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Review

Personalized Contrast-Sparing Strategies in Transcatheter Aortic Valve Implantation: Integrating Renal Risk, Preprocedural Imaging, and Procedural Planning—A Critical Narrative Review

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Abstract

Kidney dysfunction complicates transcatheter aortic valve implantation (TAVI), but iodinated contrast is only one contributor to postprocedural acute kidney injury (AKI). This critical narrative review examines how renal vulnerability and anatomical complexity can guide contrast reduction across coronary assessment, planning, and implantation. Targeted literature searches throughout 27 October 2026 informed a question-specific appraisal of imaging studies, procedural series, comparative cohorts, randomized trials, and guidance. Optimized low-contrast computed tomography angiography (CTA) can preserve comprehensive planning information; noncontrast computed tomography, cardiovascular magnetic resonance, three-dimensional transesophageal echocardiography, and targeted vascular imaging provide alternatives when their combined diagnostic adequacy is established. Clinical studies demonstrate that minimal-contrast and procedural zero-contrast implantation are achievable, but their renal findings are inconsistent. Positive observational associations coexist with neutral AKI comparisons, dialysis despite contrast avoidance, and substantial conversion to rescue contrast in some protocols. Definitions, baseline kidney function, prior iodine exposure, and follow-up differ markedly. Randomized evidence for alternative planning and supportive care addresses specific populations and does not establish that a complete contrast-sparing pathway prevents dialysis, persistent kidney dysfunction, or death. We propose explicit anatomical prerequisites, escalation criteria, and longitudinal renal surveillance within an unvalidated clinical framework. Technical feasibility is better established than comparative renal effectiveness. Prospective studies should compare complete pathways, preserve diagnostic and procedural safety, and measure persistent renal dysfunction and patient-centered outcomes alongside contrast exposure.

Keywords: transcatheter aortic valve implantation; acute kidney injury; chronic kidney disease; iodinated contrast; computed tomography; cardiovascular magnetic resonance; procedural planning; personalized medicine

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1. Introduction

Kidney dysfunction influences imaging, fluid management, and procedural risk in patients considered for transcatheter aortic valve implantation (TAVI). Contemporary guidelines place these decisions within multidisciplinary assessment of anatomy, comorbidity, life expectancy, and patient preferences [1,2]. Valve replacement can have opposing renal effects: bleeding, hypotension, embolization, and contrast exposure may cause injury, whereas relief of obstruction may improve forward flow and reduce congestion. Stable or improved kidney function after TAVI, therefore, cannot, by itself, demonstrate protection from a particular contrast protocol [3,4].

Contrast reduction can target coronary investigation, dedicated valve and access planning, implantation, or the complete treatment episode. These are different interventions. Procedural zero-contrast implantation may follow contrast-enhanced CTA and coronary angiography; iodine-free planning may still be followed by procedural iodine. Table 1 defines the terminology and minimum reporting needed to interpret these pathways.

The clinical objective is adequate information for safe treatment with the least avoidable renal burden. An incompletely characterized annulus, coronary relationship, or access route may pose greater risk than a limited, justified contrast injection. This review links renal vulnerability to the anatomical questions that each imaging strategy must resolve.

We examine contrast exposure at each stage of TAVI care, distinguish diagnostic and procedural feasibility from renal effectiveness, and propose practical criteria for additional imaging and renal follow-up. The proposed framework requires prospective validation. The framework is a proposed clinical synthesis rather than a validated algorithm.

Table 1. Definitions and minimum reporting requirements for renal and contrast outcomes.

Term or Measure	Definition Used in This Review	Reporting Requirement or Qualification
Acute kidney injury	KDIGO: creatinine rise ≥ 0.3 mg/dL within 48 h, rise to ≥1.5 times baseline within 7 days, or urine output < 0.5 mL/kg/h for ≥6 h [5].	State baseline, assessment window, staging system, laboratory schedule and use of urine output. Do not infer causation from timing.
Contrast-associated AKI	AKI occurring after contrast exposure, without establishing that contrast caused the event [6,7].	Identify other potential causes, route of administration and interval from exposure.
Contrast-induced AKI	The component of AKI causally attributable to contrast [6,7].	A postoperative creatinine rise alone cannot establish this diagnosis after TAVI.
VARC kidney outcomes	Contemporary valve-study definitions, including separately identified new renal replacement therapy [8].	Name the VARC version and report new RRT explicitly.
Procedural zero-contrast	No iodinated contrast during the index implantation.	Disclose CTA, coronary angiography/PCI and any safety or rescue injections before or during TAVI.
Planning without iodine	Dedicated root/access assessment performed without iodinated contrast.	Implantation may still use contrast. Disclose prior coronary investigation and use of noniodine agents.
Complete treatment episode without iodine	No iodinated contrast in the defined coronary assessment, planning and implantation episode.	Define the episode and look-back interval; verify outside-hospital studies.
Low/minimal/ultra-low contrast	Study-specific designation based on absolute volume or a renal-function-related index.	Quote the original target and stage of care. The labels do not share one validated numerical definition.
Iodine mass	Iodine mass (g) = undiluted-equivalent volume (mL) × iodine concentration (mg iodine/mL)/1000.	For diluted injections, report undiluted equivalent. Volume alone is insufficient when concentrations differ.

Contrast-volume/eGFR index	Procedural mL divided by eGFR in mL/min/1.73 m².	Use the source study's equation and units. This is a numerical planning index, not a dimensionless biological safe-dose threshold.
Persistent kidney dysfunction	Deterioration remaining at a prespecified follow-up relative to a defined baseline [9].	State the numerical threshold, time point, laboratory completeness and handling of death or dialysis.

Definitions of contrast strategies are proposed reporting conventions for this review, not externally validated treatment thresholds. AKI, acute kidney injury; CTA, computed tomography angiography; eGFR, estimated glomerular filtration rate; KDIGO, Kidney Disease: Improving Global Outcomes; PCI, percutaneous coronary intervention; RRT, renal replacement therapy; TAVI, transcatheter aortic valve implantation; VARC, Valve Academic Research Consortium.

2. Scope and Approach to the Evidence

This is a critical narrative review of adult TAVI, not a systematic review or meta-analysis. Targeted PubMed searches, supplemented by journal websites, official guidance, and reference lists, informed this manuscript throughout 27 October 2026. Supplementary Material File S1 provides representative, reproducible PubMed strategies for the principal renal, contrast, imaging, and procedural concepts. These strategies specify an upper publication date limit of 27 October 2026, no lower publication date limit, and no language filter at retrieval; the evidence discussed in this review is drawn from English-language reports. The supplied strategies make the search concepts explicit but are not presented as a verbatim contemporaneous log of every original targeted search.

We prioritized primary adult clinical studies of renal outcomes after TAVI, contrast-sparing coronary and imaging strategies, and authoritative endpoint or practice guidance. Non-TAVI randomized trials were included when they addressed a relevant preventive intervention, with their applicability to TAVI considered separately. Early case reports and preclinical work were retained only to explain a distinct technical approach, not to establish clinical effectiveness. Subsequent full publications took precedence over preliminary abstracts. Duplicate reports were considered as one study; distinct follow-up publications were retained for their additional outcomes without treating overlapping participants as independent cohorts. Studies were selected for relevance, design, population, exposure ascertainment, and available outcomes rather than a favorable result.

G.E.P., I.N., S.E., and A.I. contributed to the literature review; G.E.P. curated and synthesized the evidence, with validation and critical scientific revision by G.G. and V.N. Article selection was not performed independently in duplicate, and screening counts were not recorded. Appraisal considered imaging adequacy and decision agreement, anatomical selection, prior contrast, rescue or conversion, confounding, endpoint definition, and follow-up completeness. No formal risk-of-bias instrument or GRADE assessment was applied. No pooled estimate was calculated because interventions, populations, exposure windows, and outcome definitions were heterogeneous. These features limit reproducibility and permit study selection and interpretive bias, as discussed in Section 14.2.

3. What Renal Protection Means in TAVI

3.1. Acute Kidney Injury and Attribution

AKI requires a change from a defined baseline. KDIGO criteria include a serum creatinine increase ≥ 0.3 mg/dL within 48 h, ≥1.5 times baseline within seven days, or urine output < 0.5 mL/kg/h for at least six hours. Studies should state the baseline sample, ascertainment schedule, observation window, and staging system. VARC-3 provides contemporary valve-study endpoints, including separately reported new renal replacement therapy (RRT) [5,8].

Contrast-associated AKI (CA-AKI) denotes a temporal association; contrast-induced AKI (CI-AKI) requires causal attribution. Timing alone cannot establish contrast causation after TAVI. ACR–National Kidney Foundation guidance concerning intravenous contrast should also not be transferred uncritically to intra-arterial TAVI exposure, which occurs alongside different hemodynamic and procedural insults [6,7].

Ascertainment affects incidence: testing only the morning after TAVI may miss later AKI, whereas a prolonged window may include intercurrent illness. The Danish nationwide study used a ≥50% creatinine increase within 21 days, or dialysis during admission, rather than the complete standard KDIGO definition and seven-day window. Its events should not be directly equated with those in a protocol using daily 48–72 h surveillance [10].

Urine output is difficult to standardize with diuretic use and avoidance of urinary catheters. Creatinine responds with delay and is influenced by muscle mass and fluid balance. These limitations require explicit surveillance and separate reporting of AKI stage, recovery, and dialysis; they do not justify interpreting an isolated postoperative value as a renal outcome.

3.2. The Kidney Trajectory Before and After Valve Replacement

The renal trajectory after TAVI includes reversible cardiorenal dysfunction as well as intrinsic CKD. In 5190 PARTNER participants, CKD stage usually remained unchanged or improved shortly after TAVR [3]. In Witberg's 894-patient cohort, 720 had stable or improved CKD stage at one month; a ≥10% eGFR deterioration, a different endpoint from CKD-stage change, was associated with higher adjusted two-year mortality (hazard ratio [HR]: 2.16; 95% confidence interval [CI]: 1.24–5.25) [4]. These observations support reassessment beyond discharge without attributing recovery to contrast reduction.

