

REVIEW ARTICLE

Silent cerebral injury after cardiac interventions:
The role of advanced cardiac imaging at the
neurocardiology interface

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Abstract

Silent cerebral injury (SCI) has emerged as a frequent but underrecognized consequence of contemporary cardiac interventions. Advances in diffusion-weighted magnetic resonance imaging have revealed that subclinical ischemic brain lesions occur commonly after procedures such as transcatheter valve implantation, surgical valve replacement, and percutaneous coronary intervention. Although these lesions do not produce immediate neurological symptoms, growing evidence suggests that they are associated with long-term adverse outcomes, including cognitive decline and increased risk of future stroke. The mechanisms underlying SCI are multifactorial and involve both embolic phenomena and transient cerebral hypoperfusion during procedural manipulation of cardiac and aortic structures. Traditional clinical risk models are limited in their ability to predict subclinical neurovascular injury. In this context, advanced cardiac imaging modalities, including transesophageal echocardiography, cardiac computed tomography, and, in selected cases, cardiac magnetic resonance imaging, may offer valuable insights by identifying structural features such as complex aortic plaques, prosthetic abnormalities, or extensive calcification that predispose to embolization. Integrating imaging-derived markers into pre-procedural assessment may support improved risk stratification and individualized procedural planning. This narrative review summarizes current evidence on SCI following cardiac interventions and explores the potential role of advanced cardiac imaging in predicting neurovascular complications. A greater integration of cardiovascular imaging and neurovascular outcomes may contribute to more effective strategies aimed at reducing subclinical brain injury in modern cardiac practice.

Keywords: Silent cerebral injury; Cardiac interventions; Transesophageal echocardiography; Cardiac computed tomography; Neurocardiology

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1. Introduction

The interaction between the cardiovascular and nervous systems represents a complex and bidirectional relationship increasingly recognized in modern clinical medicine. Traditionally, cardiac interventions have been evaluated primarily in terms of hemodynamic success and overt neurological complications such as clinically evident stroke. However, advances in neuroimaging, particularly diffusion-weighted magnetic resonance imaging (DWI-MRI), have revealed that cerebral injury following cardiac procedures is far more common than previously appreciated, frequently occurring in a subclinical form known as silent cerebral injury (SCI).^{1,2}

Silent cerebral injury is defined as the presence of new ischemic brain lesions detected on imaging without accompanying focal neurological deficits. Although clinically inapparent at the time of occurrence, these lesions are not benign. Accumulating evidence links SCI to long-term adverse outcomes, including cognitive decline, impaired executive function, and an increased risk of future clinical stroke.^{3,4} Importantly, SCI appears to be highly prevalent following a broad spectrum of cardiac interventions, including transcatheter valve therapies, surgical valve replacement, and percutaneous coronary procedures.^{5,6}

Recent multicenter studies and pooled analyses using contemporary-generation transcatheter devices have confirmed that DWI-MRI-detected silent cerebral lesions remain frequent despite technological advances and increasing operator experience.⁷ Although improvements in device profiles and procedural strategies have contributed to reductions in overt neurological complications, subclinical ischemic lesions continue to be observed in a substantial proportion of patients undergoing structural and transcatheter cardiac interventions, highlighting the persistent relevance of imaging-based neurovascular risk assessment in modern practice.^{8,9}

The pathogenesis of SCI is multifactorial. Cardiac procedures often involve manipulation of calcified structures, intracardiac devices, or atherosclerotic aortic segments, creating conditions conducive to microembolization. In addition, transient cerebral hypoperfusion during procedural steps such as rapid pacing or cardiopulmonary bypass may further contribute to ischemic injury.^{10,11} Despite these well-recognized mechanisms, current clinical risk stratification models remain largely focused on predicting overt stroke and fail to adequately identify patients at risk for subclinical neurological injury.

Parallel to the growing awareness of SCI, advances in

cardiac imaging have expanded the diagnostic capabilities of modalities such as transesophageal echocardiography (TEE), three-dimensional echocardiography, and cardiac computed tomography (CT). These tools are increasingly able to identify potential embolic substrates, including complex aortic plaques, intracardiac thrombi, and prosthetic valve abnormalities, before intervention.^{12,13} This raises the possibility that cardiac imaging may evolve from a purely anatomical and procedural planning tool into a predictive modality for neurovascular risk.

Within this emerging neurocardiology framework, understanding the relationship between imaging-derived cardiac pathology and cerebral injury is of growing clinical importance. Identifying imaging markers that correlate with SCI may allow for improved procedural planning, individualized neuroprotection strategies, and ultimately better long-term neurological outcomes.

