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Research Article

Effect of Kidney Dysfunction on Disease Mechanisms and Outcomes in Acute Ischemic Stroke Managed with Endovascular Thrombectomy

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Abstract:

Background

Endovascular thrombectomy (EVT) has revolutionized the treatment of acute ischemic stroke (AIS), offering improved recanalization and functional outcomes. However, patient heterogeneity especially the presence of renal dysfunction remains a critical determinant of EVT success. Renal impairment is common in stroke populations and may negatively affect short-term post-procedural outcomes, yet it remains under-investigated in EVT-specific protocols.

Objective

This review aims to synthesize current evidence on the pathophysiological, clinical, and procedural implications of renal dysfunction in patients undergoing EVT for AIS and to highlight management considerations and future research directions.

Methods and Scope

We provide an overview of renal dysfunction classifications (acute kidney injury and chronic kidney disease), their prevalence in stroke cohorts, and the shared vascular and inflammatory pathways that link renal impairment with adverse cerebrovascular outcomes. Emphasis is placed on periprocedural risks such as contrast-induced nephropathy and hemodynamic instability and their contribution to delayed recovery, increased in-hospital mortality, hemorrhagic

transformation, and prolonged ICU stays. We summarize key clinical studies, including retrospective analyses and available meta-analyses.

Results

Renal dysfunction is consistently associated with poorer neurological outcomes and increased post-EVT complications. Mechanisms include altered drug metabolism, systemic inflammation, and endothelial dysfunction.

Conclusion

Renal dysfunction should be recognized as a vital component in EVT risk stratification. Integrated, multidisciplinary management and personalized care approaches are essential. Future directions include the use of renal biomarkers, AI-driven predictive modeling, and renal-specific EVT protocols to optimize outcomes for this high-risk subgroup.

keywords: ischemic stroke; mechanical thrombectomy; renal dysfunction; acute kidney injury; prognosis the influence of renal dysfunction on pathophysiology and clinical outcomes in patients with acute ischemic stroke treated with endovascular thrombectomy

Introduction

Acute ischemic stroke (AIS) remains one of the most devastating neurological emergencies worldwide, characterized by the sudden loss of cerebral blood flow due to arterial occlusion. It accounts for approximately 85% of all stroke cases and is a leading cause of disability and death globally [1]. The burden is especially profound in low- and middle-income countries, where timely access to specialized stroke care is often limited. In recent years, endovascular thrombectomy (EVT) has revolutionized the treatment landscape for AIS, particularly in patients with large vessel occlusion (LVO). Following the success of pivotal trials such as MR CLEAN, ESCAPE, and REVASCAT, EVT has been firmly established as a standard of care in eligible patients, offering significantly improvement in functional outcomes compared to best medical therapy alone [2–4]. These interventions, when performed within defined time windows, have shown remarkable efficacy in achieving recanalization and improvement in neurological recovery. Despite these advancements, not all patients benefit equally from EVT. Growing attention is now being directed toward understanding how pre-existing comorbidities influence the efficacy and safety of this intervention. Among various systemic factors, conditions such as hypertension, diabetes mellitus, atrial fibrillation, and renal dysfunction are emerging as key determinants of post-thrombectomy outcomes [5]. The complexity of managing stroke in patients with multiple comorbidities necessitates a more nuanced and individualized approach to EVT. Renal dysfunction, in particular, has gathered increasing interest in this context. Both acute kidney injury (AKI) and chronic kidney disease (CKD) are prevalent in AIS populations and have been independently associated with worse clinical outcomes, including increased mortality rates, hemorrhagic transformation, and prolonged hospitalization [6, 7]. The bidirectional pathophysiological relationship between cerebrovascular and renal systems often referred to as the "brain-kidney axis" adds another layer of complexity to the management of these patients [8]. This review aims to explore the impact of renal dysfunction on short-term outcomes after EVT in AIS. By examining current literature, underlying mechanisms, and clinical implications, we hope to provide clinicians and researchers with a deeper understanding of how renal impairment may alter the course and prognosis of EVT-treated stroke patients. We also highlight existing knowledge gaps and propose future directions for research and practice in this evolving field.