Longer follow-up identifies both recovery and residual risk. In the Danish cohort of 4091 patients, 193 developed AKI. Subsequent one-year dialysis incidence was 9.2% with AKI versus 1.4% without AKI, and mortality was 16.4% versus 6.9%. The adjusted HRs were 7.20 (95% CI: 4.10–12.66) and 2.39 (95% CI: 1.59–3.58), respectively. Complete recovery at 90–180 days occurred in 106/143 AKI survivors with available laboratory measurements (74.1%); this is a survivor and available-case estimate, not a recovery rate for all patients with AKI [10].

A 314-patient retrospective cohort with median two-year follow-up reported attenuation of the adjusted annual eGFR decline in patients with baseline CKD, from −2.83 before TAVR to −0.83 mL/min/1.73 m^2/year afterward [11]. Recovery and subacute-injury cohorts likewise associate persistent dysfunction with worse prognosis [12,13]. However, no randomized contrast pathway comparison establishes prevention of CKD progression. Persistent dysfunction, later dialysis, recurrent AKI, and eGFR slope require assessment separately from early AKI and from hemodynamic improvement after valve replacement.

3.3. Mechanisms Beyond Contrast

Baseline nephron loss, low forward flow, and venous congestion reduce tolerance of rapid pacing, anesthesia-related hypotension, and residual regurgitation (Figure 1). These mechanisms remain active during a contrast-free procedure. Effective perfusion and appropriate volume management are, therefore, integral to renal care.

Bleeding or vascular injury may cause hypotension, transfusion, prolonged instrumentation, and additional angiography. Their observational associations with AKI are clinically relevant, but transfusion may also mark the severity of the underlying complication [14,15].

Aortic manipulation may produce atheromatous or calcific embolization; inflammation and oxidative stress provide further plausible pathways. Associations with aortic atherosclerosis or biomarkers support susceptibility assessment, without identifying a specific benefit from contrast avoidance [16,17].

Contrast is measurable and modifiable, but higher use may partly follow a complication that itself causes AKI. This reverse causation, together with confounding by anatomy and procedural difficulty, limits inference from contrast-volume associations.

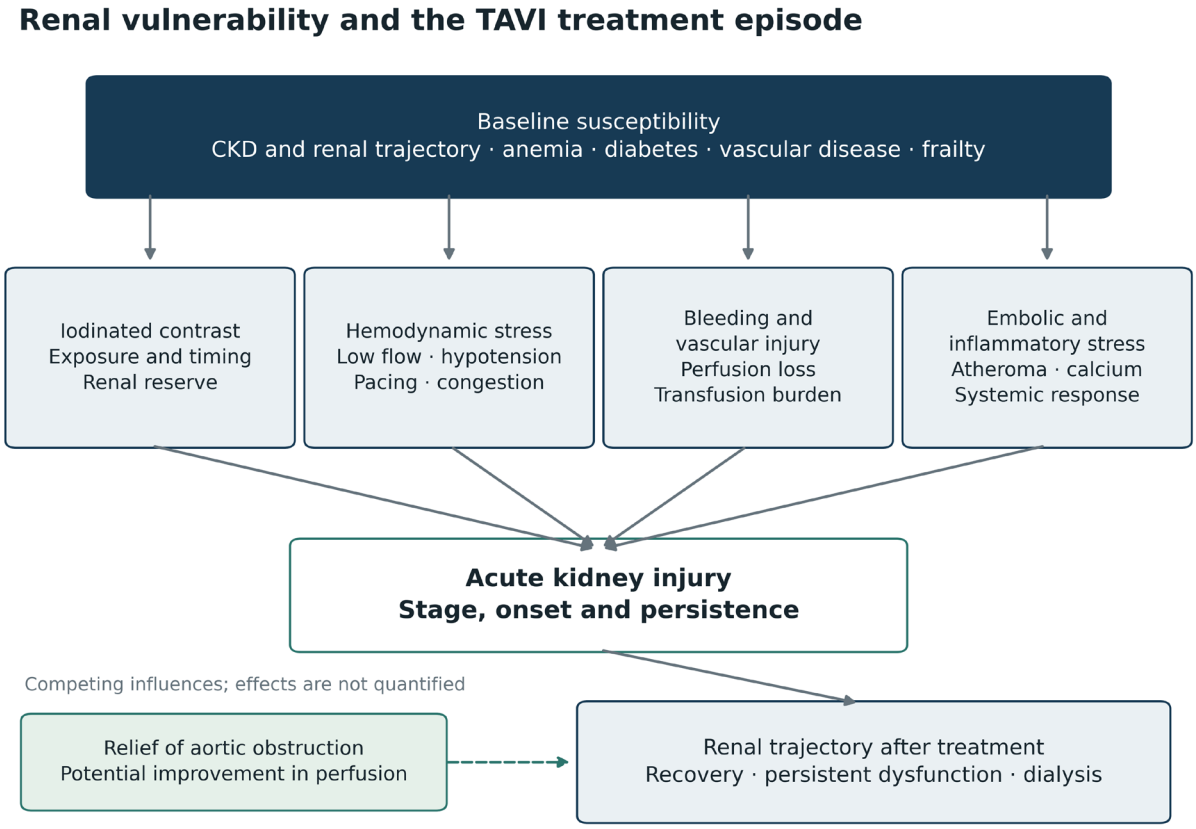


Figure 1. Mechanisms and competing influences on renal outcomes after TAVI. The diagram organizes baseline susceptibility and potential procedural renal insults. The arrows indicate plausible relationships rather than quantified causal effects; the relative contribution varies between patients. Relief of aortic stenosis may improve cardiorenal physiology, while bleeding, sustained hypoperfusion, embolization, inflammation and contrast exposure can contribute to injury. AKI and subsequent recovery should be assessed separately [3,4,14–17]. AKI, acute kidney injury; CKD, chronic kidney disease; TAVI, transcatheter aortic valve implantation.

4. Personalizing Renal Risk Assessment

4.1. Establishing a Credible Baseline

Establish the usual renal trajectory, current creatinine, recent contrast exposure, and whether active AKI is present. A sample during decompensated heart failure may not represent stable function. CKD requires abnormal kidney structure or function for at least three months; one low eGFR does not establish chronicity [18].

State the filtration equation and recognize uncertainty from sarcopenia, body size, and changing creatinine. Cystatin C or a combined estimate can help when it would alter care, but steady-state eGFR equations are unreliable during active AKI [19].

Indexed eGFR, unindexed filtration, and Cockcroft–Gault creatinine clearance are not interchangeable. Retain the source study's measure and units when comparing renal strata or reproducing a contrast index.

In active AKI, evaluate reversibility and treatment urgency. Elective reassessment may be appropriate after correcting a reversible cause; low-output stenosis or recurrent pulmonary edema may instead make prompt valve treatment necessary.

4.2. Clinical Modifiers and Competing Vulnerability

Diabetes, anemia, heart failure, vascular disease, and frailty should trigger specific assessment: identify treatable anemia, define a safe access route, evaluate congestion, and consider whether low muscle mass overestimates filtration. A generic high-risk label does not specify which exposure is modifiable.

STS/ACC TVT and UK registry analyses associate baseline kidney dysfunction and procedural complications with dialysis and death. Their absolute risks depend on treatment era and case mix and should not be presented as precise predictions for every contemporary patient [20,21].

Dialysis-dependent patients require assessment of residual function, volume tolerance, vascular access, and competing prognosis; new-dialysis endpoints and eGFR-derived contrast ratios are inapplicable when filtration is negligible. Transplant recipients also need individualized graft and medication assessment rather than assumed equivalence with native-kidney CKD [22].

4.3. What Risk Scores Can and Cannot Provide

The six-variable bedside TAVR AKI score had moderate discrimination (c-statistic: 0.713), but its calibration and transportability require assessment in the intended setting. It predicts baseline susceptibility rather than benefit from a contrast-sparing strategy [23].

TRITAVI studied dialysis prediction in 10,071 patients at 19 centers. Its preprocedural model is relevant to planning; the postprocedural model additionally incorporates contrast, transfusion, and vascular complications and cannot be used as though these future events were known. Random derivation–validation splitting is also less informative about transportability than independent external validation [24].

Mehran models were developed for PCI. TAVI evaluations of imported and contemporary scores support checking calibration, discrimination, and clinical utility in the target population rather than assuming validity from the original coronary setting [25–28].

Use risk estimates to guide baseline testing, nephrology input, imaging, and follow-up rather than to exclude intervention or prescribe a fixed contrast allowance. Table 2 links susceptibility to the decisions it should change.

Table 2. Renal risk features and the decisions they should inform.

Feature	Why It Changes Assessment	Action or Unresolved Question	Evidence Context
Stable advanced CKD	Limited reserve; greater consequences of additional injury.	Verify chronicity, current trend and alternative imaging adequacy; consider nephrology input.	Registry prognosis; individualized planning [18,20].
Active AKI or rapidly changing creatinine	Steady-state eGFR is unreliable; deterioration may be reversible or driven by stenosis.	Identify cause and urgency; reassess if elective, or expedite necessary valve treatment with targeted imaging.	Clinical assessment; no validated eGFR-derived dose target in active AKI.
Congestion/low output	Renal perfusion depends on forward flow and venous pressure.	Assess volume and hemodynamics; avoid indiscriminate hydration or diuretic withdrawal.	Kidney improvement after TAVI occurs in selected cohorts [3,4].
Anemia/bleeding susceptibility	May reflect reduced reserve, occult disease or risk of transfusion.	Identify treatable causes and plan access/hemostasis; distinguish bleeding from transfusion effects.	Observational association with AKI and dialysis [14,24].
Diabetes and vascular disease	Higher baseline renal vulnerability; more complex coronary/access disease.	Integrate coronary indications and access imaging rather than assigning risk from eGFR alone.	Included in renal prediction models [24,26].

