI in 415 patients [65]. Nevertheless, not all studies found a clear association between the amount of CM and the incidence of kidney injury, especially when small amounts of CM were used [66,67].

The comparative interpretation of CM-associated periinterventional complications from various studies is difficult as patient populations were very heterogeneous with respect to comorbidity and the various definitions of kidney injury that were used. Nevertheless, due to the likely harmful effects of CM, unnecessarily high amounts of CM should be avoided [61, 68, 69,70]. Finally, a good preprocedural echocardiographic assessment of the aortic valve stenosis may help to reduce the amount of contrast media [71].

The prevalence of acute kidney failure (AKI) after TAVI varies considerably, depending on the definition used for acute kidney injury. Correspondingly, the incidence of AKI after TAVI ranges between 8.3-58% [33], while 2–40% of these patients require hemodialysis [50]. In hospital mortality, as well as 30-day and 1-year mortality rates are 4- 6 times higher in patients with

AKI as compared with patients with preserved kidney function after TAVI regardless of whether kidney function recovered later on [7,27]. Again, it is noteworthy that the transapical approach is significantly more likely to induce AKI than the transfemoral route [5, 6,11,54,55,64,72].

Different predictors are described for AKI in relation to TAVI. Regardless of a pre-existing anemia or vascular/periprocedural bleeding, the intra- and post-procedural erythrocyte concentrates given represent a strong and incremental risk predictor of kidney failure (≥5 EC, odds ratio (OR): 4.81 [1.45-15.95]; 3–4 EK, OR: 3.05 [1.24-7.53]; 1-2 EK, OR: 1.47 [0.98 to 2.22] [73]). While valve thrombosis and cerebral ischemic events post-TAVI are still a significant issue, antithrombotic therapy is associated with higher rates of bleeding [74] that again would increase the risk of acute kidney injury. Patients undergoing TAVI often have multiple co-morbidities like chronic kidney disease, that on its own already augment the bleeding risk [49]. Bleeding post-TAVI thus is associated with adverse clinical outcomes and increased mortality, diminishing the benefits of antithrombotic therapy [75]. A recent study revealed that patients undergoing TAVI treated with oral anticoagulation alone were at lower risk for bleeding over a period of 1 month or 1 year than those who additionally were prescribed clopidogrel [76]. Therefore, tailoring antithrombotic therapy and optimizing duration of treatment appears as being important. Scoring systems, such as the EuroSCORE II and the Society of Thoracic Surgeons (STS) risk score, can be utilized to stratify the risk of patients undergoing TAVI and to support the use of an adequate anticoagulation [77-79]. Peripheral atherosclerosis, chronic heart failure, blood transfusion and increase in leukocytes up to 72 h after TAVI may be used for further risk prediction of AKI [73]. Accordingly, several studies are currently ongoing (see ClinicalTrials.gov) that investigate anticoagulation strategies and occurring bleeding risks in patients undergoing TAVI.

End stage renal disease and TAVI

The prevalence of valvular heart disease in dialysis patients (ESRD) is significantly increased as compared to the general population. After a mean dialysis period of 4 years more than half of patients show structural changes in the area of the aortic valve, while functional aortic stenosis is documented in 13% of patients [80]. The incidence of aortic stenosis amounts to 3.3% per year for dialysis patients [81]. Uremia-related changes in the calcium-phosphate balance within the framework of CKD-MBD ("mineral and bone disorder") contribute to the high prevalence and rapid progression of valve calcifications in patients with ESRD [82]. Ultimately, post-TAVI patients with ESRD, have an increased 30-day mortality rate (13% vs 6%) and a lower 1-year survival rate (57.4% vs 77.4%) as compared to non-dialysis patients [83]. Due to

the heterogeneity of these patients, it is difficult to make a statement which applies to all patients.

As far as dialysis patients are concerned a recent metaanalysis calculated higher short- and long-term mortality, a higher rate of life threatening and/or major bleeding complications, a higher pacemaker implantation and device failure rate, while vascular complications were comparable to non-dialysis patients [84,85]. Dialysis patients at the same time often display a worse natural outcome while not being eligible for surgery. Thus, although dialysis patients do have a worse postprocedural outcome TAVI often turns out to be the only option available. One must therefore, carefully select the patients and consider specific hemodynamic problems that arise with the placement of arterio-venous shunts in these multimorbid group of patients. Therapeutic decisions would thus have to be made on an individual basis in each case.