2. Understanding Renal Dysfunction

2.1 Definitions and Classifications

Renal dysfunction represents a spectrum of disorders affecting the kidneys' ability to filter blood, regulate fluid and electrolyte balance, and eliminate waste products. Among these, acute kidney injury (AKI) and chronic kidney disease (CKD) are the most clinically significant and commonly encountered in both general and neurologic populations. AKI is characterized by a rapid decline in kidney function occurring over hours to days. It is often triggered by hemodynamic instability, nephrotoxic agents, or systemic insults such as sepsis. The Kidney Disease: Improving Global Outcomes (KDIGO) guidelines define AKI based on a rise in serum creatinine ≥0.3 mg/dL within 48 hours, or a 1.5-fold increase from baseline within 7 days, or urine output <0.5 mL/kg/h for six hours [9]. In the context of AIS, the use of contrast-enhanced procedures like endovascular thrombectomy (EVT) increases the risk of AKI. In contrast, CKD refers to long-standing kidney damage or reduced kidney function that persists for more than three months. It is classified based on estimated glomerular filtration rate (eGFR) and the presence of kidney damage indicators such as proteinuria. According to KDIGO staging, CKD is divided into five stages: Stage 1 (eGFR ≥90 mL/min/1.73 m² with evidence of kidney damage), through to Stage 5 (eGFR <15 mL/min/1.73 m²), which signifies end-stage renal disease (ESRD) (Table 1) [10]. Patients with advanced CKD often exhibit vascular, inflammatory, and metabolic disturbances that may influence cerebrovascular outcomes. The eGFR remains the most widely used clinical tool for estimating renal function. It is derived from serum creatinine using equations such as CKD-EPI or MDRD, accounting for variables like age, sex, and race. While convenient, eGFR has limitations in acute settings, especially when renal function is fluctuating or serum creatinine is not in steady state [11].

Stage	eGFR (mL/min/1.73 m²)	Description
1	≥90	Normal or high GFR with kidney damage
2	60–89	Mildly decreased GFR
3a	45–59	Mild to moderate decrease
3b	30–44	Moderate to severe decrease
4	15–29	Severe decrease
5	<15	Kidney failure (end-stage renal disease)

Table 1: KDIGO Classification of Chronic Kidney Disease (CKD).

Source: KDIGO 2012 Clinical Practice Guidelines [10]

2.2 Prevalence of Renal Dysfunction in Stroke Patients

Renal dysfunction is frequently observed in patients presenting with AIS. Studies estimate that up to 30–40% of stroke patients have some degree of CKD, with higher rates observed in those with large vessel atherosclerosis or cardioembolic sources [12, 13]. In addition to CKD, acute kidney injury (AKI) is also a common complication, occurring in approximately 13–36% of AIS patients, particularly in those undergoing procedures involving contrast exposure or with underlying risk factors like diabetes or heart failure [14] Even mild reductions in renal function have been independently associated with worse stroke outcomes, including increased mortality and poor functional recovery. Several factors contribute to this overlap between stroke and renal impairment. Both conditions share common vascular risk factors, including hypertension, diabetes mellitus, dyslipidemia, and smoking [15]. These shared etiologies create a synergistic burden on the cerebral and renal vasculature, predisposing individuals to arterial stiffness, endothelial dysfunction, and accelerated atherosclerosis. Additionally, systemic inflammation, oxidative stress, and prothrombotic states are heightened in both diseases, further amplifying the risk of adverse outcomes [16]. Importantly, renal dysfunction not only predisposes to stroke but also complicates its management. Patients with impaired renal function are at increased risk of contrast-induced nephropathy during imaging or interventional procedures, and they may be less tolerant of medications commonly used in stroke management, such as anticoagulants and antiplatelets [17]. This necessitates careful monitoring and a multidisciplinary approach to optimize care in this high-risk group.

3. Pathophysiological Links Between Renal Dysfunction and Stroke

The intricate relationship between the kidneys and the brain goes beyond shared vascular risk factors it is underpinned by complex pathophysiological mechanisms that interconnect renal dysfunction with cerebrovascular injury. Understanding these mechanisms is essential, especially in the context of AIS, as renal impairment can amplify brain damage and influence recovery. The “brain–kidney axis” illustrates this multidirectional interaction through several overlapping pathways.