Frailty or low muscle mass	Creatinine-based eGFR may overestimate actual filtration; treatment burden and recovery differ.	Consider cystatin C when a better estimate would change care; incorporate patient priorities.	Filtration estimation and CKD guidance [18,19].
Recent CTA/angiography/PCI	Repeated exposure and evolving renal function may be overlooked.	Retrieve outside records; document dose, concentration, date and intervening kidney trajectory.	Timing depends on clearance and urgency [29].
Hostile access/non-femoral route	Increased procedural complexity, bleeding and embolic potential.	Resolve lumen and route before delivery; select a reliable closure/rescue strategy.	Risk model and access evidence [23,30].
Dialysis dependence	New-dialysis endpoints and eGFR ratios are inapplicable.	Prioritize volume tolerance, residual function and vascular access; coordinate clinically indicated dialysis.	No prophylactic dialysis solely for contrast removal [31].
Kidney transplant	Graft vulnerability and comorbidity may differ from native CKD.	Assess graft trajectory, concomitant treatment and transplant-specific issues.	Multicenter observational evidence [22].

Actions describe an evidence-informed clinical framework, not a validated score. Model discrimination does not establish treatment benefit from contrast avoidance. AKI, acute kidney injury; CKD, chronic kidney disease; CTA, computed tomography angiography; eGFR, estimated glomerular filtration rate; PCI, percutaneous coronary intervention; TAVI, transcatheter aortic valve implantation.

4.4. Interpreting Risk in the Presence of Competing Outcomes

Communicate calibrated absolute risks with their time horizons, separating death, dialysis, and less severe AKI. A high predicted renal risk does not establish that the patient will benefit from contrast avoidance or outweigh the expected benefit of treating severe stenosis.

SWEDEHEART associated AKI persisting at discharge with higher adjusted long-term mortality (HR: 2.04; 95% CI: 1.49–2.81). The analysis excluded baseline eGFR < 15 mL/min/1.73 m^2 or dialysis and included patients surviving TAVI. It establishes a prognostic association, not the mortality effect of lowering contrast [32].

5. Measuring Contrast Exposure Across the Treatment Pathway

5.1. From a Procedural Volume to a Longitudinal Record

Report contrast product, concentration, undiluted-equivalent volume, and timing. Iodine mass equals volume multiplied by iodine concentration; dilution can increase injected volume without increasing iodine mass.

Record CTA, coronary angiography/PCI, TAVI, and contrast-based complication investigations separately, including outside-hospital exposure. Their sum describes treatment burden but is not a validated renal dose because route, clearance, and clinical state differ between episodes. ESUR waiting-time guidance relates clearance to renal function rather than defining one optimal CT-to-TAVI interval [29].

5.2. Renal Function Adjusted Contrast Indices

Among 7112 Michigan registry procedures, AKI occurred in 8.8%; contrast-volume/eGFR indices >2 and >3 were associated with adjusted odds ratios of 1.36 and 1.38, respectively [33]. Other cohorts found less consistent relationships for absolute or cumulative exposure [34–36]. These discrepancies may reflect different exposure windows, case mix, and adjustment for procedural complexity.

The registry association is useful for planning but does not define a safe biological threshold. General anesthesia and complications also track with AKI, and a contrast-volume association cannot separate planned exposure from iodine required to investigate an evolving complication.

The contrast-volume/eGFR index combines mL with an indexed filtration estimate and is not dimensionless. For example, 40 mL/eGFR 25 yields a numerical index of 1.6, not a validated safe allowance. Concentration, active AKI, perfusion, and diagnostic need remain relevant.

5.3. Planning a Contrast Budget

Define a contrast budget by the intended acquisitions and the clinical question each must answer. Treat it as a planning target, with explicit exceptions for coronary compromise, vascular injury, or uncertain residual regurgitation.

Terms such as low, minimal, and ultra-low contrast are study-specific. Report the actual target and care stage rather than assuming a shared definition; use Table 1 for the exposure classification.

6. Preprocedural CT as an Opportunity for Contrast Reduction

6.1. Defining the Information That Must Be Preserved

CTA remains the reference for integrated annular/root measurements, coronary relationships, calcium distribution, aortic anatomy, and access lumen. SCCT consensus recommendations treat acquisition and reconstruction as a single planning task. The relevant adequacy criterion is a reliable device, access, and deployment decision [37,38].

Appropriate cardiac phase and multiplanar alignment are essential. Interpret annular area or perimeter with outflow-tract calcium, coronary height, sinus dimensions, and the sinotubular junction; image noise or an oblique boundary can make an apparently precise measurement unreliable.

Access assessment must resolve the narrowest lumen, calcium, tortuosity, previous stents or grafts, and femoral puncture anatomy. Adequate annular images cannot compensate for an uncertain delivery route, or vice versa.

A recent observational pre-TAVI CT cohort found predominantly transient renal changes and no independent adjusted association between contrast volume and AKI. Without an unexposed comparator, it cannot establish either contrast causation or absence of risk in unstable advanced CKD [39].

6.2. Tailoring Acquisition and Injection

Tailor iodine delivery to scanner coverage, tube voltage, body size, and circulation time. Lower voltage increases iodine attenuation but can also increase noise; injection rate, saline displacement, bolus timing, and cardiac/peripheral acquisition design determine whether reduced iodine remains diagnostic [40,41].

Body-size-adapted and third-generation dual-source protocols achieved comprehensive planning with reduced volumes, including 38 and 40 mL acquisitions. Their scanner and injection conditions limit direct transfer as fixed-volume prescriptions [42–44].

Pulerwitz and colleagues evaluated 20 mL CTA in 26 patients with eGFR < 30 mL/min alongside 26 standard-volume studies; vascular imaging was diagnostic in 96% of the very-low-volume group. This supports retaining CTA in selected advanced CKD when a locally validated protocol can answer the necessary questions [45].

Suchá and colleagues found high measurement reliability but more valve size and access eligibility disagreement with reduced-volume CTA. A high intraclass correlation does not exclude consequential disagreement near a device-size or access threshold [46].

When readers disagree on a treatment-changing measurement, review reconstructions and direct any additional test at the unresolved question. Reader confidence or average vascular attenuation should not substitute for a secure decision.

Earlier 20 mL and low-volume acquisitions, and the prospective SPECTACULAR study in 116 candidates, extend the technical evidence across different designs [47–49]. They support acquisition feasibility rather than comparative renal effectiveness.

6.3. Spectral Imaging and Reconstruction

In renal impairment, a 30 mL dual-energy protocol improved annular measurement reliability with 50 keV reconstruction compared with conventional images [50]. Low-energy reconstructions can enhance iodine conspicuity, but biochemical follow-up and renal-event power remain limited.

A 2026 photon-counting comparison of 162 matched examinations supported preserved imaging quality with 50 versus 80 mL [51]. Annular assessment and virtual noncontrast calcium-scoring studies address additional technical questions, not kidney failure prevention [52,53].

Iterative and deep learning reconstruction can improve image quality [54–56]. Validation should, nevertheless, include decision agreement, nondiagnostic examinations, and performance across scanners, body habitus, and calcium burden.

Count repeat acquisitions and alternative tests when auditing a low-volume protocol. Radiation, iodine, and eventual diagnostic adequacy must be evaluated across the attempted examination, including failures [57].

6.4. Reporting Diagnostic Adequacy

Report adequacy separately for annular sizing, coronary relationships, and the intended access route. Describe how each limitation could change treatment rather than assigning only an overall "diagnostic" label.

Report categorical valve-size agreement and access eligibility alongside continuous measurement reproducibility, particularly around device thresholds.

Include rescans, additional iodine, and redirection to CMR or TEE in the denominator. Excluding failures overstates the efficiency of a low-volume protocol.

7. Alternatives and Complements to Contrast-Enhanced CT

7.1. Noncontrast CT

Noncontrast CT shows calcium, the aortic course, prosthetic material, and vascular calcification. It may not define annular blood–tissue boundaries, coronary relationships, or the patent access lumen reliably; an external calcific vessel diameter is not the internal lumen.

Elison and colleagues developed balloon-expandable valve sizing from sinus and sinotubular junction dimensions in 100 patients and validated it in 60, followed by 35 noncontrast implants. The approach uses a device-specific surrogate rather than direct conventional annular planimetry [58].

Complementary imaging must resolve coronary risk and access suitability. A noncontrast sizing surrogate should not be extended to another device or excluded anatomy without validation.

7.2. Cardiovascular Magnetic Resonance

Noncontrast CMR can measure the root and annulus without iodine or radiation. Whole-heart and angiographic sequences provide complementary information, while calcium distribution remains better assessed with CT [59–61].

In 26 patients, whole-heart CMR with quiescent-interval single-shot access angiography showed good agreement with CTA for access decisions despite differences in

vessel measurements. Borderline delivery system dimensions still require resolution of disagreement [62].

In TAVR-CMR, 380 patients were randomized, and 267 underwent TAVR and entered the modified intention-to-treat analysis. Implantation success was 93.5% with CMR and 90.7% with CT, meeting noninferiority. Stage 4–5 CKD was excluded, and the primary endpoint was implantation success. The trial supports alternative planning in its population, not renal benefit in advanced CKD [63].

CMR may be limited by orthopnea, hemodynamic instability, motion, claustrophobia, hardware, and availability. Specify how complementary noncontrast CT or vascular imaging will resolve information that CMR does not provide.

7.3. Three-Dimensional Echocardiography

Three-dimensional TEE can measure the annulus and assess deployment, ventricular function, and regurgitation. Calcium shadowing, reconstruction, operator experience, and the burden of probe placement or anesthesia affect its usefulness; the complete vascular route requires separate assessment.

Hana and colleagues evaluated 54 renal-dysfunction patients using TEE without prior cardiac CTA against a larger CTA cohort. Procedural contrast was lower, but nonrandom allocation and different baseline renal function limit comparative inference [64].

Interpret TEE measurements with device-specific sizing guidance. Resolve discordance by image quality and reconstruction rather than averaging incompatible measurements. The feasibility of TTE-guided implantation depends on acoustic windows and procedural complexity.

A later TEE/noncontrast-CT comparison reported similar paravalvular leak and pacemaker outcomes to CTA-based sizing; it did not establish a comparative renal endpoint [65].