This narrative review was based on a structured literature search performed in PubMed/MEDLINE, Embase, Scopus, and Web of Science covering studies published between January 2000 and December 2025. The search terms included combinations of “silent cerebral infarction,” “cardiac interventions,” “transcatheter aortic valve replacement,” “TAVR,” “percutaneous coronary intervention,” “PCI,” “structural heart procedures,” “transesophageal echocardiography,” “cardiac computed tomography,” “cardiac magnetic resonance,” “CMR,” “left ventricular thrombus,” “myocardial fibrosis,” and “neurocardiology.” Reference lists of relevant studies were also screened. Peer-reviewed human studies in English reporting clinical, imaging, or mechanistic data related to cerebral injury after cardiac procedures were included. Case reports and non-systematic opinion papers were excluded. No meta-analysis was performed due to the narrative design.

The aim of this review is to summarize the current understanding of SCI following cardiac interventions and to examine the potential role of advanced cardiac imaging in identifying patients at increased risk of neurovascular complications. In addition, this review seeks to highlight imaging-derived markers that may contribute to improved procedural risk stratification and support the integration of neurocardiology principles into routine cardiovascular practice.

2. Silent cerebral injury: Definition and clinical relevance

Silent cerebral injury refers to newly detected ischemic lesions on DWI-MRI that occur in the absence of overt neurological symptoms. These lesions are increasingly recognized after a wide range of cardiac interventions

and represent a form of subclinical brain injury that often remains undiagnosed in routine clinical practice.^{14,15} Unlike clinically apparent stroke, silent lesions do not produce immediate focal deficits, which has historically led to their underestimation in procedural risk assessment.

Despite their subclinical presentation, silent cerebral infarcts carry important prognostic implications. Longitudinal studies have demonstrated that these lesions are associated with an increased risk of future symptomatic stroke, cognitive decline, and functional impairment.¹⁶ The cumulative burden of silent infarction has also been linked to structural brain changes, including white matter damage and cortical atrophy, suggesting a progressive impact on neurovascular integrity over time.¹⁷

The incidence of SCI varies according to the type of cardiac procedure performed. Transcatheter interventions, particularly those involving manipulation of calcified valves or traversal of the aortic arch, have been associated with high rates of new diffusion-restricted lesions detected post-procedure.¹⁸ Surgical interventions may similarly contribute to cerebral injury through embolic phenomena or hemodynamic instability during cardiopulmonary bypass.¹⁹ Importantly, silent lesions frequently outnumber clinically apparent strokes, emphasizing that neurological injury extends beyond events detectable through standard clinical evaluation.

Growing evidence suggests that even small ischemic lesions may disrupt cognitive networks and contribute to subtle but measurable deficits in executive function, memory, and processing speed.²⁰ This is particularly relevant in elderly patients undergoing structural heart procedures, where baseline cerebrovascular reserve may already be limited.

Silent cerebral injury is not a uniform entity, and its clinical relevance may depend on lesion burden and anatomical distribution. Diffusion-weighted imaging (DWI) studies have demonstrated that these lesions frequently follow embolic patterns involving cortical and subcortical territories, although watershed-type infarcts related to transient hypoperfusion have also been described. Importantly, lesion location may influence functional outcomes, as infarcts affecting strategic regions such as the frontal-subcortical networks or posterior circulation may contribute to subtle cognitive impairment despite the absence of overt neurological deficits. Increasing lesion burden has been associated with cumulative disruption of neural connectivity and may partially explain the link between procedural SCI and long-term cognitive decline.¹⁷

Beyond lesion burden alone, growing neuroimaging evidence suggests that DWI-MRI-detected silent

cerebral lesions may represent early markers of long-term neurovascular vulnerability.²⁰ Several longitudinal studies have demonstrated an association between silent infarcts and an increased risk of future symptomatic stroke as well as progressive cognitive impairment, particularly in domains such as executive function and processing speed.²¹ In addition, lesion location appears to influence clinical relevance, as infarcts involving frontal-subcortical networks and posterior circulation territories may contribute disproportionately to subtle cognitive decline despite the absence of overt neurological symptoms. These observations support the concept that imaging-detected SCI should be interpreted not only as a procedural finding but also as a marker of cumulative cerebrovascular risk.²²

Recognition of SCI shifts the focus from purely symptomatic neurological outcomes toward a broader understanding of brain health after cardiac intervention. This perspective highlights the need for improved risk prediction strategies capable of identifying patients who may be vulnerable to subclinical neurovascular injury.