3.1 Endothelial Dysfunction

One of the earliest and most significant contributors to both renal and cerebral vascular disease is endothelial dysfunction. In chronic kidney disease (CKD), there is a marked reduction in the bioavailability of nitric oxide (NO), a key vasodilator, alongside elevated levels of endothelin-1, a potent vasoconstrictor [18]. These changes compromise vascular tone and promote thrombogenesis [18]. The cerebral endothelium, responsible for regulating blood flow to neuronal tissue, becomes similarly impaired in patients with renal dysfunction, increasing the risk of ischemic events [19]. Moreover, uremic toxins such as asymmetric dimethylarginine (ADMA) further inhibit NO synthesis and damage vascular integrity [20].

3.2 Inflammation and Oxidative Stress

Chronic systemic inflammation and oxidative stress are hallmarks of both CKD and AKI. Elevated levels of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) are frequently observed in patients with renal impairment and have been directly linked to stroke severity and poor neurological outcomes [21]. These inflammatory mediators disrupt endothelial function, attract leukocytes, and promote thrombosis. Furthermore, oxidative stress contributes to lipid peroxidation and DNA damage in cerebral cells, exacerbating the ischemic injury [22]. This inflammatory cascade is not only a trigger for stroke but also a driver of secondary brain injury post-stroke.

3.3 Impaired Cerebral Autoregulation

Cerebral autoregulation refers to the brain's ability to maintain consistent cerebral blood flow despite fluctuations in systemic blood pressure. In patients with renal dysfunction, this autoregulatory mechanism is often compromised. Studies have shown that CKD patients exhibit blunted cerebrovascular reactivity and increased cerebral pulsatility, both of which predispose to hypoperfusion or hyperperfusion during acute ischemic events [23]. Autoregulatory failure may contribute to increased infarct size and limited protective capacity of collateral circulation, especially during interventions like EVT.

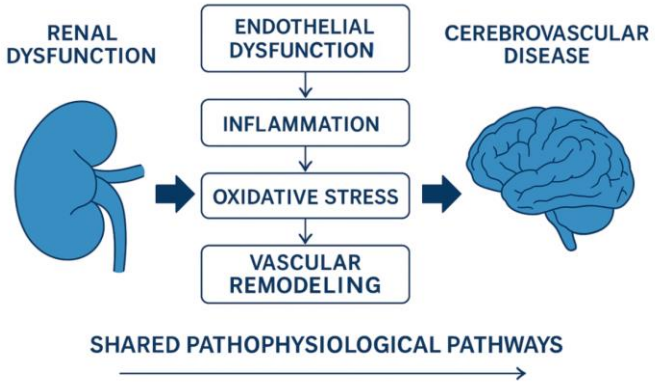
3.4 Atherosclerosis and Microangiopathy

Renal and cerebrovascular disease are shared consequences of atherosclerosis In CKD, there is an accelerated form of vascular calcification and arterial stiffness, even in younger individuals, leading to early-onset large vessel disease [24]. At the same time, microangiopathy damage to small cerebral vessels is prevalent, particularly in advanced CKD and diabetic nephropathy. This microvascular injury results in white matter changes, lacunar infarctions, and cerebral small vessel disease, all of which are associated with poorer functional recovery after stroke [25]. These vascular pathologies not only increase the risk of initial stroke but also worsen prognosis after reperfusion therapy.

3.5 Effects on Blood–Brain Barrier Integrity

The blood–brain barrier (BBB) is a specialized structure that regulates the movement of substances between the blood and the central nervous system. Renal dysfunction impairs BBB integrity through multiple mechanisms, including uremic toxin accumulation, oxidative damage, and systemic inflammation [26]. Disruption of the BBB allows crossing of inflammatory cells and neurotoxic molecules into brain tissue, contributing to cerebral edema and hemorrhagic transformation after

ischemic events. This is particularly concerning in EVT candidates, where BBB compromise may increase the risk of post-procedural complications such as symptomatic intracerebral hemorrhage [27].



