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Review Article

Renal Vein Thrombosis: Causes, Pathophysiology, Management, Emerging Therapies, and the Role of Artificial Intelligence—A Narrative Review

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ABSTRACT

Renal vein thrombosis (RVT) is an uncommon but clinically important condition that arises most often in nephrotic syndrome (NS) and in association with malignancy—particularly renal cell carcinoma with venous tumor extension—inherited or acquired thrombophilias, and, more recently, COVID-19–associated coagulopathy. Because RVT is rare and no randomized trials address it, practice is largely extrapolated from the broader venous thromboembolism (VTE) and chronic kidney disease (CKD) literature. Its pathophysiology follows Virchow's triad, with hypercoagulability predominating in NS through urinary loss of antithrombin and other regulatory proteins. Anticoagulation remains the cornerstone of treatment: direct oral anticoagulants (DOACs) offer a more favorable benefit–risk profile than vitamin K antagonists in early-stage CKD, whereas evidence in dialysis-dependent patients is insufficient, and management should be individualized to renal function and bleeding risk. Catheter-directed therapy, etiology-directed oncologic treatment, and factor XI inhibitors are promising adjuncts. Artificial intelligence tools for VTE risk prediction, ultrasound diagnosis, and anticoagulation dosing show encouraging performance but remain unvalidated in RVT; disease-specific cohorts and prospective validation are essential before they can be adopted.

Key words: Renal vein thrombosis, venous thromboembolism, nephrotic syndrome, direct oral anticoagulants, renal cell carcinoma, artificial intelligence; chronic kidney disease

INTRODUCTION

Renal vein thrombosis (RVT) is an uncommon vascular disorder defined by the formation of a thrombus within one or both renal veins. Depending on the acuity and completeness of venous occlusion, RVT may manifest as acute kidney injury (AKI), progressive renal impairment, flank pain and hematuria, or it may remain clinically silent and be discovered incidentally on imaging. Pulmonary embolism (PE) is a recognized complication when thrombus propagates or embolizes. [1]

RVT is most strongly associated with hypercoagulable states, and historically the dominant association has been with nephrotic syndrome (NS). In older prospective series using systematic venography, RVT was detected in a substantial minority of adults with NS—in 33 of 151 patients (≈22%) in one foundational series, with membranous nephropathy

predominating. [1] Larger contemporary cohorts confirm that the risk of venous thromboembolism (VTE) is markedly increased and is highest in membranous nephropathy. [2,3] Malignancy, in particular renal cell carcinoma (RCC) with direct venous tumor extension, accounts for a substantial further proportion of cases, and a venous tumor thrombus involving the renal vein is reported in approximately 10% to 18% of patients with RCC. [4,5] Since 2020, COVID-19 has been described as an additional precipitant through its associated prothrombotic state, although reported renal venous involvement is largely confined to case reports and small series. [6,7]

Because RVT is rare, no randomized controlled trials address its management, and contemporary practice is necessarily informed by indirect evidence from the wider VTE and CKD literature. The aim of this review is therefore to synthesize current understanding of the causes, mechanisms, diagnosis, and treatment of RVT; to describe emerging therapies of potential relevance; and to appraise honestly what AI can and cannot yet contribute. Throughout, we distinguish RVT-specific evidence from data extrapolated from related populations.

SCOPE AND APPROACH

A systematic literature search was conducted across PubMed, Embase, Scopus, and Google Scholar from January 1, 2015, to December 31, 2025, using the Boolean search string ("renal vein thrombosis" OR "renal venous thrombosis" OR "renal vein occlusion") AND ("causes" OR "etiology" OR "pathophysiology" OR "management" OR "anticoagulation" OR "thrombolysis" OR "novel therapy" OR "artificial intelligence" OR "machine learning"), adapted per database syntax. Supplementary targeted searches addressed VTE in chronic kidney disease (CKD), direct oral anticoagulants (DOACs) in renal impairment, and AI applications in VTE. Only peer-reviewed, English-language publications were eligible for inclusion. Older foundational studies identified through reference-list screening were also included where they remain the primary evidence for a specific clinical observation. Priority was given to peer-reviewed primary studies, systematic reviews, and clinical guidance. Where RVT-specific data were unavailable, evidence from cognate populations (general VTE, CKD-associated VTE, and deep vein thrombosis [DVT]) is cited and explicitly identified as extrapolated. Given the rarity of the condition, selected foundational and illustrative case reports are included and are interpreted as hypothesis-generating. This review concerns RVT in adults; neonatal and pediatric RVT, which differs in its predisposing factors, clinical presentation, and management, represents a mechanistically distinct entity and lies outside the scope of this review.