7.4. Vascular Ultrasound, Carbon Dioxide, and Selective Intravascular Imaging

Duplex ultrasound can guide puncture and assess accessible femoral lumen and postclosure flow. Deep pelvic vessels, obesity, and calcium limit its ability to exclude disease along the entire delivery route.

Arterial CO_2 angiography must not be used above the diaphragm or as a substitute for coronary or aortic-root angiography because of cerebral or coronary gas embolization risk. Its selected below-diaphragm vascular role requires dedicated equipment, technique, and specialist assessment of cardiopulmonary disease and shunting [66].

Multimodality planning without iodine has been evaluated in small clinical cohorts, including Prado's ten-patient series [67].

IVUS provides intraluminal measurements in selected access or valve-in-valve applications. Small human reports and preclinical studies establish specialized technical possibilities, with insufficient evidence for routine replacement of conventional planning [68–70].

7.5. Gadolinium and Contrast Substitution

Group II gadolinium agents have a very low nephrogenic systemic fibrosis risk when appropriately used for MRI [71]. This does not establish the safety or diagnostic equivalence of intra-arterial gadolinium as an angiographic substitute. Dose, route, and image quality require separate evaluation; noncontrast CMR avoids substitution when adequate.

Choose the imaging combination that resolves the anatomy with acceptable total burden, including appointments, anesthesia, and treatment delay. Table 3 and Figure 2 summarize the information and limitations of each modality.

Table 3. Imaging options for reducing iodine while preserving TAVI planning information.

Modality	Information Retained	Principal Limitation	Selected Evidence
Optimized low-contrast CTA	Annulus/root, coronary relationships, calcium distribution and access lumen in one pathway.	Scanner/body size dependence; low enhancement can cause decision disagreement or rescanning.	Pulerwitz: 20 mL in 26 advanced-CKD patients; 96% diagnostic vascular imaging [45].
Spectral/dual-energy CTA	Low-energy reconstruction improves iodine conspicuity; root and access assessment.	Image quality benefit is not evidence of AKI prevention; energy/noise trade-offs vary.	SPECTACULAR: 116 candidates; Chang: 30 mL protocol [49,50].
Photon-counting CTA	High-resolution/spectral assessment with lower-iodine protocols.	Availability, protocol validation and outcome evidence remain limited.	Layer: 162 examinations; 50 versus 80 mL; imaging performance study [51].
Noncontrast CT	Calcium, aortic course, prosthetic material and vessel calcification; selected surrogate sizing.	Uncertain blood–tissue boundaries and access lumen; incomplete coronary characterization.	Elison: device-specific surrogate sizing algorithm and no-contrast series [58].
Noncontrast CMR	Root/annular measurements; access lumen with suitable angiographic sequences.	Limited calcium depiction; motion, tolerance and expertise; may require CT complement.	Randomized planning trial excluded CKD stages 4–5; implantation endpoint, not renal benefit [62,63].
Three-dimensional TEE	Annular measurements, ventricular function, deployment and regurgitation assessment.	Acoustic shadowing, measurement dependence, and probe/sedation burden; incomplete access assessment.	Hana: selected nonrandom comparison; later TEE/noncontrast-CT cohorts [64,65].
Duplex ultrasound	Common femoral lumen, puncture targeting and postclosure flow.	Pelvic depth, obesity and calcification can limit complete route evaluation.	Access outcome evidence supports careful puncture; not a full CTA substitute [30].
CO_2 angiography	Selected aortoiliac/peripheral luminal assessment without iodine.	Dedicated technique; no coronary or aortic root use; arterial use above diaphragm is unsafe.	Used in selected noncontrast pathways [66,72,73].
IVUS	Selected intraluminal access measurements and device–coronary relationships.	Invasive catheter manipulation; specialized interpretation; limited clinical validation.	Selected human reports and a porcine feasibility study [68–70].

Modality choice follows the unresolved anatomical question and local diagnostic reliability. Published injection volumes are study descriptions, not general dosing instructions. AKI, acute kidney injury; CKD, chronic kidney disease; CMR, cardiovascular magnetic resonance; CO_2, carbon dioxide; CT, computed tomography; CTA, CT angiography; IVUS, intravascular ultrasound; TAVI, transcatheter aortic valve implantation; TEE, transesophageal echocardiography.

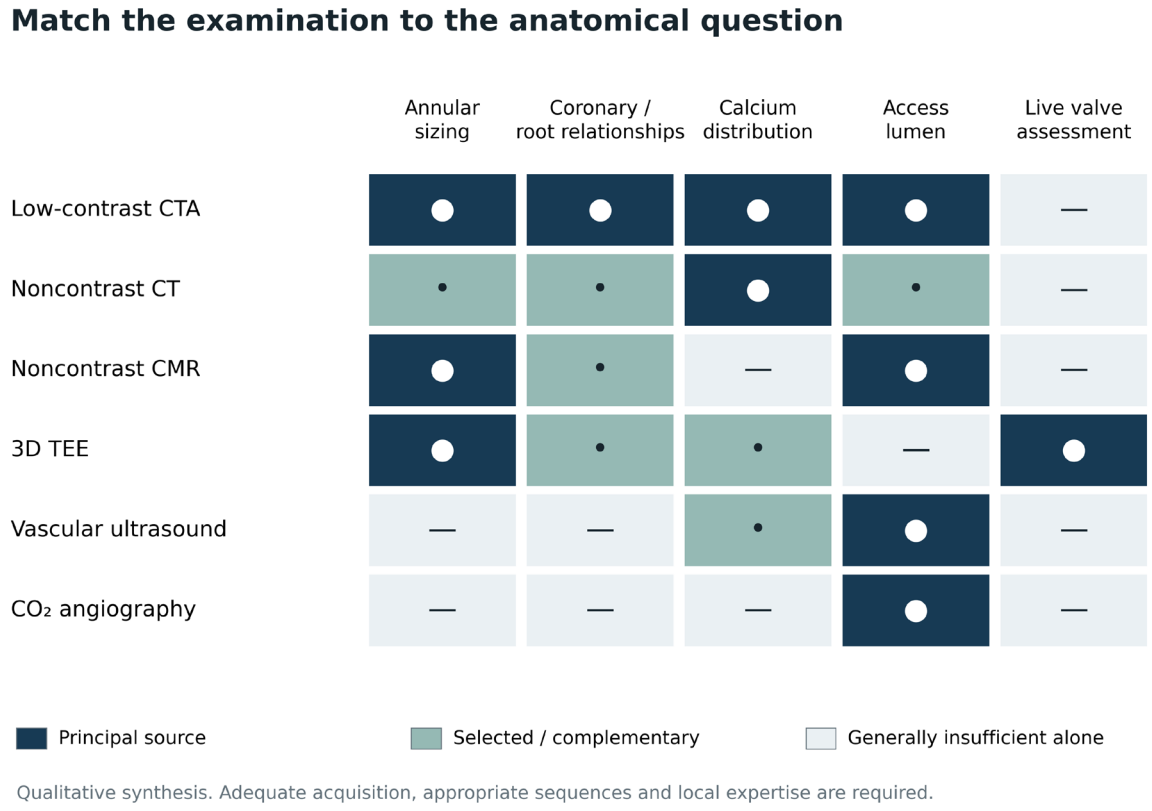


Figure 2. Anatomical information available from contrast-sparing imaging modalities. Qualitative mapping of information generally obtainable with an adequate examination. Dark cells indicate a principal source of the specified information; intermediate cells indicate a selected or complementary role; pale cells indicate that the modality is generally insufficient alone. The matrix is not a diagnostic-accuracy score or a recommendation that one examination replace another. CMR access assessment requires suitable angiographic sequences; ultrasound primarily evaluates accessible vascular segments; CO_2 angiography is restricted to appropriate below-diaphragm arterial applications. Noncontrast CT annular sizing may use device-specific surrogate approaches rather than direct conventional planimetry [37,58,62–64,66]. CMR, cardiovascular magnetic resonance; CO_2, carbon dioxide; CT, computed tomography; CTA, CT angiography; TEE, transesophageal echocardiography.

8. Coronary Assessment and Revascularization

8.1. Avoiding Unnecessary Invasive Examinations

Review existing coronary studies, symptoms, revascularization history, and coronary interpretability on TAVI CTA before scheduling invasive angiography. In selected patients, adequate-quality CTA may permit avoidance of a separate diagnostic examination. Severe calcium, stents, arrhythmia, or motion can make this inappropriate.

Coronary CTA performance depends on disease prevalence, scanner quality, and the lesion definition. A negative interpretable segment and a nondiagnostic segment are not equivalent; avoiding redundant testing requires resolving clinically important uncertainty [74].

8.2. What Randomized PCI Trials Establish

ACTIVATION randomized 235 patients with significant coronary disease and limited angina. Numerically similar death/rehospitalization did not meet the prespecified noninferiority criterion, and PCI increased bleeding [75].

NOTION-3 randomized 455 patients with an FFR ≤ 0.80 lesion or ≥90% diameter stenosis. PCI reduced death, myocardial infarction, or urgent revascularization at median two-year follow-up (26.0% versus 36.0%; HR: 0.71; 95% CI: 0.51–0.99), with more bleeding [76].

PRO-TAVI randomized 466 patients with visually estimated 70–99% stenosis, or 40–70% stenosis with positive invasive physiology. PCI deferral met noninferiority for death, myocardial infarction, stroke, or major bleeding at one year (24.0% versus 26.0%; risk difference of −1.7 percentage points; 95% CI: −9.5 to 6.2; margin: 11 percentage points). Its lesion criteria, endpoint including bleeding, follow-up, and noninferiority design differ from those of NOTION-3 [77].

These trials address different coronary management questions, not contrasting renal strategies. Select PCI according to coronary indication and future access, then minimize avoidable exposure; neither establishes a renal benefit from routine revascularization or routine deferral. Table 4 makes their populations and endpoints explicit.

8.3. Staged Versus Concomitant Procedures

Staging PCI may permit renal reassessment but adds another encounter, vascular puncture, and potentially repeat angiography. Concomitant treatment consolidates care but can increase procedural duration and hemodynamic burden. Consider total exposure, coronary complexity, urgency, and physiological tolerance.