4. Renal Dysfunction and Outcomes after Endovascular Thrombectomy (EVT)

4.1 Periprocedural Risks

Patients with renal dysfunction face several periprocedural complications during EVT for AIS. One prominent concern is contrast-induced nephropathy (CIN), a form of AKI triggered by iodinated contrast agents used during imaging and EVT. Although the incidence of CIN in general stroke populations is relatively low, patients with pre-existingCKD), diabetes mellitus, or dehydration are at significantly higher risk [17]. Studies have shown that CIN can occur in up to 10–15% of stroke patients with baseline eGFR <60 mL/min/1.73 m², potentially worsening kidney function and prolonging hospitalization [28]. Another important consideration is hemodynamic instability, particularly in those with advanced renal disease. CKD and ESRD are often associated with impaired autonomic function and volume dysregulation, which can lead to hypotension during anesthesia or procedural stress [29]. Such instability can compromise cerebral perfusion during EVT, increasing infarct volume and negatively affecting neurological outcomes. Moreover, delayed recovery is commonly observed in patients with renal dysfunction undergoing EVT. This may be partly attributable to systemic inflammation, metabolic derangements, and reduced physiological reserve. These patients are also more likely to have complications such as infection or fluid overload post-procedure, which may delay neurological rehabilitation and extend intensive care needs [30].

4.2 Impact on Short-Term Outcomes

Renal dysfunction has a tangible effect on short-term functional outcomes after EVT. Neurological scoring systems such as the National Institutes of Health Stroke Scale (NIHSS) and the modified Rankin Scale (mRS) consistently show worse outcomes in patients with impaired kidney function. For instance, reduced eGFR levels were significantly associated with worse functional outcomes after stroke. Specifically, patients with CKD had a 1.54-fold higher risk of poor outcome (mRS >2) at 3 months (adjusted OR: 1.54; 95% CI: 1.33–1.79; $p < 0.001$) compared to those with normal renal function. Additionally, lower eGFR was associated with higher NIHSS scores at discharge, indicating more severe neurological deficits [25]. In-hospital mortality is also significantly higher among EVT-treated stroke patients with CKD.. In a large multicenter cohort study, patients with CKD had a significantly higher in-hospital mortality rate (10.4%) compared to those with normal renal function (5.8%; $P < 0.001$). After adjusting for age, stroke severity, and comorbidities, CKD remained an independent predictor of mortality (adjusted odds ratio 1.82; 95% CI, 1.33–2.50; $P < 0.001$) [31]. This trend is particularly pronounced in patients requiring dialysis, who have limited physiologic reserve and higher baseline cardiovascular risk. Renal dysfunction also appears to increase the risk of hemorrhagic transformation, a feared complication of reperfusion therapy. Endothelial fragility, altered coagulation profiles, and uremic platelet dysfunction may contribute to this heightened risk. In a retrospective cohort analysis, advanced CKD was linked with an increased incidence of both symptomatic and asymptomatic intracranial hemorrhage post-EVT [32]. Finally, renal impairment often correlates with prolonged ICU and hospital stays. This is due not only to the direct effects of kidney dysfunction but also to the increased burden of medical management, including dialysis, fluid/electrolyte monitoring, and complication management. Patients with CKD frequently require extended rehabilitation services and are more likely to be discharged to skilled nursing facilities rather than home [33].

4.3 Evidence from Clinical Studies

A growing body of clinical research supports the association between renal dysfunction and poor outcomes post-EVT. Patients with eGFR <60 mL/min/1.73 m² had significantly higher 3-month mortality and worse functional outcomes after thrombectomy compared to those with preserved renal function [34]. Similarly moderate-to-severe CKD predicted worse mRS scores and higher in-hospital complication rates . Even mild reductions in eGFR were associated with delayed neurological recovery and increased ICU admission rates following EVT [14, 35]. These findings underscore the prognostic relevance of kidney function in the acute stroke care continuum. Several meta-analyses have further validated this link. CKD was independently associated with a lower likelihood of achieving functional independence (mRS ≤2) and a higher risk of mortality after EVT for AIS [36]. Importantly, the adverse effect of renal dysfunction persisted regardless of age, stroke etiology, or collateral status. When comparing EVT outcomes in patients with and without renal dysfunction, the differences are striking. In a pooled analysis of three tertiary care centers, patients with CKD had a 20% lower chance of good outcome (mRS 0–2) and a 2.5-fold higher risk of in-

hospital death than those with normal renal function, even after controlling for stroke severity and procedural variables [37].