TAVI in kidney transplant patients

There are hardly any available data in kidney transplanted patients with catheter-assisted aortic valve replacement. A small German study retrospectively compared kidney-transplant patients over a 12-month time period. While in the TAVI group (n = 8) all patients were alive after 12 month, the surgical group (n = 18) showed an 11% hospital and a 16% 1-year mortality [86]. It was concluded that in this specific patient group, TAVI can be carried out safely. This study however has a rather episodic character as only a small number of patients were included without randomization. Moreover, the logistic EuroSCORE in both groups ran below 10% and in the surgical group 11 patients underwent combined interventions.

Pathophysiology and prophylaxis of kidney injury after TAVI

In the past, numerous studies have been carried to find pharmacological interventions that would be able to prevent CM induced kidney injury. With the possible exception of adequate hydration, none of them showed any significant effects [87]. These findings may be due to very heterogeneous patient populations investigated and to the fact that creatinine performs rather poorly as an end point marker.

High molar contrast media carry a higher risk of kidney injury than low or isoosmolar ones do. The term “low osmolar” (400–800 mosmol/kg H_2O) is, however, misleading, as these CM still have an osmolality that is significantly higher than that of plasma (290 mosmol/kg H2O). Further development towards isoosmolar CM (actually being hypoosmolar before the addition of electrolytes) appeared initially logical, since the goal was to achieve lower rates of kidney injury. But importantly, a low osmolality is usually bought with a significantly higher viscosity (two times on the average), which is a crucial pathophysiological factor contributing to the development of AKI [88]. Thus the perfect CM would have to be balanced for both the

osmolality and viscosity. Albeit CM is initially diluted in the bloodstream, they are critically reconcentrated in the tubuli as they are freely filtered but not reabsorbed. An exponential concentration-viscosity relationship leads to a disproportionate increase in fluid viscosity in the distal tubule defining the tubular toxicity of CM. The exponential rise in viscosity significantly reduces the flow rate, thus extending contact time of the tubular cells with the CM thereby increasing its toxic effects (oxidative stress with vasoconstriction and tubular cell death). This leads to simultaneous vasoconstriction of the vasa recta with an ensuing medullary hypoperfusion. This toxic effect may thus be aggravated even with so-called isoosmolar CM, because they exhibit a lower osmotic pressure, a higher molecular viscosity and higher iodine concentrations. Bearing this in mind, it is highly conceivable, that a postinterventional removal of CM by dialysis would never have any effect on tubular toxicity, as tubular cells get into contact with CM during the intervention as soon as it reaches the kidney. It thus appears logic that the pre-interventional state of hydration is of importance as much as is a forced diuresis already being installed before any CM is injected. This might help to decrease toxic contact time of contrast medium with tubular cells and therefore might help to avoid CM-induced kidney failure.

Contrast agent-induced kidney damage can possibly be prevented by real-time balancing

The use of the RenalGuard® system (RenalGuard Solutions, Inc., Milford, USA), allows a forced diuresis with a real time monitoring concerning urine output and as such would help to shorten contact time of CM with tubular cells without endangering patients with fluid overload. In this system, the excreted amount of urine is replaced by an identical amount of sterile liquid in real time. So far, two small monocentric studies showed beneficial effects of the RenalGuard® system in patients with high grade aortic valve stenosis and planned TAVI. The data from the PROTECT-TAVI study showed a significantly lower rate of AKI in the patient group with RenalGuard® therapy in the first 3 days after TAVI, than in patients with saline infusion alone (n = 56; 5.4% vs. 25.0%; [89]). However, there was no indication for hemodialysis in this small study. Significant differences in 30-day mortality or morbidity (according to VARC [Valve Academic Research Consortium] criteria) were also not documented. Comparable results were shown by Visconti and co-workers [90]. The primary endpoint in this study was the appearance of AKI within 7 days of TAVI. In the control group, 10 of 26 patients (38.5%) suffered from AKI, in the group of patients with RenalGuard® therapy (n = 22) only 1 patient (4.5%). Two patients from the control group had to be dialyzed during their inpatient stay, whereas no patient from the RenalGuard® group. Both studies are very limited to their small number of patients, - 112 pts [89] (Protect-TAVI trial) and 48 pts [90], it is possible that a similar effect may be achieved by simply enhancing diuresis by administration of a higher fluid volume. Real-time balancing may, however, turn out to be a useful approach

to prevent contrast media-induced kidney injury, even if no final recommendations can be given so far due to the small number of patients investigated.