3. Pathophysiological mechanisms of silent cerebral injury during cardiac interventions

Silent cerebral injury following cardiac procedures arises from a complex interplay of embolic and hemodynamic mechanisms that may occur during or immediately after intervention. Although overt neurological complications are relatively infrequent, subclinical ischemic lesions detected on DWI suggest that cerebral microinjury is a common procedural consequence.²³ These lesions are most often attributed to microembolization originating from intracardiac structures, prosthetic material, or the aortic wall.

Procedural manipulation of calcified valves, atherosclerotic plaques, and intracardiac devices can generate particulate debris that enters the cerebral circulation. Studies using transcranial Doppler have demonstrated frequent microembolic signals during interventions such as transcatheter valve implantation and percutaneous coronary procedures, supporting embolization as a principal mechanism of injury.²⁴ In addition to solid particles, gaseous emboli introduced through catheter systems may contribute to cerebral ischemia, particularly during prolonged or complex procedures. The relative contribution of gaseous emboli compared with particulate debris remains difficult to quantify in clinical imaging studies, as most available evidence derives from intra-procedural monitoring techniques rather than post-procedural lesion characterization using DWI-MRI.²⁵ Unlike particulate emboli, which may cause persistent occlusion of distal

cerebral arterioles and result in focal ischemic injury detectable on DWI-MRI, gaseous emboli typically produce transient microvascular obstruction that may resolve more rapidly through dissolution and redistribution within the circulation. However, even short-lived air embolization may disrupt endothelial integrity and impair microcirculatory perfusion, potentially contributing to subclinical ischemic lesions during catheter-based cardiac interventions.^{11,23,26,27}

Hemodynamic instability represents another important contributor to SCI. Transient reductions in cerebral perfusion may occur during rapid pacing, balloon inflation, or cardiopulmonary bypass, creating conditions

that predispose vulnerable brain regions to ischemia.²⁸ Even brief periods of hypoperfusion can lead to diffusion-restricted lesions, particularly in patients with impaired autoregulatory capacity or pre-existing cerebrovascular disease.

Endothelial dysfunction and procedural inflammation may further amplify the risk of neurovascular injury. Mechanical stress and device interaction with vascular structures can disrupt endothelial integrity, promoting thrombus formation and microvascular obstruction.^{11,29} Systemic inflammatory responses triggered by intervention have also been associated with increased blood-brain barrier permeability, potentially exacerbating cerebral

Table 1. Mechanisms of silent cerebral injury during cardiac procedures

Mechanism	Source of injury	Procedural triggers	Imaging correlate	References
Particulate embolism	Calcific debris/thrombus	Valve deployment, catheter manipulation	Cortical infarcts	10,25,30,31
Gaseous embolism	Air entry during device use	Long procedural time	Scattered lesions	23,26,27
Hypoperfusion	Transient cerebral flow reduction	Rapid pacing, cardiopulmonary bypass	Watershed lesions	11,32,33
Endothelial injury	Vascular wall trauma	Device interaction	Microvascular ischemia	4,11,34
Inflammatory activation	Systemic inflammatory response	Surgical manipulation	Diffuse injury patterns	2,15,35

vulnerability to ischemic insult.

Silent cerebral injury may arise through two principal pathophysiological patterns. Embolic lesions are typically distributed within cortical or subcortical territories and are most often linked to particulate or gaseous embolization originating from the heart, prosthetic material, or aortic atheroma. In contrast, watershed-type lesions are more frequently associated with transient reductions in cerebral perfusion and tend to occur in border-zone regions between major vascular territories. Procedural factors such as rapid pacing, cardiopulmonary bypass, or brief hypotensive episodes may contribute to this hypoperfusion-related injury. The coexistence of embolic and hypoperfusion mechanisms suggests that SCI represents a heterogeneous phenomenon rather than a single pathophysiological entity.^{23,28}

These mechanisms often coexist, reflecting the multifactorial nature of SCI. Understanding the procedural and patient-specific factors that contribute to embolic generation and hypoperfusion is essential for improving risk prediction and guiding strategies aimed at minimizing neurological complications. A summary of the principal mechanisms and their procedural correlates is presented in [Table 1](#), and a schematic overview

illustrating the relationship between procedural embolic sources, pathophysiological mechanisms of SCI, imaging-based detection, and potential long-term neurological consequences is presented in [Figure 1](#).

4. Procedural risk burden

The occurrence of SCI varies substantially across different cardiac interventions, reflecting differences in procedural complexity, embolic potential, and hemodynamic impact. Among contemporary procedures, transcatheter aortic valve replacement has been consistently associated with a high incidence of new ischemic lesions detected on post-procedural DWI.³⁶ The need to traverse the aortic arch and manipulate heavily calcified native valves creates a significant embolic substrate, while procedural steps such as valve deployment may further dislodge particulate material into the cerebral circulation.