5. Mechanisms by Which Renal Dysfunction May Affect EVT Outcomes

The interaction between renal dysfunction and clinical outcomes after endovascular thrombectomy (EVT) in acute ischemic stroke (AIS) is complex and multifactorial. Several underlying mechanisms have been proposed that may explain why patients with impaired renal function are more likely to experience complications and suboptimal recovery after EVT (Table 2).

5.1 Altered Pharmacokinetics of Drugs

Renal dysfunction significantly affects the pharmacokinetics and pharmacodynamics of commonly used medications in stroke management, including sedatives, antihypertensives, anticoagulants, and antiplatelets. Reduced glomerular filtration impairs drug clearance, leading to drug accumulation and toxicity, or alternatively, subtherapeutic effects due to altered protein binding or metabolism [38]. For example, renally cleared anticoagulants like low molecular weight heparin or direct oral anticoagulants may accumulate in CKD patients, increasing the risk of bleeding during or after EVT [39]. In contrast, drugs such as clopidogrel may be less effective in uremic patients due to impaired platelet function and altered drug metabolism, potentially increasing the risk of re-occlusion or stent thrombosis [40]. Careful dose adjustment and monitoring are essential in this group, but emergency EVT scenarios often lack the luxury of time, thereby compounding risk.

Mechanism	Description
Altered pharmacokinetics	Drug accumulation or reduced efficacy due to impaired renal clearance
Systemic inflammation	Elevated cytokines (e.g., IL-6, CRP) worsen neurovascular injury
Coagulation abnormalities	Uremia-induced platelet dysfunction; bleeding and thrombotic risks
Impaired vascular healing	Endothelial dysfunction and impaired endothelial progenitor cell (EPC) repair
Immunosuppression	Higher risk of post-stroke infections (e.g., pneumonia, UTI, sepsis)

Table 2: Mechanisms Linking Renal Dysfunction to Poor EVT Outcomes.

Sources: [20, 42-47]

5.2 Increased Systemic Inflammation

Chronic kidney disease is known to be a pro-inflammatory state, marked by elevated levels of C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), even in the absence of acute infection [20]. This systemic inflammation accelerates vascular injury, promotes plaque instability, and compromises neurovascular repair following EVT. Inflammatory cytokines can impair collateral circulation, reduce neuroplasticity, and enhance BBB permeability, making reperfusion more harmful than beneficial in some cases [41]. These factors together may explain why renal dysfunction is independently associated with worse neurological recovery and higher rates of hemorrhagic transformation after EVT.

5.3 Coagulation Abnormalities

Patients with renal dysfunction, particularly those in advanced stages or on dialysis, often display coagulation abnormalities. Uremia impairs platelet aggregation and function, leading to a paradoxical state of both bleeding and thrombosis [42]. Altered levels of fibrinogen, D-dimer, and von Willebrand factor are commonly observed, making periprocedural management of EVT complex and risky. In the context of AIS and EVT, this imbalance may increase the risk of intracranial hemorrhage post-recanalization or impair control of thrombus formation, thus impacting recanalization success and recovery [43]. Furthermore, the use of intravenous thrombolysis prior to EVT in patients with renal dysfunction adds another layer of bleeding risk.

5.4 Impaired Vascular Healing

Effective vascular healing after EVT is essential for maintaining vessel patency, reducing re-occlusion risk, and preventing complications such as dissection or aneurysm formation. However, in CKD patients, endothelial dysfunction and a pro-oxidative environment hinder vascular repair mechanisms [44]. Uremic toxins like indoxyl sulfate and p-cresyl sulfate have been shown to directly impair endothelial progenitor cell (EPC) function, which is crucial for endothelial regeneration following vascular injury [45]. As a result, the damaged cerebral vasculature in renal patients may not heal effectively post-thrombectomy, increasing the risk of complications such as intracranial hemorrhage and delayed neurological recovery.