With- or without using a real-time balancing system, it must be taken into consideration, that the use of large amounts of isotonic saline for a forced diuresis should be regarded as critical, since high volumes of 0,9% saline may induce hyperchloraemic acidosis with an ensuing renal vascular constriction even increasing the risk of AKI. The use of saline in trials may thus have blunted the clinical benefit of a flow-induced diuresis. Therefore, balanced electrolyte solutions should be used in future studies. Furthermore, as kidney filtration pressures critically depend on central venous pressure, while at the same time up to 70% of CKD patients display the comorbid state of pulmonary hypertension, a preemptive right heart catheterization seems warranted to clarify hydration conditions and to exclude a significant renal venous congestion as the underlying mechanism of a diuretic resistance hindering a forced diuresis. Importantly, the hemodynamic evaluation by right-heart catheterization is the only way to differentiate an isolated postcapillary pulmonary hypertension (ipcPH) from a combined pre- and postcapillary hypertension (cpcPH) that is encountered in up to 25% of renal patients and carries an even greater risk of mortality.

Practical implications

A preinterventional individual risk assessment is mandatory to identify additional risk factors for the development of a CIN (e.g. diabetes mellitus, multiple myeloma, heart failure, and proteinuria). In patients with more severe kidney disease commencing with CKD 3b A1-A3, RAAS-inhibitors should be paused 48 hours prior to the intervention and hypovolemic states should be avoided. In dialysis patients, additional dialysis should only be performed post-procedurally if hypervolemia is imminent due to increased amounts of KM. See table 1 for recommendations for prophylaxis prior to CM administration. Postinterventionally, kidney function parameters should be controlled after 2-5 days [91].

Kidney injury following TAVI is mostly a combination of different pre-renal and intrinsic factors. Therefore, a close supervision of creatinine, BUN and eGFR is necessary, and in specific cases the urine fractional excretion of sodium (FE_{Na}), urine osmolality, and specific weight should be monitored. The FE_{Na} may, however, be difficult to be interpreted under diuretic therapy (table 2).

Conclusions

Renal insufficiency is one of the strongest periinterventional risk predictors for TAVI patients. The degree of renal insufficiency not only reflects the severity of a systemic cardiovascular inflammation, but is also associated with a vascular morphology, that implies an increased intraprocedural risk with respect to renal cholesterol embolizations. Last but not least, a CM-induced AKI is positively associated with the incidence of renal failure and mortality both steeply increasing with an eGFR <45 ml/min/1.73 m^2. A careful consideration of patients at risk with a mindful planning of the interventional setting is therefore of paramount importance to achieve full advantage of the hemodynamic improvement by TAVI. This includes a close supervision of the state of hydration and an adequately increased tubular flow rate [69]. This is of crucial importance, since the CM-associated pathomechanisms are correlated with the tubular contact time of CM. Postinterventional dialysis for the prevention of acute kidney injury is less reasonable than prophylactic measures like a forced diuresis during intervention and reducing CM to the minimum required.

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Table 1. Recommendations for performing prophylaxis prior to CM administration depending on the stage of chronic kidney disease [91].

CKD	Venous CM application	Arterial CM application
G1 und G2 (GFR ≥ 60) A1-A3	-	-
G3a (GFR 45-59) A1-A3	-	-
G3a (GFR 45-59) A1-A3 + RF	-	√
G3b (GFR 30-44) A1-A2	-	√
G3b (GFR 30-44) A3	√	√
G4 (GFR 15-29) und G5* (GFR <15) A1-A3	√	√

GFR, ml/min/1,73 m^2

Legend: - no prophylactic measures recommended, √ prophylactic measures recommended. According to the KDIGO Clinical Practice Guidelines for the Evaluation and Management of Chronic Kidney Disease (Kidney International Supplements. 2013; 3 (1): 1) with CKD stage G1 eGFR ≥90 ml / min , G2 eGFR> 60 ml / min, G3a eGFR 60 ml / min to ≥45 ml / min, G3b eGFR 45 ml / min to 30 ml / min, G4 (eGFR 30 ml / min to 15 ml / min, G5 eGFR> 15 ml / min; stage A1 albumin / creatinine quotient of <30mg / g, A2 30-300mg / g and A3> 300mg / g; * if dialysis is not required.

Table 2: Differentiation between prerenal and intrinsic renal damage.

	prerenal	intrinsic
Urine-osmolality (mmosmol/kg)	>400-500	<350
Urine-sodium (mmol/l)	<20	>30
FE_{Na}	<1%	>2%
U-specific weight	>1020-1030	<1015-1010