A substantial proportion of the RVT-specific literature cited in this review consists of case reports and small case series, reflecting the rarity of the condition and the absence of randomized controlled trials. [1,5,8] This body of evidence is inherently susceptible to reporting and publication bias and does not permit formal quality or risk-of-bias scoring. Consequently, such sources are treated throughout this review as hypothesis-generating rather than as a basis for firm recommendations, which instead draw where possible on higher-quality evidence from related conditions such as CKD-associated VTE. [9]

EPIDEMIOLOGY

Precise incidence figures for RVT are difficult to establish because many cases are asymptomatic and detection depends on imaging of at-risk patients. In the NS, the reported frequency of RVT varies substantially with the underlying glomerular lesion and the intensity of screening; membranous nephropathy carries a particularly high thromboembolic risk relative to other glomerular diseases. [2,10] Much of this variability likely reflects differences in screening practice rather than true differences in disease frequency: centers that perform routine screening imaging in high-risk NS (e.g., membranous nephropathy with severe hypoalbuminemia) detect a substantially higher proportion of subclinical RVT than centers relying on symptom-triggered imaging, in which only clinically overt cases are identified. [2,10] In a large cohort of patients with NS, the adjusted risk of VTE was several-fold higher in membranous nephropathy than in IgA nephropathy, and the majority of events occurred within the first six months after diagnosis. [2,10,11]

In RCC, venous tumor thrombus is a characteristic feature, reported in roughly 10% to 18% of patients at the level of the renal vein, with extension into the inferior vena cava in a smaller proportion and into the right atrium in approximately 1%. [4,5] Contemporary multicenter cohorts of patients with diagnosed RVT confirm that NS and malignancy together account for the majority of cases and that worsening kidney function is common, underscoring the clinical significance of timely recognition. [5] Outcomes depend chiefly on the underlying cause and the acuity of occlusion: acute, complete, or bilateral thrombosis may precipitate irreversible loss of function, whereas gradual or unilateral disease—in which venous collaterals develop—is often compatible with preserved or recoverable renal function once anticoagulation is established. [5,12]

ETIOLOGY AND RISK FACTORS

The principal causes of RVT are summarized in **Table 1** and discussed in turn below.

Nephrotic Syndrome

NS is the prototypical cause of RVT. The hypercoagulable state results from a multifactorial imbalance of hemostasis: urinary loss of natural anticoagulant and profibrinolytic proteins (notably antithrombin and free protein S), compensatory hepatic over-synthesis of procoagulant factors (including fibrinogen and factors V and VIII), hypoalbuminemia with associated platelet hyper-reactivity, and impaired fibrinolysis. [3,10] The renal veins may be especially susceptible owing to post-glomerular hemoconcentration and locally high concentrations of procoagulant factors. [10,12] The degree of hypoalbuminemia is a consistent predictor of thrombotic risk across histological subtypes. [2,10]

Malignancy and RCC

Malignancy contributes to RVT both through generalized paraneoplastic hypercoagulability and, in the case of RCC, through direct intravascular tumor extension. RCC is distinctive in its propensity to invade contiguous veins, producing a venous tumor thrombus that may extend from the renal vein into the inferior vena cava. [4,5] The optimal anticoagulant

Table 1: Major causes of RVT.

Cause	Principal mechanism	Typical frequency/context	Key references
Nephrotic syndrome	Urinary loss of antithrombin and protein S; raised hepatic procoagulants; hypoalbuminemia and platelet hyperreactivity.	Leading cause; RVT detected in ≈22% of adults with nephrotic syndrome in older venographic series, with membranous nephropathy predominating.	[2,10]
Malignancy/ RCC	Direct venous tumor extension; paraneoplastic hypercoagulability.	Venous tumor thrombus at the renal-vein level in ~10%–18% of RCC.	[5,13]
Thrombophilia	Inherited/acquired hypercoagulable states (antiphospholipid syndrome, factor V Leiden, protein C/S deficiency).	Especially younger patients without other precipitants.	[14]
COVID-19	Endothelial injury, complement activation, inflammatory prothrombotic state.	Reported in case reports and small series; true incidence unknown.	[6,7]
Other	Volume depletion, trauma, extrinsic venous compression.	Less common precipitants.	[5,12]

RVT, renal vein thrombosis; RCC, renal cell carcinoma.

management of bland thrombus accompanying tumor thrombus remains uncertain and is the subject of ongoing systematic evaluation. [13] It should be made explicit that for pure tumor thrombus without a superimposed bland (coagulated) clot, anticoagulation is generally not indicated in the absence of a separate independent indication (e.g., atrial fibrillation), since the thrombus in this setting is structural tumor tissue rather than a coagulation-derived clot and would not be expected to respond to anticoagulant therapy. [13] Anticoagulation becomes relevant only when bland thrombus is confirmed or strongly suspected to coexist with the tumor thrombus.