REVASC-TAVI associated post-TAVI PCI with better adjusted outcomes, whereas national and single-center matched studies evaluated staged versus concomitant strategies [78–80]. Treatment selection, coronary stability, era, and survival to deferred PCI limit causal interpretation; none establishes universal renal superiority for a timing strategy.

8.4. Contrast-Sparing PCI When Revascularization Is Indicated

MOZART randomized IVUS-guided versus angiography-guided PCI and demonstrated substantial contrast reduction, without sufficient power for important kidney outcomes [81].

Imaging- and physiology-guided PCI without procedural contrast was subsequently reported in 31 advanced-CKD patients after minimal-contrast diagnostic angiography [82]. Such techniques require a reliable roadmap, expertise, and predefined indications for contrast to exclude complications.

9. Periprocedural Measures That Accompany Contrast Reduction

9.1. Hydration According to Physiology

Hydration should reflect perfusion and congestion rather than eGFR alone. Severe stenosis, right ventricular dysfunction, pulmonary hypertension, and recent pulmonary edema may make fixed regimens hazardous. Adjust indicated contrast prophylaxis to clinical volume tolerance [6,31].

POSEIDON reduced creatinine-defined injury with filling-pressure-guided hydration during cardiac catheterization but did not validate that algorithm in severe stenosis or TAVI [83]. AMACING supported omission of prophylactic hydration in a selected elective population with an eGFR of 30–59 mL/min/1.73 m^2; it excluded eGFR < 30 and cannot justify omission in advanced CKD or unstable TAVI [84].

A 74-patient pre-TAVI CTA trial found a short bicarbonate regimen noninferior to prolonged saline for percentage creatinine change. It did not establish superiority of bicarbonate or equivalence for uncommon hard renal events [85].

9.2. Why Forced Diuresis Requires Caution

The favorable AKI result in the 112-patient, single-center PROTECT-TAVI trial (5.4% versus 25.0%) was not confirmed by the sham-controlled REDUCE-AKI trial or a later 100-patient CKD trial. REDUCE-AKI stopped early for futility and possible harm. Differences in design, population, and cointerventions limit direct comparison, but later evidence does not support routine forced diuresis solely for TAVI-associated AKI prevention [86–88].

This conclusion concerns prophylactic forced diuresis, not therapeutic decongestion when clinically indicated. Table 4 distinguishes those preventive interventions from ordinary volume management.

Table 4. Randomized preventive and management trials relevant to renal planning.

Trial/Population	Comparison and Size	Principal Result	Implication for TAVI
POSEIDON; cardiac catheterization [83]	LVEDP-guided versus standard hydration; 396 randomized.	Creatinine-defined CA-AKI: 6.7% versus 16.3% among evaluable patients.	Supports physiological fluid tailoring; algorithm not directly validated in severe AS/TAVI.
AMACING; elective contrast [84]	No prophylactic hydration versus hydration; eGFR 30–59 population.	Noninferiority met for trial-defined creatinine-based AKI.	Does not support omission in eGFR < 30, active AKI or unstable TAVI patients.
PRESERVE; high-risk angiography [89]	Bicarbonate versus saline; acetylcysteine versus placebo; 5177 randomized and 4993 analyzed.	No reduction in death, dialysis/persistent dysfunction, or contrast-associated AKI.	No routine acetylcysteine or preferential bicarbonate solely for kidney protection.
Pre-TAVI CTA hydration [85]	Short bicarbonate versus prolonged saline; 74 patients.	Noninferiority for percentage creatinine change; no observed dialysis or CA-AKI.	Small imaging trial; not proof of bicarbonate superiority or hard-outcome equivalence.
PROTECT-TAVI; valve implantation [86]	Matched hydration/forced diuresis versus saline; 112.	AKI 5.4% versus 25.0%.	Small favorable trial; must be interpreted with later contradictory studies.
REDUCE-AKI; valve implantation [87]	Active matched hydration/diuresis versus sham; 136.	AKI 25.0% versus 19.1%, nonsignificant; stopped for futility/possible harm.	Does not support routine forced diuresis for TAVI prophylaxis.
Voigtländer-Buschmann; CKD/TAVI [88]	RenalGuard versus standard hydration; 100 randomized.	AKI 21.3% versus 15.7%, nonsignificant.	Later randomized evidence did not confirm benefit.
SOLVE-TAVI; transfemoral implantation [90]	Conscious sedation versus general anesthesia; 447 in factorial trial.	AKI: 9.0% versus 9.2%; primary composite equivalent within trial margin.	Sedation is feasible; anesthesia choice alone has no established renal superiority.
DapaTAVI; high-risk post-TAVI patients [91]	Dapagliflozin versus standard care; 1,257 randomized, 1,222 in primary analysis.	Death or worsening HF: 15.0% versus 20.1%; HR: 0.72.	Supports selected post-TAVI HF care; not immediate contrast-AKI prophylaxis.
ACTIVATION; stable CAD/TAVI [75]	PCI versus no PCI; 235 randomized; limited angina.	Death/rehospitalization numerically similar; noninferiority not met; more bleeding with PCI.	Does not establish routine PCI benefit or renal protection from deferral.

NOTION-3; CAD/TAVI [76]	PCI versus conservative care; 455; FFR ≤ 0.80 or ≥90% stenosis.	Death/MI/urgent revascularization: 26.0% versus 36.0%; HR: 0.71 (95% CI: 0.51–0.99); median: two years.	Ischemic benefit with more bleeding in the selected population; not a renal strategy trial.
PRO-TAVI; CAD/TAVI [77]	PCI deferral versus pre-TAVI PCI; 466; 70–99% stenosis or 40–70% with positive physiology.	Death/MI/stroke/major bleeding: 24.0% versus 26.0% at one year; noninferiority margin: 11 percentage points.	Different lesions, endpoint, horizon, and hypothesis from NOTION-3; no established renal advantage.

Trials differ in intervention, endpoint and population. Coronary/imaging evidence is not automatically transferable to TAVI. Trial doses are not reproduced as general prescribing instructions. PROTECT-TAVI here refers to the renal trial, not the later cerebral-embolic-protection trial with a similar acronym. AKI, acute kidney injury; AS, aortic stenosis; CI-AKI, contrast-induced AKI, as named in the original study; CKD, chronic kidney disease; CTA, computed tomography angiography; eGFR, estimated glomerular filtration rate; HF, heart failure; HR, hazard ratio; LVEDP, left ventricular end-diastolic pressure; TAVI, transcatheter aortic valve implantation.

9.3. Medication Review and Pharmacological Prevention

In PRESERVE, neither bicarbonate rather than saline nor acetylcysteine rather than placebo improved death, dialysis, persistent renal dysfunction, or CA-AKI after angiography [89]. These treatments should not substitute for appropriate fluid and procedural planning.

Review nephrotoxins and medications affecting perfusion or volume. Renin–angiotensin system inhibitors and diuretics require individualized decisions in hypotension, congestion, or AKI. Metformin is not nephrotoxic, but interruption may be indicated when impaired clearance increases lactic acidosis risk; follow renal and contrast guidance [18,31].

SGLT2 inhibitors may be indicated for CKD or heart failure, with perioperative management for fasting and acute illness. DapaTAVI supports selected post-TAVI heart failure care, not immediate contrast prophylaxis; observational AKI findings remain subject to treatment selection bias [91,92].

Do not initiate dialysis solely to remove contrast. Use standard clinical RRT indications and coordinate fluid, electrolyte, and residual-function needs in patients already receiving dialysis [31].

10. Procedural Planning and Execution

10.1. Translating Imaging into a Usable Plan

Agree on access, valve, coronary and annular risks, fluoroscopic projections, anesthesia, pacing, and final valve assessment before implantation. Specify which angiograms are redundant and which uncertainties require confirmation.

Verify CT-derived projections against actual patient rotation, respiration, device position, and catheter location. Landmark mismatch should prompt reassessment or targeted imaging.

Describe platform-specific projections precisely, including the isolated cusp, catheter position, and device markers. An “overlap view” is not interchangeable across valve platforms.

10.2. Access and Hemostasis

Ultrasound-guided puncture identifies the common femoral artery, anterior-wall calcium, bifurcation, and adjacent vein. The propensity-matched PULSE analysis associated ultrasound guidance with fewer vascular or bleeding events, without establishing a specific renal treatment effect [30].

Select closure or surgical exposure according to vessel anatomy and local expertise. A single-center matched access comparison informed vascular and bleeding management but did not directly evaluate a renal prevention strategy [93].

Selective completion angiography requires reassuring clinical and, when appropriate, duplex findings. A 2026 observational comparison supported feasibility of selective surveillance but did not establish an adjusted AKI benefit [94].

Unexplained hypotension, hemoglobin decline, expanding hematoma, pain, pulse loss, or difficult device withdrawal should prompt immediate assessment and definitive imaging when needed.

Report clinically consequential bleeding and vascular injury using contemporary definitions alongside kidney outcomes; reducing angiography cannot justify less reliable complication surveillance [95].

10.3. Anesthesia, Pacing, and Hemodynamic Stability

Observational sedation comparisons favor less invasive practice in selected patients, but SOLVE-TAVI found similar AKI with conscious sedation and general anesthesia (9.0% versus 9.2%) [90,96].

Choose anesthesia for the complete procedure, including the need for TEE, cooperation, airway management, and anticipated rescue. Maintain conversion capability.

Minimize avoidable repeat pacing and sustained hypotension, with recovery between episodes. No universal pacing duration threshold has been validated for renal protection.

Predilatation and postdilatation should follow anatomy and device behavior rather than a requirement for a contrast-sparing label; direct implantation is appropriate only when mechanically suitable [97].

10.4. Fluoroscopic Landmarks and Selective Root Angiography

Known cusp catheters, calcium, surgical frames, wires, and device markers can provide fluoroscopic references. Early TEE-guided, single-injection, and fusion reports demonstrate these technical approaches [98–100].