Surgical valve replacement is also associated with cerebral microinjury, although the mechanisms differ in part from those observed in transcatheter procedures. Cardiopulmonary bypass and aortic cross-clamping may introduce both embolic and hypoperfusion-related risks. Microemboli may arise from aortic manipulation or air entrainment, whereas fluctuations in cerebral perfusion during bypass can contribute to ischemic vulnerability,

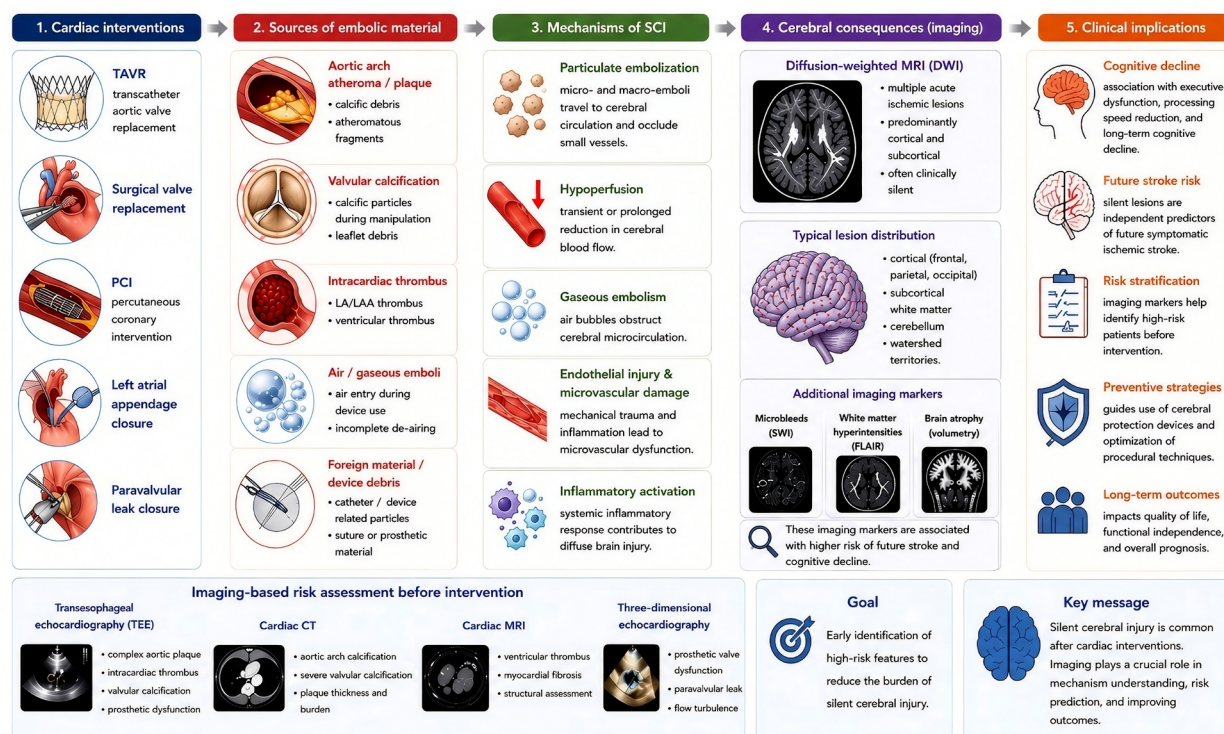


Figure 1. This schematic illustration summarizes the relationship between cardiac procedural factors, embolic sources, pathophysiological mechanisms of SCI, neuroimaging findings, and potential long-term neurological consequences. Cardiac interventions such as transcatheter valve procedures, surgical valve replacement, PCI, and structural heart interventions may generate embolic material originating from calcified valves, aortic arch atheroma, intracardiac thrombi, gaseous emboli, or device-related debris. These factors contribute to cerebral injury through mechanisms including particulate embolization, transient hypoperfusion, endothelial injury, and inflammatory activation. Diffusion-weighted magnetic resonance imaging commonly detects procedure-related silent ischemic lesions with typical cortical, subcortical, or watershed distributions. Imaging-derived structural markers identified by TEE, three-dimensional echocardiography, cardiac computed tomography, and cardiac magnetic resonance imaging may help identify patients at increased embolic susceptibility before intervention. These imaging findings are associated with an increased risk of cognitive decline, future stroke, and cumulative neurovascular vulnerability, supporting the integration of cardiac imaging into pre-procedural neurovascular risk stratification. Abbreviations: CT: Computed tomography; DWI: Diffusion-weighted imaging; FLAIR: Fluid-attenuated inversion recovery; LA: Left atrium; LAA: Left atrial appendage; MRI: Magnetic resonance imaging; PCI: Percutaneous coronary intervention; SCI: Silent cerebral injury; SWI: Susceptibility-weighted imaging; TAVR: Transcatheter aortic valve replacement; TEE: Transesophageal echocardiography.