Thrombophilias and Other Acquired Factors

Inherited and acquired thrombophilias—including antiphospholipid syndrome, factor V Leiden, and deficiencies of protein C or S—are recognized contributors, particularly in younger patients without other identifiable precipitants. [14] Volume depletion, trauma, and extrinsic venous compression by retroperitoneal masses may also provoke RVT. [5,12]

COVID-19–Associated Coagulopathy

SARS-CoV-2 infection is linked with a prothrombotic status driven by endothelial injury, heightened inflammatory background, and complement activation. RVT has been reported in this context, generally in the form of individual case reports and small series describing successful treatment with anticoagulation, including DOACs. [6,7] As with case-report literature generally, these accounts are subject to substantial publication bias, since atypical presentations and cases with favorable treatment outcomes are disproportionately likely to be published. [6,7] True incidence, the spectrum of severity, and outcomes with alternative anticoagulation strategies cannot be reliably estimated from this evidence base, and the associations described here should be interpreted as descriptive rather than causal. The true incidence of RVT among patients with COVID-19 is not established, and reported cases are subject to ascertainment bias. **Table 1** presents the RVT causes, key mechanisms, and epidemiology.

PATHOPHYSIOLOGY

The mechanisms of RVT map onto the three components of Virchow's triad (**Figure 1**). Hypercoagulability predominates in NS through the hemostatic derangements described above. [3,10] Endothelial injury is prominent in RCC, where the tumor invades the venous wall, and in inflammatory states such as COVID-19. Venous stasis may result from extrinsic compression, hypovolemia, or sluggish flow within a tumor-laden vein. [12]

Left-sided RVT is reported more frequently than right-sided disease, an asymmetry attributed to anatomical factors: the left renal vein is longer and crosses anterior to the aorta (occasionally becoming compressed between the aorta and superior mesenteric artery, the so-called nutcracker phenomenon), and it additionally receives the left gonadal vein, both of which promote venous stasis and increased hemodynamic pressure relative to the right side. [12,14] This laterality is consistent with, and adds anatomical specificity to, the stasis component of Virchow's triad described above.

Acute, complete renal venous occlusion produces venous outflow obstruction, renal congestion, interstitial edema, and, in severe cases, hemorrhagic infarction with AKI. More gradual or partial thrombosis permits the development of venous collaterals, which may render the process clinically silent but can also serve as a route for thrombus propagation and PE. [1,12] Recognition of this spectrum—from fulminant bilateral RVT to indolent unilateral disease detected only by proteinuria or incidental imaging—is central to clinical decision-making. **Figure 1** describes the Pathophysiology of RVT mapped to Virchow's triad.

CLINICAL PRESENTATION AND DIAGNOSIS

The clinical presentation of RVT ranges from acute flank pain, hematuria, and deteriorating renal function to an entirely asymptomatic course. [1,12] A high index of suspicion is warranted in patients with nephrotic-range proteinuria (especially membranous nephropathy), active malignancy involving the kidney, or unexplained AKI. Doppler ultrasonography is a reasonable initial investigation but is operator-dependent and may yield false-negative results; cross-sectional imaging with computed tomography or magnetic resonance venography provides more reliable confirmation and delineates the extent of thrombus, including tumor thrombus in RCC. [5] D-dimer is of limited diagnostic value in this population: NS is associated with an acute-