Reconfirm pigtail cusp position after delivery system or ventricular-wire movement. A displaced catheter or incorrect projection can create a false annular reference.

Use each acquisition to resolve a defined uncertainty. Required volume depends on catheter position, iodine concentration, circulation, and injection technique; poor confirmation warrants further imaging.

10.5. Assessing the Implanted Valve

Integrate device position, ventricular–aortic hemodynamics, echocardiographic gradients, regurgitation, and pericardial assessment. TTE or TEE can reduce angiography only when the examination is adequate [101].

Acute regurgitation or instability requires prompt differentiation of malposition, incomplete expansion, leaflet dysfunction, paravalvular leak, ischemia, and tamponade, using angiography when noncontrast assessment leaves consequential uncertainty.

LANDMARK echocardiographic and videodensitometric substudies evaluate complementary valve performance methods. A method validated using a specified final aortogram cannot be assumed to retain its performance when that acquisition is substantially reduced or omitted [102,103].

10.6. Image Fusion and Technological Assistance

Echo–fluoroscopy or CT–fluoroscopy fusion can display soft-tissue relationships and planned landmarks. Registration error, motion, and changing geometry require verification against native images and physiology [104].

A 2026 proof-of-concept CT-fusion study reduced root angiograms without a significant contrast-volume reduction. Fewer acquisitions, therefore, do not necessarily establish lower iodine exposure, and renal benefit was not demonstrated [105].

Table 5 summarizes checkpoints and escalation criteria. A robust protocol should define these triggers prospectively; rescue contrast is part of safe implementation.

Table 5. Procedural checkpoints and triggers for additional imaging.

Checkpoint	Contrast-Sparing Information Source	Finding That Should Prompt Reassessment	Safety Principle
Before arterial access	Reviewed CTA or adequate multimodality route assessment; ultrasound.	Uncertain lumen, severe calcium, graft anatomy or unexpected resistance.	Resolve route before large-bore advancement [37].
Puncture and closure plan	Real-time ultrasound; vessel-specific closure/exposure plan.	Uncertain puncture level, anterior-wall calcium or difficult guidewire passage.	Avoid bleeding and access injury; no obligatory access technique [30,93].
Working projection	CT plan, calcium landmarks, known cusp catheter and device markers.	Landmark mismatch, catheter displacement, parallax or patient rotation.	Verify actual annular reference before deployment.
Deployment confirmation	Secure fluoroscopy and appropriate echocardiography; selective limited injection.	Uncertain depth, coronary clearance, frame position or ventricular wire behavior.	Use contrast when needed to resolve consequential uncertainty.
Balloon intervention/pacing	Anatomy, device behavior and hemodynamic recovery.	Poor expansion, sustained hypotension, repeated unsuccessful attempts.	Avoid unnecessary intervention while treating genuine mechanical problems.
Valve result	TTE/TEE, gradients and hemodynamics.	Significant/uncertain AR, ventricular dysfunction, ischemia or pericardial fluid.	Integrative diagnosis; do not rely on one limited modality [101].
Access completion	Pulses, examination, duplex where appropriate.	Hypotension, bleeding, pain, pulse loss, closure uncertainty or difficult withdrawal.	Prompt angiography or other definitive assessment when indicated [94].
Before discharge	Creatinine/electrolytes, clinical volume status and treatment record.	Rising creatinine, oliguria, congestion, anemia or incomplete follow-up arrangements.	Evaluate cause and assign laboratory/clinical follow-up responsibility.

This checklist is proposed clinical synthesis and has not been prospectively validated. A contrast target is subordinate to diagnosis and treatment of complications. AR, aortic regurgitation; CTA, computed tomography angiography; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.

11. Clinical Evidence for Contrast-Sparing Implantation

11.1. Early Reports and Small Series

Early reports established the technical feasibility of combining noncontrast planning, echocardiography, and fluoroscopic landmarks, including a 12-patient advanced-CKD series [106,107].

Conscious sedation, CMR-based, and multimodality reports extended the available techniques but remain small, selected experiences [108–110].

Bose's eight-patient pilot found no significant one-year eGFR deterioration. The 575-patient Rzucidlo cohort provided longer clinical follow-up but compared baseline renal strata under low-contrast practice rather than assigned contrast strategies [111,112]. Table 6 separates these questions from direct dose-group comparisons.

11.2. Comparative Minimal-Contrast Cohorts

In Choi's 93-patient retrospective cohort, minimal contrast was associated with fewer composite events, but AKI was 16.2% versus 19.6% and dialysis 5.4% versus 12.5%, without a demonstrated renal between-group benefit. The comparator received much more contrast (mean: 147.7 versus 2.6 mL), and recruitment spanned 2011–2020. Composite findings, therefore, cannot isolate a contrast effect from selection and era [113].

Giraldo-Grueso's larger cohort compared ≤20 with >20 mL, with a much lower comparator volume (mean: 33 mL). AKI was 3.0% versus 5.5% (p = 0.146), and dialysis 1.4% versus 0.6% (p = 0.435). These neutral comparisons do not exclude benefit, but do not demonstrate it [114].

Kazzi's 437-patient stage G3b-or-worse cohort associated procedural volume below the numerical eGFR with lower matched AKI (17.9% versus 28.3%; p = 0.0217), without differences in mortality or new dialysis. The reported creatinine definition differs from standard KDIGO wording, and residual confounding remains [115].

In Koga's balloon-expandable series, 12/32 intended zero-contrast cases required iodine (37.5%). The strategy should, therefore, be analyzed by intended allocation with conversion retained, rather than as 32 completed zero-contrast implants [116].

11.3. Prospective and Contrast-Free Pathways

Freire's 25-patient prospective pilot tested noncontrast decisions while retaining blinded contrast CTA and procedural safety checkpoints (mean procedural contrast: 45.0 mL). Device success and 30-day early safety were each 92.0%; AKI occurred in 6/25 patients (24.0%), with no dialysis. It evaluates a decision pathway against contrast-based safety checks, not complete iodine avoidance [72].

Nerla compared 44 zero-contrast patients with 63 standard-practice patients. Dedicated planning and implantation avoided iodine after prior coronary angiography at least one month earlier. In-hospital AKI did not differ; later AKI events favored zero contrast, but operator allocation, cointerventions, and unequal baseline renal function limit causal interpretation [73].

Elison's 35-patient balloon-expandable series achieved 100% technical success, but 2/35 patients required dialysis within one week. This provides a direct illustration that technical success and contrast avoidance do not eliminate renal risk [58].

Papadopoulos's 50-patient prospective MyVal series used CT-based projection planning, a noncoronary-cusp pigtail reference in right–left cusp overlap, and a limited confirmation injection. Median implantation contrast was 8 mL after planning CTA of median 45 mL. One patient developed stage 1 AKI, and none required new RRT within 30 days. Table 6 reports the available six-month follow-up; this uncontrolled series is considered on the same feasibility basis as the other single-arm protocols [117].

The five protocols in Figure 3 were selected purposively to illustrate contrasting exposure patterns: ultra-low procedural iodine after CTA, iodine-free dedicated planning with procedural iodine, retained contrast-based safety checks, and dedicated planning/implantation without iodine with different documentation of earlier coronary exposure. They are examples, not a ranking or an exhaustive study set.

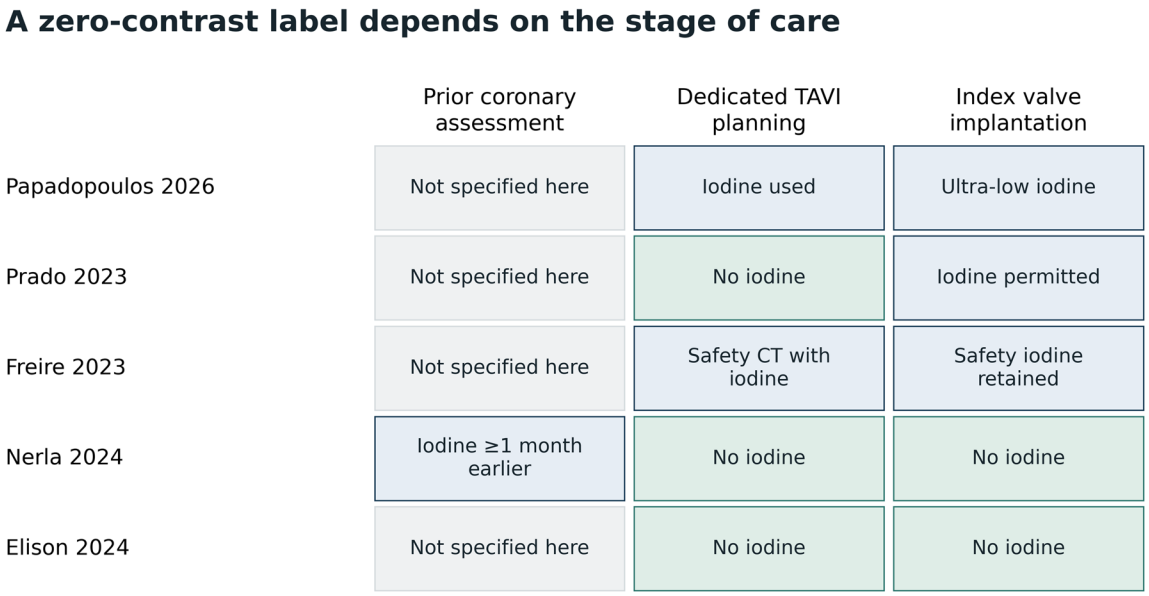


Figure 3. Iodinated contrast exposure by stage of care in five illustrative protocols. Selection was purposive to demonstrate different exposure patterns, not relative effectiveness or methodological superiority. Papadopoulos used planning CTA and ultra-low procedural contrast; Prado avoided iodine during dedicated planning but used it for implantation; Freire retained contrast-based safety CT and procedural checks; Nerla used prior coronary angiography before iodine-free dedicated planning and implantation; Elison used noncontrast CT and no procedural iodine. Gray cells indicate exposure not specified in this comparison and must not be read as zero exposure. These categories do not compare renal outcomes [58,67,72,73,117]. CTA, computed tomography angiography; TAVI, transcatheter aortic valve implantation.