particularly in patients with impaired autoregulatory reserve.³⁷

Percutaneous coronary intervention carries a comparatively lower but still measurable risk of SCI, with DWI-MRI studies reporting new silent cerebral lesions in approximately 10–30% of cases, compared with rates of approximately 60–90% reported after transcatheter aortic valve replacement.^{2,8,38} Catheter manipulation within the aortic root and coronary ostia may disturb atherosclerotic plaques, resulting in embolization to the cerebral circulation. Although these lesions are often small, their cumulative burden may be clinically relevant over time.^{39,40}

Structural heart interventions beyond valve

replacement, including procedures such as paravalvular leak closure and left atrial appendage occlusion, have also been linked to diffusion-restricted cerebral lesions. Device delivery systems and repeated intracardiac manipulation may increase the likelihood of embolic material entering the systemic circulation.⁴¹

Although reported incidence rates of SCI differ substantially across procedural categories, direct comparisons between studies should be interpreted with caution. Considerable variability exists in patient selection, imaging timing after intervention, approaches to lesion definition, and procedural techniques across cohorts.^{2,9,32,42} In addition, differences between early- and newer-generation transcatheter devices and increasing operator

experience over time may further influence embolic burden and lesion detection rates.^{8,43,44} These factors collectively contribute to the wide range of reported SCI incidence across contemporary cardiac interventions and may partially limit cross-study comparability.

Taken together, these observations suggest that

SCI is not confined to a single procedural category but represents a broader consequence of contemporary cardiac intervention. The variability in risk across procedures underscores the need for improved identification of patients who may be particularly susceptible to subclinical neurovascular injury. A comparative overview of reported SCI rates across commonly performed cardiac procedures is summarized in [Table 2](#).

Table 2. Incidence of silent cerebral injury (SCI) across cardiac interventions

Procedure type	Reported SCI incidence (%)	Predominant mechanism	Typical lesion pattern	Representative studies
Transcatheter aortic valve replacement (TAVR)	60–90	Particulate embolization from the calcified valve and aortic arch manipulation	Cortical/multifocal	5,8,36
Surgical valve replacement	30–60	Embolization + hypoperfusion during cardiopulmonary bypass	Watershed/subcortical	31,32,33
Percutaneous coronary intervention (PCI)	10–30	Aortic arch and catheter-related embolization	Small focal lesions	38,40,45
Left atrial appendage closure	20–40	Device-related thromboembolism	Cortical	35,46
Paravalvular leak closure	30–50	Device manipulation and residual thrombotic material embolization	Multifocal	23,47

5. Limitations of current neurological risk prediction

Current approaches to neurological risk assessment in cardiac interventions remain largely centered on the prediction of clinically overt stroke. Traditional risk models incorporate demographic variables, vascular comorbidities, and procedural characteristics, yet they do not adequately capture the risk of subclinical cerebral injury. This limitation partly reflects the absence of immediate neurological deficits in silent cerebral infarction, which has historically limited its inclusion in procedural outcome frameworks.³⁵

Standard clinical predictors such as age, atrial fibrillation, or prior cerebrovascular disease may provide a general estimate of stroke susceptibility but lack specificity in identifying patients who are particularly vulnerable to microembolic or hypoperfusion-related injury during intervention.³³ Furthermore, these models do not account for structural cardiac or aortic features that may serve as embolic sources.

Another important limitation lies in the absence of imaging-based parameters within routine neurological risk stratification. While cerebral imaging has clarified the high prevalence of silent lesions after cardiac procedures, pre-procedural evaluation rarely incorporates cardiac imaging

markers that may signal increased embolic potential. Features such as complex aortic plaques, prosthetic valve dysfunction, or intracardiac thrombus burden are often assessed for procedural planning but not systematically linked to neurological outcomes.⁴⁸

As a result, the current paradigm focuses on managing manifest complications rather than anticipating subclinical injury. This gap underscores the need for a more integrated approach that combines procedural, clinical, and imaging-derived data to improve identification of patients at risk for SCI.