5.5 Immunosuppression and Infection Risk

Finally, immunosuppression in CKD is a well-established phenomenon, often described as “uremic immunodeficiency.” These patients exhibit impaired leukocyte function, reduced chemotaxis, and decreased phagocytic activity, predisposing them to a higher incidence of nosocomial infections, including pneumonia, urinary tract infections, and bloodstream infections following EVT [46]. Infections are a major contributor to early neurological deterioration and prolonged hospitalization in stroke patients, especially those in intensive care units (ICUs). Moreover, the systemic inflammatory response triggered by infections may exacerbate cerebral edema, increase infarct size, and diminish the benefits of successful recanalization [47].

6. Clinical Implications and Management Considerations

The intersection of renal dysfunction and AIS management particularly in the setting of EVT demands careful clinical decision-making. Since patients with impaired renal function face both heightened procedural risks and worse short-term outcomes, individualized strategies must be employed to optimize safety and therapeutic benefit.

6.1 Patient Selection for EVT

Evaluating renal function pre-procedure is essential when considering EVT. Baseline renal assessment, particularly the estimated glomerular filtration rate (eGFR), helps identify patients at increased risk of contrast-induced nephropathy, bleeding, and poor neurological outcomes. According to the KDIGO guidelines, an eGFR <60 mL/min/1.73 m² indicates significant renal impairment and warrants caution in procedural

planning [10]. Risk-benefit stratification should integrate not only stroke severity and imaging findings but also renal status, comorbidities, and expected life expectancy. Studies have shown that although patients with CKD have poorer outcomes post-EVT, the procedure may still offer net benefits in terms of survival and functional independence if carefully selected [34,48]. Therefore, renal dysfunction should not be viewed as an absolute contraindication but rather as a high-risk marker requiring tailored care.

6.2 Periprocedural Management

Hydration strategies are the cornerstone of renal protection in interventional procedures. Isotonic saline administered prior to and after EVT can help reduce the risk of CIN, especially in patients with pre-existing CKD. The use of sodium bicarbonate and N-acetylcysteine has been explored, though data remain mixed [49]. Minimizing contrast use is another key strategy. Many modern thrombectomy techniques allow successful recanalization with limited or even no contrast if roadmaps and non-contrast CT imaging are optimally used. Moreover, the adoption of low-osmolar or iso-osmolar contrast agents has been shown to reduce nephrotoxicity in at-risk patients [50].

Monitoring and managing AKI is crucial in the hours to days following EVT. Serial serum creatinine, urine output monitoring, and biomarkers like neutrophil gelatinase-associated lipocalin (NGAL) can provide early warning signs of renal deterioration [51]. If AKI develops, timely nephrology consultation and supportive measures such as fluid balance correction, electrolyte management, and avoidance of nephrotoxic drugs can prevent progression to severe renal failure.

6.3 Post-EVT Monitoring and Care

Following EVT, renal support remains a critical aspect of comprehensive post-stroke care, especially in patients with pre-existing CKD or perioperative AKI. This includes careful fluid management, judicious use of diuretics, and renal dose adjustment of commonly used medications like antibiotics, anticoagulants, and sedatives [52]. A multidisciplinary stroke care model that includes nephrologists, neurologists, critical care physicians, and rehabilitation specialists improves outcomes for this vulnerable subgroup. Studies have demonstrated that integrated stroke units with renal consultation services lead to earlier detection of complications and more precise intervention planning [53]. Lastly, early identification of clinical deterioration neurologic or renal is essential. Tools like the mRS and the NIHSS should be complemented with renal surveillance indicators. Deterioration may manifest subtly through worsening cognitive status, electrolyte imbalances, or fluid overload. Timely action can prevent secondary insults that may compromise recovery.

7. Limitations in Current Literature and Knowledge Gaps

Despite increasing awareness of the intersection between renal dysfunction and outcomes after endovascular thrombectomy (EVT) for acute ischemic stroke (AIS), significant gaps persist in our current understanding. These limitations span definitional inconsistencies, trial design shortcomings, and lack of focused protocols hindering the development of evidence-based strategies for this high-risk population.