11.4. Synthesizing a Heterogeneous Evidence Base

Comparative results conflict across different baseline risks, comparator doses, anatomical selection, cointerventions, and AKI definitions. In the six-study meta-analysis of 1,505 patients, the primary pooled AKI estimate was inconclusive (risk ratio: 0.66; 95% CI: 0.20–2.17; I^2 = 71.26%). A favorable sensitivity estimate after study exclusion (0.47; 95% CI: 0.24–0.92) should not displace the primary analysis or overcome nonrandom allocation [118].

There is no established randomized renal-outcome comparison of procedural zero contrast versus ultra-low contrast, or of complete iodine-free versus optimized low-contrast pathways. Table 6, therefore, reports population, actual exposure, AKI, dialysis, follow-up, and the limitations of each study separately. Cross-study event rates are not used to rank protocols or valves.

Table 6. Selected clinical studies of contrast-sparing planning and implantation.

Study and Design	Baseline Renal Function	Actual Contrast Exposure	AKI and Ascertainment	New Dialysis or RRT	Follow-Up and Interpretation
Maffeo 2020 [107] 12; single-arm series	Stage ≥ 4 CKD; mean eGFR 23.00 ± 5.69.	Noncontrast CT; 0 mL implantation. Earlier coronary exposure not fully quantified.	0/12 (0.0%); VARC-2 at early assessment.	Separate dialysis outcome NR-A.	Mean eGFR change +1.3 at 48 h; procedural feasibility in a small selected cohort.
Rzucidlo 2021 [112] 575; renal-stratum cohort	GFR ≥ 60, 30–60, <30 mL/min in 51.7%, 42.1%, 6.3%.	Median implantation 23 (15–33), 24 (14–33), 13 (8–20) mL across those strata.	NR-A.	NR-A.	Median 811 days. Lower GFR associated with worse unadjusted survival; no assigned contrast-strategy comparator.
Choi 2021 [113] 37 minimal vs. 56 conventional; retrospective	eGFR ≤ 50 eligibility; mean 32.5 ± 7.2 vs. 38.5 ± 9.2.	Mean implantation 2.6 ± 4.4 vs. 147.7 ± 62.7 mL; minimal group ≤ 10 mL. Prior coronary iodine permitted.	6/37 (16.2%) vs. 11/56 (19.6%); creatinine increase ≥50% or ≥0.3 mg/dL.	2/37 (5.4%) vs. 7/56 (12.5%); p = 0.256.	30 days. Composite events 16.2% vs. 42.9%; mortality 0.0% vs. 16.1%. Selection/era confounding; composite benefit is not established AKI prevention.
Prado 2023 [67] 10; imaging pilot	Mean CrCl 26.85 ± 12.55 mL/min.	0 mL dedicated planning; median implantation 42.5 (35.0–63.7) mL.	Aggregate staged AKI incidence NR; serial creatinine/CrCl reported.	1/10 (10.0%) required dialysis for refractory hypervolemia.	In-hospital. Mean discharge CrCl 29.84 ± 13.98 mL/min; no deaths. Iodine-free planning, not an iodine-free procedure.
Freire 2023 [72] 25; prospective pilot	CKD ≥ G3a; median eGFR 49 (44–56).	Noncontrast decisions with retained safety CTA; procedural safety contrast mean 45.0 ± 35.2 mL.	6/25 (24.0%): 5 stage 1, 1 stage 2; VARC-2.	0/25 (0.0%).	30 days. Device success and early safety each 92.0%; no death/stroke. Safety-check iodine precludes complete zero-exposure classification.
Giraldo-Grueso 2023 [114] 429 ≤ 20 vs. 165 > 20 mL; retrospective	Mean creatinine 1.24 ± 0.4 vs. 1.13 ± 0.3 mg/dL; more advanced CKD in low-dose group.	Mean implantation 8.5 ± 6 vs. 33 ± 16 mL; planning exposure separate.	13/429 (3.0%) vs. 9/165 (5.5%); p = 0.146; study creatinine-based VARC-3 definition.	6/429 (1.4%) vs. 1/165 (0.6%); p = 0.435.	30 days. Death 2.1% vs. 1.2% (p = 0.477); renal differences inconclusive.
Koga 2024 [116] 32 intended zero vs. 488 conventional; retrospective	Intended-zero eligibility eGFR < 30; group summaries NR-A.	20/32 completed at 0 mL; 12/32 required median 20.7 (11.0–31.2) mL.	Separate AKI incidence NR-A; not inferred from the early-safety composite.	Assessed within 3 months; separate event rate NR-A.	Early-safety success 78.1% vs. 86.8% (p = 0.184). Conversion 37.5%; retain all attempted cases.

Nerla 2024 [73] 44 zero vs. 63 standard; observational	GFR ≤ 35 mL/min eligibility; baseline mean eGFR 28.73 ± 11.06 vs. 34.33 ± 13.50.	0 mL dedicated planning/implantation vs. procedural mean 82 ± 78 mL; coronary angiography ≥ 1 month earlier.	No significant in-hospital difference. Later AKI 2/44 (4.5%) vs. 10/63 (15.9%) in source Table 4.	Two zero-group in-hospital deaths involved dialysis; standardized between-group new-RRT rate not tabulated separately.	Median ≈ 3.3 years. Later AKI favored zero contrast; adjusted mortality difference nonsignificant. Nonrandom operator allocation and inconsistent abstract reporting limit inference.
Elison 2024 [58] 35 implants; separate 100/60 sizing cohorts	Mean eGFR 21.5 ± 9.2.	Noncontrast CT surrogate sizing; 0 mL implantation. Earlier coronary exposure not uniformly quantified here.	Standard staged AKI rate NR; 9 patients had an eGFR fall ≥ 2 mL/min/1.73 m² at 24 h, not a KDIGO AKI definition.	2/35 (5.7%) within one week.	In-hospital/early. Technical success 100%; dialysis despite avoidance underscores persistent noncontrast renal risks.
Kazzi 2025 [115] 173 low vs. 264 normal before matching	CKD ≥ G3b; baseline eGFR 31 vs. 33 before matching.	Low contrast defined as procedural mL below numerical eGFR; absolute dose summaries NR-A.	Matched 17.9% vs. 28.3%; $p = 0.0217$. Abstract defines creatinine elevation >200% or ≥0.3 mg/dL.	No significant group difference; exact rate NR-A.	Early/30-day outcomes. No difference in mortality or readmission; matching does not exclude residual confounding.
Papadopoulos 2026 [117] 50; prospective series	Median eGFR 38 (29–48); 15/50 < 30.	Planning CTA median 45 mL; median interval 18 days; implantation 8 (6–10) mL, all ≤ 10.	1/50 (2.0%), KDIGO stage 1 through 30 days.	0/50 at 30 days; 0/49 with six-month clinical follow-up.	Six-month paired labs in 46/50: median eGFR 44 (34–56); clinical follow-up 49/50. Available-case, uncontrolled outcomes do not establish renal benefit.

AKI, acute kidney injury; CKD, chronic kidney disease; CrCl, creatinine clearance; CT, computed tomography; CTA, CT angiography; eGFR, estimated glomerular filtration rate; GFR, glomerular filtration rate; IQR, interquartile range; KDIGO, Kidney Disease Improving Global Outcomes; RRT, renal replacement therapy; SD, standard deviation; TAVI, transcatheter aortic valve implantation; VARC, Valve Academic Research Consortium.

12. Anatomical Complexity and the Limits of Contrast Avoidance

12.1. Valve-in-Valve Procedures

A visible surgical frame can guide valve-in-valve deployment but cannot establish coronary clearance. True internal diameter, leaflet configuration, coronary position, and the sinotubular junction remain decisive; registry evidence documents the importance of coronary obstruction [119].

Planned fracture, leaflet modification, or coronary protection increases the information required before implantation. A selected IVUS-guided zero-contrast case does not replace comprehensive assessment [68].

Multicenter valve-in-valve studies inform device–anatomy interaction and residual hemodynamics, not the renal effectiveness of contrast-sparing protocols [120,121].

12.2. Bicuspid Anatomy, Coronary Risk, and Noncalcified Valves

Bicuspid valves, a calcified raphe, horizontal aorta, or extensive outflow-tract calcium increase sizing and deployment uncertainty. Eligibility from straightforward calcific tricuspid stenosis should not be transferred without reassessing these features.

A zero-contrast BASILICA case demonstrates expert technical feasibility rather than routine eligibility for coronary-risk anatomy [122].

Pure noncalcified regurgitation provides fewer landmarks and different anchoring requirements; isolated reports cannot define a general contrast-free strategy [123].

12.3. Unusual Aortic and Access Anatomy

A prior coarctation bypass is an anatomical example requiring route-specific delivery planning, not evidence of renal benefit from contrast reduction [124].

Grafts, reconstruction, severe tortuosity, and alternative access may require definitive lumen and route imaging before instrumentation. The chosen pathway must address vascular, cerebral, and surgical risk alongside iodine exposure.

12.4. Valve Selection and Device-Specific Considerations

Choose the valve according to anatomy, anchoring, hemodynamics, coronary access, and deployment mechanics. Marker visibility and recapturability may help a contrast-sparing workflow but do not establish renal superiority.

LANDMARK provides randomized platform comparisons, not a randomized comparison of contrast protocols [125,126]. Its outcomes cannot serve as a contemporaneous renal control for a later single-center feasibility series.

13. Clinical Implementation and Renal Follow-Up

Table 7 translates renal vulnerability and anatomical uncertainty into explicit prerequisites for a proposed pathway. Stable eGFR < 30 mL/min/1.73 m^2 identifies advanced CKD requiring particular attention, and eGFR 30–44 with additional insults may warrant similar measures; these are vulnerability categories, not validated cutoffs assigning treatment. Active AKI requires a cause-and-urgency assessment because steady-state eGFR ratios are unreliable. Before attempting iodine-free planning and implantation, the team should have reliable device sizing, calcium assessment, coronary-risk characterization, complete access-lumen assessment, usable deployment landmarks, adequate echocardiographic assessment, and immediate rescue capability. Any treatment-changing uncertainty should direct further imaging rather than an automatic zero-contrast attempt.