6. Advanced cardiac imaging as a predictor of neurovascular risk

Advances in cardiac imaging have significantly improved the detection of structural and functional abnormalities associated with cerebral embolic risk before cardiac interventions. Modalities such as TEE, three-dimensional echocardiography, and cardiac CT are routinely employed for anatomical evaluation and procedural planning. Beyond their traditional roles, these techniques increasingly provide information that may help identify patients at heightened risk of neurovascular complications.⁴⁹

Transesophageal echocardiography offers a detailed assessment of the thoracic aorta, cardiac chambers, and

prosthetic structures. In particular, complex aortic plaques visualized by TEE have been shown to correlate with an elevated risk of embolic events. Features such as plaque thickness, ulceration, or mobility are recognized markers of embolic potential and may indicate an increased likelihood of cerebral microembolization during catheter-based or surgical manipulation.³⁴ In addition, TEE enables detection of intracardiac thrombi and prosthetic valve abnormalities that may otherwise remain clinically silent yet represent potential sources of embolic material.

Three-dimensional echocardiography enhances structural visualization and allows more precise characterization of valvular and periprosthetic anatomy. Subtle flow disturbances related to prosthetic dysfunction or paravalvular leak can be better delineated with three-dimensional imaging. These abnormal flow patterns may promote thrombus formation or contribute to embolic release during intervention, providing a mechanistic link between imaging findings and the development of SCI.⁵⁰

Cardiac CT complements echocardiographic assessment by offering high-resolution visualization of calcific burden within the aortic arch and valvular apparatus. Extensive calcification identified on CT has been associated with increased susceptibility to particulate embolization during device delivery and deployment.⁵¹ Pre-procedural recognition of these structural features may therefore help anticipate neurovascular risk and guide procedural strategy.

Cardiac magnetic resonance (CMR) imaging provides additional complementary information for the identification of potential embolic substrates that may not be fully characterized by echocardiography or cardiac CT. In particular, CMR allows sensitive detection of left ventricular thrombus, myocardial fibrosis, and regional wall motion abnormalities that may contribute to thrombus formation and subsequent embolization.^{52,53} Late gadolinium enhancement imaging can also identify areas of myocardial scar that may serve as substrates for impaired contractility and intracavitary thrombus development. In selected patients, CMR may therefore support a more comprehensive assessment of structural cardiac abnormalities associated with increased embolic susceptibility prior to intervention.^{47,54}

Beyond structural assessment, advanced cardiac imaging may contribute to procedural risk prediction by identifying embolic substrates before intervention.⁴⁴ Features such as complex aortic plaque morphology, leaflet thickening suggestive of subclinical thrombosis, or extensive valvular and aortic calcification may indicate

an increased likelihood of particulate embolization during device manipulation. Incorporating these findings into pre-procedural evaluation may allow clinicians to anticipate neurovascular vulnerability rather than react to post-procedural injury.⁵¹ In this context, imaging may evolve from a diagnostic tool into a predictive instrument supporting individualized procedural planning.

Despite the growing interest in imaging-derived predictors of embolic susceptibility, most currently available evidence remains based on observational associations rather than prospective validation studies. Although structural abnormalities such as complex aortic plaques, intracardiac thrombi, or extensive calcification are biologically plausible embolic substrates, their incremental predictive value beyond traditional clinical risk factors has not yet been consistently established. Standardized imaging-based risk models integrating cardiac structural markers with neurological outcomes remain limited, highlighting the need for multicenter prospective validation studies.^{11,55}

Taken together, these imaging modalities enable a more comprehensive evaluation of potential embolic substrates. The integration of echocardiographic and tomographic findings may support a shift toward imaging-informed risk stratification, in which cardiac imaging contributes not only to anatomical assessment but also to the prediction of SCI. Advanced cardiac imaging modalities may identify structural features associated with increased embolic susceptibility. Imaging-derived markers that may contribute to neurovascular risk prediction are outlined in [Table 3](#).

7. Imaging markers associated with silent cerebral injury

Several imaging-derived features have emerged as potential indicators of increased susceptibility to SCI following cardiac interventions. These markers often reflect structural abnormalities or flow-related disturbances that may serve as sources of embolization during procedural manipulation.

Complex aortic plaques are among the most consistently identified imaging findings associated with cerebral embolic risk. Plaques characterized by increased thickness, irregular surface morphology, or mobility have been linked to a higher likelihood of embolic events. In clinical imaging practice, complex aortic plaques are commonly defined as lesions measuring ≥ 4 mm in thickness and/or demonstrating ulceration, mobile components, or superimposed thrombus, features that are associated with increased embolic potential during cardiac