7.1 Heterogeneity in Definitions of Renal Dysfunction

One of the most prominent challenges is the lack of standardized definitions for renal dysfunction across stroke studies. Terms such as CKD, AKI, and reduced eGFR are often used interchangeably or with varying thresholds. Some studies define CKD using an eGFR cutoff of <60 mL/min/1.73 m², while others incorporate proteinuria, serum creatinine, or staging from KDIGO without consistency [54,55]. This variability makes it difficult to draw uniform conclusions or perform valid meta-analyses. Moreover, the classification of renal dysfunction severity is inconsistently applied. For example, patients with mild renal impairment may be grouped with those in advanced renal failure, despite their vastly different risks and outcomes. This definitional heterogeneity introduces bias and limits generalizability across clinical studies.

7.2 Lack of Renal-Specific EVT Protocols

Although EVT has become a gold-standard intervention for large vessel occlusion strokes, renal-specific considerations are often absent from standard protocols. Unlike contrast-based procedures in cardiology or nephrology where preventive strategies for AKI are routine, neurointerventional guidelines rarely include specific precautions for patients with reduced renal reserve [56]. There is limited consensus on optimal hydration regimens, contrast agent choice, or thresholds for withholding EVT in patients with severe renal dysfunction. Most EVT guidelines such as those from the American Heart Association/American Stroke Association focuses on neurological and imaging criteria, without integrating renal parameters into decision-making algorithms [57]. This gap highlights the need for protocols that consider the delicate renal-neurologic interplay.

7.3 Underrepresentation in Trials

Another major shortcoming is the underrepresentation of patients with renal dysfunction in major EVT trials. Landmark studies like MR CLEAN, EXTEND-IA, and DAWN excluded or poorly reported data on individuals with CKD or elevated creatinine levels [2, 58,59]. Consequently, the external validity of these trials to real-world populations where renal dysfunction is common, especially among older patients is questionable. This underrepresentation is often due to concerns over contrast toxicity, bleeding risk, or poor prognosis, leading to exclusion either explicitly or implicitly during recruitment. As a result, clinicians are left to extrapolate findings from healthier cohorts, which may not accurately reflect outcomes in patients with significant renal comorbidity.

7.4 Need for Prospective Studies

While retrospective studies and subgroup analyses have begun to shed light on the impact of renal impairment in EVT-treated patients, well-designed prospective studies are urgently needed. These should focus on both safety and efficacy outcomes, stratified by renal function categories, and include predefined renal endpoints such as AKI incidence, contrast nephropathy, and renal-related morbidity. Furthermore, randomized controlled trials (RCTs) incorporating renal-protective strategies like low-contrast protocols, hydration interventions, or contrast-free imaging could provide evidence for tailored management in this subgroup. Until such data is available, clinical practice must rely on limited observational evidence and extrapolation from other disciplines.

8. Future Directions

The complex interplay between renal dysfunction and EVT outcomes in AIS patients underscores the pressing need for more personalized and

integrative approaches in stroke care. While current evidence provides valuable insights, several promising avenues are emerging that could transform how we assess, manage, and predict outcomes in patients with renal impairment undergoing EVT.

8.1 Personalized EVT Strategies

One of the most compelling directions for the future is the personalization of EVT strategies. Current treatment algorithms are largely standardized and do not sufficiently account for the nuanced risks associated with comorbid renal dysfunction. A more individualized approach factoring in renal function, hemodynamic stability, contrast load capacity, and patient frailty could better balance procedural benefit and systemic risk. This paradigm shift is supported by emerging evidence in broader stroke care, where tailored interventions based on patient phenotypes have shown potential in improving outcomes [60]. Studies suggest that personalized EVT protocols may include using alternative imaging (e.g., non-contrast MR angiography), ultra-low contrast strategies, and selective use of adjunctive therapies like antiplatelets or anticoagulants based on renal and bleeding risk profiles [61].

8.2 Integration of Renal Biomarkers in Stroke Care

Another promising area is the integration of renal biomarkers into routine stroke management. Traditional markers such as serum creatinine and eGFR may not provide an early or sensitive indication of acute renal injury in EVT candidates. Biomarkers like neutrophil gelatinase-associated lipocalin (NGAL), cystatin C, and kidney injury molecule-1 (KIM-1) have shown potential in the early detection of subclinical AKI and predicting renal outcomes after contrast exposure [51,62]. Incorporating these markers into pre-procedural risk stratification tools could help clinicians identify high-risk individuals earlier and implement renal-protective strategies proactively. Furthermore, longitudinal biomarker tracking might offer a dynamic measure of renal status during the post-EVT recovery phase.