Table 7. Evidence-informed selection of a personalized contrast-sparing pathway.

Clinical Context	Prerequisites and Planning Choice	Implantation and Escalation	Renal Surveillance and Boundary
Stable function; adequate anatomy, including eGFR ≥ 45	Review existing studies; optimized low-contrast CTA if needed. Avoid duplicate tests.	Omit redundant angiography only when landmarks and final valve assessment are reliable.	Routine risk-adapted testing. Preserved eGFR does not remove risk from shock or bleeding.
Stable eGFR 30–44 with diabetes, congestion, recent exposure, or anticipated complexity	Check trajectory and volume state; consider locally validated low-contrast CTA or complementary noncontrast tests.	Plan each acquisition and rescue criteria. These susceptibility features do not mandate zero contrast.	Assign testing after early discharge; reassess around 30 days and thereafter by course.
Stable eGFR < 30 with adequately resolved anatomy	Consider noncontrast CT plus noncontrast CMR or 3D TEE and complete vascular assessment. Confirm sizing, calcium, coronary relationships, and access lumen.	Zero/minimal contrast only with secure landmarks, adequate TTE/TEE, experienced operators, and immediate rescue capability.	Nephrology input as appropriate. Follow-up at 30 and about 90 days if injury occurs, then 6–12 months for CKD. Dialysis prevention unproven.
Advanced CKD with discordant sizing or unresolved coronary/root/access risk	Obtain targeted low-contrast CTA or another definitive test. Device-size disagreement, uncertain coronary clearance, or uncertain lumen precludes relying on incomplete imaging.	Use additional contrast for uncertain depth, ischemia, important AR, sustained hypotension, or suspected vascular injury.	Contrast target is subordinate to safe diagnosis. eGFR < 30 is not a prohibition on justified iodine.
Active AKI or decompensated HF at any eGFR	Determine cause and urgency; correct reversible factors if elective, or expedite necessary treatment for stenosis-driven deterioration.	Support perfusion and volume balance; do not calculate a safe allowance from nonsteady-state eGFR.	Earlier serial testing; nephrology for severe/progressive injury. Avoid arbitrary delay for a numerical target.
Indicated PCI or complex ViV, bicuspid, or reconstructed anatomy	Select PCI by coronary indication; consider IVUS. Resolve sizing, coronary obstruction, and delivery-route uncertainty before TAVI.	Individualize staging and total burden; preplan coronary/access rescue. Complex anatomy can require definitive contrast imaging.	No universal PCI timing or device has established renal superiority. Exceptional cases do not define routine eligibility.
Maintenance dialysis or kidney transplant	Assess residual/graft function, volume tolerance, medications, access, and prognosis.	Protect dialysis access; use clinically indicated renal support and definitive anatomical imaging.	Separate residual/graft outcomes; new-dialysis endpoints are inapplicable in maintenance dialysis.

This is a proposed framework, not a validated decision rule or a guideline recommendation. CKD, chronic kidney disease; CMR, cardiovascular magnetic resonance; CT, computed tomography; CTA, CT angiography; eGFR, estimated glomerular filtration rate; HF, heart failure; IVUS, intravascular ultrasound; PCI, percutaneous coronary intervention; TEE, transesophageal echocardiography; ViV, valve-in-valve.

Record a stable pre-pathway renal baseline where available and a recent preimplantation sample to capture deterioration during work-up. During admission, reassess creatinine/electrolytes and clinically observed urine output according to risk; high-risk or unstable patients require surveillance through the relevant AKI window. For early

discharge, assign responsibility and timing for repeat testing before the patient leaves. A pragmatic follow-up plan includes renal reassessment around 30 days, at approximately 90 days after AKI or unresolved deterioration, and at 6–12 months in CKD or after AKI, with earlier testing for rising creatinine, oliguria, congestion, or medication changes. These proposed intervals support implementation rather than prescribe a trial-validated schedule. Distinguish transient AKI, acute kidney disease persisting during days 7–90, and abnormalities persisting beyond 90 days requiring CKD reassessment [9,10,18,127].

At follow-up, report eGFR or creatinine relative to baseline, new and ongoing RRT, recurrent AKI, CKD-stage change, and death. Specify numerical deterioration and recovery thresholds prospectively, laboratory availability at each time point, and outcomes among patients who died or started dialysis. Survivor-only averages should not be interpreted as recovery for the original cohort. Audit all attempted pathways, including rescans and conversions, together with bleeding, vascular injury, significant residual regurgitation, technical success, and patient-reported burden. Nephrology review and medication reintroduction should follow the cause and persistence of injury.

14. Discussion

14.1. What the Evidence Supports

Iodinated contrast can be reduced at several stages of TAVI care without abandoning the information required for treatment. The strongest evidence concerns imaging adequacy and procedural feasibility; it is less persuasive for AKI reduction and insufficient for a causal reduction in dialysis, persistent dysfunction, or mortality. The randomized CMR trial answers a planning question in a population excluding advanced CKD, whereas observational contrast cohorts address selected procedural strategies with different comparators. Neither supplies a randomized test of a complete contrast-sparing pathway [58,63,72,73,113–116].

Post-TAVI AKI is multifactorial, and relief of stenosis may improve renal physiology. This complicates attribution both of injury to contrast and of recovery to contrast avoidance [3,4,10]. A zero-contrast target, therefore, has clinical value only when the anatomy is adequately resolved and the complete care pathway is considered. In practice, the decision turns on renal vulnerability, the consequences of missing anatomical information, the reliability of alternative imaging, and local capability. Table 7 organizes these considerations without assigning an evidence-based treatment rule. Implementation of the proposed pathway also depends on institutional imaging expertise and resource availability. CMR, advanced CT reconstruction, image fusion, and multimodality assessment require suitable equipment and experienced acquisition and interpretation teams. The Heart Team should select a pathway that can be performed reliably within the center's capabilities and use additional contrast-enhanced imaging when essential anatomical or procedural questions remain unresolved.

14.2. Limitations of This Review

A targeted narrative search is less reproducible and potentially less comprehensive than a systematic review. Representative queries improve transparency but cannot establish exhaustive retrieval or reconstruct an original screening denominator. Independent duplicate screening is not claimed; formal risk-of-bias and certainty grading were not performed. English-language evidence and incomplete access to some full reports may introduce availability or language bias. Variation in terminology, exposure windows, CKD severity, AKI definitions, anatomical exclusions, cointerventions, and laboratory follow-up limits cross-study comparison. The authors' clinical experience and participation in some

cited studies may have influenced emphasis or the proposed framework. The framework remains unvalidated.

14.3. Priorities for Comparative Research

The next informative comparison is a complete, prespecified pathway rather than a contrast-volume label alone. Studies should document coronary, planning, implantation, rescue, and repeat-imaging exposure; retain conversions in the assigned strategy; and balance renal severity and anatomical complexity. Outcomes should include diagnostic adequacy, technical success, AKI stage, RRT, persistent dysfunction, bleeding, vascular complications, survival, and patient-reported burden. Repeated renal measurements at 30 and 90 days and 6–12 months would help distinguish acute injury, recovery, and CKD progression. Death and dialysis should be reported explicitly and handled appropriately when analyzing renal trajectories. Prospective validation should test whether the prerequisites and escalation criteria in Table 7 identify patients in whom contrast avoidance preserves safety and improves outcomes, rather than assuming that lower volume itself constitutes clinical success.

15. Conclusions

Contrast-sparing TAVI is feasible in selected patients when imaging and procedural planning address renal vulnerability and anatomical complexity together. Technical feasibility is better established than comparative renal effectiveness. Additional contrast should be used whenever required for diagnostic or procedural safety. Whether reducing exposure across the complete treatment pathway prevents dialysis, persistent kidney dysfunction, or death remains unproven and requires prospective comparative evaluation.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/jpm16120602/s1. File S1: Representative PubMed strategies, selection criteria, extraction conventions, and a source verification record.

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Abbreviations

The following abbreviations are used in this manuscript:

3D	Three-dimensional
ACC	American College of Cardiology
ACR	American College of Radiology
ADQI	Acute Disease Quality Initiative
AKD	Acute kidney disease

AKI	Acute kidney injury
AR	Aortic regurgitation
AS	Aortic stenosis
BASILICA	Bioprosthetic or native aortic scallop intentional laceration to prevent iatrogenic coronary artery obstruction
CA-AKI	Contrast-associated acute kidney injury
CI	Confidence interval
CI-AKI	Contrast-induced acute kidney injury
CKD	Chronic kidney disease
CMR	Cardiovascular magnetic resonance
CO_2	Carbon dioxide
CrCl	Creatinine clearance
CT	Computed tomography
CTA	Computed tomography angiography
eGFR	Estimated glomerular filtration rate
ESUR	European Society of Urogenital Radiology
FFR	Fractional flow reserve
GFR	Glomerular filtration rate
HF	Heart failure
HR	Hazard ratio
IQR	Interquartile range
IVUS	Intravascular ultrasound
KDIGO	Kidney Disease: Improving Global Outcomes
LVEDP	Left ventricular end-diastolic pressure
MI	Myocardial infarction
MR	Magnetic resonance
MRI	Magnetic resonance imaging
NR	Not reported separately in the available source
NR-A	Not separately available in the accessible abstract
NYHA	New York Heart Association
PCI	Percutaneous coronary intervention
RRT	Renal replacement therapy
SCCT	Society of Cardiovascular Computed Tomography
SD	Standard deviation
STS	Society of Thoracic Surgeons
TAVI	Transcatheter aortic valve implantation
TAVR	Transcatheter aortic valve replacement
TEE	Transesophageal echocardiography
TTE	Transthoracic echocardiography
TVT	Transcatheter Valve Therapy
VARC	Valve Academic Research Consortium
VARC-2	Valve Academic Research Consortium 2
VARC-3	Valve Academic Research Consortium 3
ViR	Valve-in-ring
ViV	Valve-in-valve

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