Table 3. Imaging markers associated with increased neurovascular risk

Imaging modality	Risk marker identified	Mechanism of embolic risk	Potential clinical implication	Representative evidence
Transesophageal echocardiography	Complex aortic plaque	Plaque disruption and atheroembolism during catheter or surgical manipulation	Identifies patients with increased embolic susceptibility before intervention	12,48,56
	Intracardiac thrombus	Direct thromboembolism from atrial or ventricular chambers	May guide anticoagulation optimization or procedural postponement	47,52,55
Three-dimensional echocardiography	Prosthetic valve dysfunction or paravalvular leak	Flow turbulence promoting thrombus formation and embolic release	Supports detailed prosthetic assessment before repeat intervention	47,50,57
Cardiac computed tomography	Severe valvular calcification	Particulate embolization during device crossing, ballooning, or valve deployment	May support the use of embolic protection or procedural strategy modification	5,30,31
	Aortic arch calcification/atheroma	Atheromatous debris embolization during arch traversal	May influence access route, catheter manipulation strategy, or neuroprotection planning	12,58,59
Cardiac magnetic resonance	Ventricular thrombus or structural myocardial substrate	Thromboembolism from intracardiac thrombotic material	May complement echocardiography when thrombus detection is uncertain	47,52,54

interventions.^{34,56,58,60} Identification of these features prior to intervention may therefore provide insight into the potential for microembolization.

Prosthetic valve abnormalities also represent a relevant substrate. Subclinical leaflet thickening or early thrombotic changes may not produce overt hemodynamic consequences but can nonetheless serve as a nidus for embolic material.⁵⁷ Similarly, residual paravalvular flow jets may create localized regions of turbulence that promote thrombus formation and subsequent embolization.

Severe calcification of valvular or aortic structures detected on imaging has likewise been associated with procedural embolic load. Manipulation of heavily calcified tissue may result in the release of particulate matter into the systemic circulation.⁶¹ In addition, intracardiac thrombi, particularly within the left atrial appendage, remain an important embolic source even in the absence of clinical atrial fibrillation.⁴⁶

Although several structural imaging markers have been associated with increased embolic susceptibility during cardiac interventions, their independent prognostic significance for long-term neurological outcomes remains incompletely defined. In many studies, imaging findings are evaluated primarily in relation to surrogate imaging

endpoints such as diffusion-restricted lesion detection rather than clinically meaningful outcomes, including cognitive decline or future stroke risk. Furthermore, some imaging markers may reflect the overall burden of systemic atherosclerosis rather than procedure-specific embolic vulnerability, which complicates the interpretation of their causal contribution to SCI.^{49,60,62,63}

These imaging markers highlight the potential value of structural assessment in identifying patients at increased neurovascular risk. Incorporating such findings into pre-procedural evaluation may enhance the ability to anticipate SCI and tailor procedural strategies accordingly.

8. Limitations and heterogeneity of the current evidence

The interpretation of SCI following cardiac interventions remains challenging because the currently available evidence is characterized by substantial methodological heterogeneity across studies. Most investigations evaluating procedure-related SCI are observational in design and frequently originate from single-center cohorts with relatively limited sample sizes. In addition, different cardiac procedures, including transcatheter valve therapies, coronary interventions, and structural heart procedures, are often evaluated within heterogeneous study populations.

These variations complicate comparisons across studies and may limit the generalizability of reported incidence rates and imaging-derived risk associations.^{7,25,64,65}

Another important limitation relates to variability in the definition and measurement of SCI across neuroimaging protocols. Although DWI-MRI is considered the reference technique for detecting acute silent ischemic lesions, substantial differences exist between studies regarding the timing of imaging acquisition after intervention and the parameters used to quantify lesion burden. Some investigations report only the presence or absence of new lesions, whereas others provide lesion counts, volumetric measurements, or anatomical distribution patterns. This variability reduces the comparability of reported results and contributes to inconsistencies in estimating the clinical relevance of imaging-detected lesions.^{8,43,66,67}

Interpretation of procedural SCI risk is further influenced by differences in device generations and procedural techniques across study eras. Early investigations of transcatheter aortic valve replacement frequently involved first-generation delivery systems associated with larger catheter profiles and greater manipulation of the aortic arch. Contemporary procedural strategies using newer-generation devices may therefore be associated with a different embolic profile. As a result, earlier reports of lesion incidence may not fully reflect current procedural practice, particularly in centers with increasing operator experience and improved device technology.^{51,68,69}

Another key challenge arises from the widespread use of surrogate imaging endpoints rather than long-term neurological outcomes. Many studies rely primarily on the detection of new diffusion-restricted lesions as procedural endpoints, whereas systematic evaluation of cognitive decline, functional impairment, or subsequent stroke risk remains limited in many cohorts. Although imaging-detected lesions are biologically plausible markers of microvascular injury, the strength and consistency of their association with long-term clinical outcomes remain incompletely defined across procedural populations.^{2,35,45,70}