8.3 AI and Predictive Modeling in EVT Outcomes

The application of artificial intelligence (AI) and predictive modeling in stroke care is growing rapidly, and renal dysfunction could become a key variable in these models. Machine learning algorithms that synthesize clinical data, imaging features, and lab values (including renal indices) are being developed to predict EVT success, hemorrhagic transformation, and functional recovery [63]. In one recent study, an AI model incorporating renal function data alongside perfusion metrics and procedural parameters demonstrated improved accuracy in predicting 90-day functional outcomes compared to traditional logistic regression models [64]. Such tools can aid in refining patient selection, guiding real-time intraoperative decisions, and informing discussions with patients and families about prognosis.

8.4 Clinical Trials Focusing on Renal Dysfunction in Stroke

To date, few prospective trials have specifically focused on patients with renal dysfunction undergoing EVT, representing a significant knowledge gap. Most large stroke trials have either excluded this population or failed to analyze renal status as a stratifying variable. There is a pressing need for dedicated randomized controlled trials that evaluate EVT safety, efficacy, and optimal management strategies in individuals with CKD or AKI. Future studies could explore modified EVT protocols for CKD patients, such as contrast-sparing techniques, hydration optimization, or the use of renal-protective pharmacologic agents. Equally important are observational registries that capture real-world data on EVT outcomes in renal-impaired populations across different ethnicities, healthcare systems, and resource settings [65].

9. Conclusion

Renal dysfunction has emerged as a significant yet often underappreciated factor influencing the outcomes of EVT in AIS patients. Across multiple studies, reduced renal function whether chronic or acute has consistently been linked to poorer short-term neurological outcomes, increased rates of hemorrhagic transformation, prolonged hospital stays, and elevated in-hospital mortality. These associations are not merely coincidental but rooted in shared vascular pathologies, systemic inflammation, coagulation disturbances, and altered pharmacokinetics that jointly shape the procedural risk and recovery trajectory of stroke patients. Given the multifaceted impact of impaired renal function, a one-size-fits-all approach to EVT is no longer sufficient. The future of stroke care must be rooted in individualized treatment strategies, where renal parameters are not just noted but actively incorporated into patient selection, procedural planning, and post-EVT care. Such personalization could mean adjusting contrast dosages, implementing preemptive renal-protective measures, or even reconsidering EVT in patients with severe renal compromise unless clearly indicated. Equally vital is the call for an integrated renal-neurovascular management model, bringing together stroke neurologists, interventionalists, nephrologists, and critical care specialists to collaboratively guide decision-making. This multidisciplinary coordination is particularly important in the periprocedural and post-procedural phases, where fluctuations in renal function can have cascading effects on cerebral perfusion, recovery, and survival. Ultimately, improving outcomes for this vulnerable population will depend not only on refining EVT techniques but also on redefining how we understand and manage comorbidities like renal dysfunction in the broader context of cerebrovascular disease. Through further research, dedicated trials, and the adoption of evidence-based, renal-aware protocols, we can move closer to delivering safer, more equitable, and more effective care to all stroke patients.

Declarations

Consent for publication

All participants and coauthors provided consent for the publication of the data and the required information.

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Author contributions

All the authors contributed to this study and approved the final manuscript. A.V.C., M.F., and A.Y.C. conceived the study concept and design. A.Y.C., S.A., and S.S. drafted the initial manuscript. S.D., N.S., P.D., S.K.Y., P.G., P.R.S., and R.K.Y. contributed to data collection and

literature review. W.E. and A.V.C. provided critical revisions and expert input on neurointerventional perspectives. S.D. contributed to nephrology-related content and interpretation. All authors contributed to manuscript editing, reviewed the final version critically for important intellectual content, and approved the submitted manuscript.

Declarations of competing interest

The authors declare that they have no conflicts of interest.

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