Finally, caution is required when interpreting the relationship between imaging-derived structural markers and the occurrence of SCI because most available data demonstrate associations rather than direct causal effects. Imaging findings such as complex aortic plaque, valvular calcification, or intracardiac thrombus may identify patients with increased embolic susceptibility; however, these markers may also reflect the overall burden of systemic atherosclerotic disease rather than independent procedural risk determinants. Consequently, prospective multicenter studies integrating standardized imaging protocols with longitudinal neurological assessment are

needed to clarify the causal relationship between imaging-defined embolic substrates and long-term neurovascular outcomes.^{13,34,59,71}

9. Future directions

The recognition of SCI as a frequent consequence of cardiac interventions has prompted increasing interest in strategies aimed at reducing neurovascular risk. Future developments are likely to focus on the integration of imaging findings into procedural planning, allowing clinicians to anticipate embolic vulnerability before intervention.³⁰ Such an approach may facilitate individualized procedural strategies, including selection of access routes or devices that minimize aortic manipulation.

Advances in imaging may also support the implementation of targeted neuroprotective measures. For example, identification of extensive aortic plaque or severe calcification could influence the use of embolic protection devices during transcatheter procedures.^{31,69} Similarly, detection of prosthetic valve abnormalities or intracardiac thrombi may guide optimization of antithrombotic therapy prior to intervention.

The integration of cardiac imaging with cerebral imaging techniques represents another potential direction. Combining pre-procedural structural assessment with post-procedural neuroimaging may enhance understanding of the relationship between embolic substrates and resulting cerebral lesions.^{25,72} Such multimodal approaches could contribute to improved risk stratification and inform the development of preventive strategies.

As neurocardiology continues to evolve, imaging-driven prediction models may play an increasingly important role in identifying patients who are susceptible to SCI. These advances may ultimately support a transition toward more personalized procedural planning and improved neurological outcomes.

10. Clinical implications

Although SCI does not produce immediate neurological symptoms, accumulating evidence suggests that its long-term clinical impact may be significant.³ Repeated subclinical ischemic events have been associated with cognitive decline, impaired executive function, and an increased risk of future overt stroke.¹⁷ In this context, SCI should not be regarded as a benign procedural byproduct but rather as a marker of cumulative neurovascular vulnerability that may influence long-term outcomes after cardiac intervention.

The growing recognition of SCI following cardiac interventions has important implications for clinical

practice. Traditionally, procedural success has been defined by hemodynamic outcomes and the absence of overt neurological complications. However, the high incidence of subclinical ischemic lesions suggests that neurological injury may occur even in the absence of clinically evident stroke.⁷³ This observation underscores the need to expand the concept of procedural safety to include neurovascular integrity.

Advanced cardiac imaging may contribute to this shift by identifying structural features associated with embolic risk before intervention. Detection of complex aortic plaques, intracardiac thrombi, or prosthetic abnormalities could allow clinicians to anticipate vulnerability to cerebral injury and adjust procedural strategies accordingly.^{60,74} Such considerations may influence decisions regarding access routes, device selection, or the use of adjunctive protective measures.

Incorporating imaging findings into pre-procedural evaluation may therefore support a more comprehensive risk assessment framework. Rather than relying solely on clinical predictors, combining structural and procedural information could improve the identification of patients at increased risk of SCI. This approach aligns with the broader movement toward individualized procedural planning and may ultimately contribute to improved long-term neurological outcomes.

11. Conclusion

Silent cerebral injury has emerged as a frequent yet underrecognized consequence of contemporary cardiac interventions. Although these lesions often occur without immediate neurological symptoms, accumulating evidence suggests that their long-term impact on cognitive function and cerebrovascular health may be clinically meaningful. The traditional focus on overt stroke does not fully capture the spectrum of neurological injury associated with procedural manipulation of cardiac and aortic structures.

Advances in cardiac imaging provide an opportunity to move beyond reactive management toward anticipatory risk assessment. Among currently available modalities, TEE represents the most immediately applicable technique for routine clinical integration because of its ability to detect complex aortic plaques, intracardiac thrombi, and prosthetic valve abnormalities in real time during procedural planning. Cardiac CT also provides valuable complementary information regarding aortic and valvular calcific burden and may further support procedural risk stratification in selected patients. Incorporating these findings into procedural planning may help identify individuals at increased risk of SCI.

Greater integration between cardiovascular imaging and neurovascular outcomes represents an evolving area within neurocardiology. As understanding of the mechanisms underlying subclinical cerebral injury continues to improve, imaging-guided approaches may support more individualized procedural strategies and contribute to the reduction of neurological complications following cardiac interventions.

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