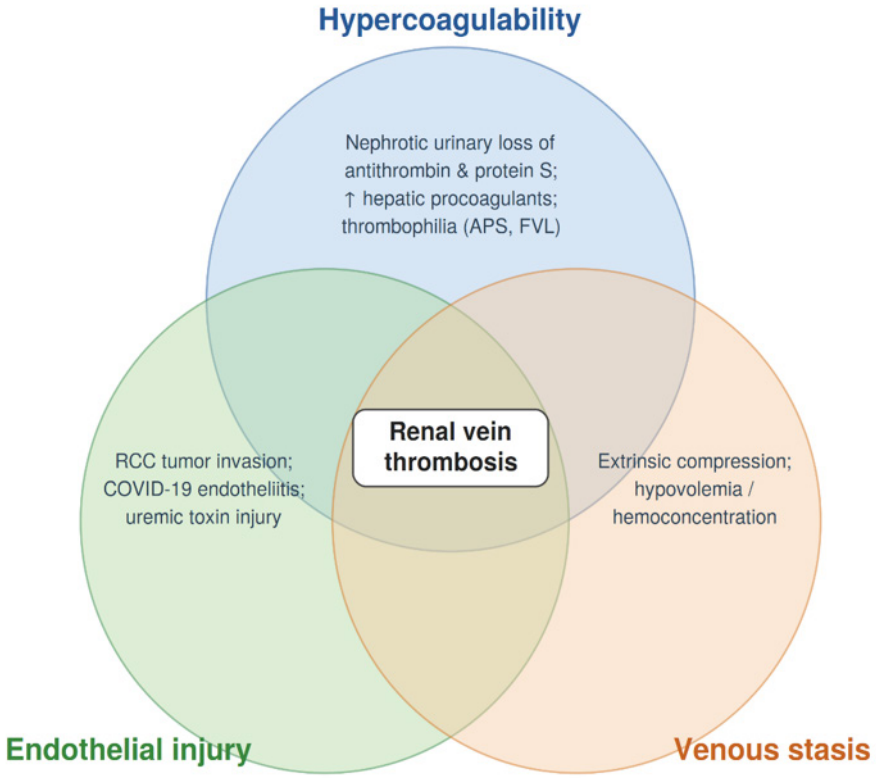

Figure 1: Pathophysiology of renal vein thrombosis mapped to Virchow's triad. APS, antiphospholipid syndrome; FVL, factor V Leiden; RCC, renal cell carcinoma.

Table 2: Suggested anticoagulant approach in renal vein thrombosis according to renal function..

Renal status	Preferred approach	Notes	Key references
Normal/early CKD (stage 1–3)	Initial LMWH, then DOAC (e.g., apixaban) generally preferred.	DOACs showed a superior benefit–risk profile versus VKAs in early-stage CKD.	[9,16]
Advanced CKD (stage 4)	Individualize; DOAC with caution or VKA.	Evidence limited; weigh bleeding risk explicitly.	[9]
Dialysis-dependent (ESKD)	Individualize; no agent clearly preferred.	Evidence insufficient to establish benefit or harm of either class.	[9]
All patients	Treat the underlying cause; individualize duration.	Duration depends on persistence of the prothrombotic stimulus (e.g., ongoing nephrotic syndrome).	[10,12]

RVT, renal vein thrombosis; CKD, chronic kidney disease; ESKD, end-stage kidney disease; INR, international normalized ratio; LMWH, low-molecular-weight heparin; DOAC, direct oral anticoagulant; VKA, vitamin K antagonist.

phase response and compensatory hepatic overproduction of fibrinogen and other clotting factors, which can elevate D-dimer independent of active thrombosis. [3,10] As a result, D-dimer lacks adequate specificity for RVT in nephrotic patients and should not be used to rule in or rule out the diagnosis; clinical suspicion based on presentation and risk factors, rather than serum biomarkers, should drive the decision to pursue confirmatory imaging. Because subclinical RVT may be common in high-risk groups, the threshold for imaging should be low when clinical features are suggestive. PE is a recognized and potentially serious sequela of RVT and may occasionally be its presenting manifestation; in a large prospective study of patients with NS undergoing systematic CT, PE was in fact detected more often than RVT and was frequently clinically silent, with one or both present in roughly one-third of patients. [15] Its occurrence reinforces the rationale for prompt therapeutic anticoagulation, the duration of which is individualized to the persistence of the underlying prothrombotic stimulus. [1,12]

MANAGEMENT

Anticoagulation

Anticoagulation is the foundation of RVT treatment. Initial parenteral therapy with low-molecular-weight heparin (LMWH), followed by transition to longer-term oral anticoagulation, is a common approach, with the duration individualized according to whether the underlying

prothrombotic stimulus (for example, ongoing NS) persists. [10,12] The choice between a DOAC and a vitamin K antagonist (VKA) is informed chiefly by renal function, since DOACs depend to varying degrees on renal clearance (**Table 2**).

In patients with CKD, a systematic review and meta-analysis of anticoagulation found that in early-stage CKD, non-vitamin K oral anticoagulants had a benefit–risk profile superior to that of VKAs. In contrast, for advanced CKD and dialysis-dependent end-stage kidney disease, the evidence was insufficient to establish benefit or harm for either class. [9] Accordingly, in dialysis-dependent patients, no oral anticoagulant can be regarded as clearly preferred, and the choice, including whether to anticoagulate at all, should be individualized to bleeding risk. [9] These findings, although derived from broader CKD and atrial fibrillation/VTE populations rather than from RVT specifically, support the cautious preference for apixaban (which has comparatively lower renal elimination) in non-dialysis patients. At the same time, VKAs with monitoring remain a pragmatic option where DOACs are contraindicated. [9] Early pharmacokinetic data for apixaban in NS are emerging and will help refine dosing in this specific population. [16] Decisions should remain individualized and should weigh bleeding risk explicitly. The general principles of RVT management are extrapolated from CKD-associated VTE evidence rather than RVT-specific trial data, and agent and dose should be individualized accordingly.

Monitoring during anticoagulation should include periodic assessment of renal function, given its influence on drug clearance and dosing, together with clinical surveillance for bleeding. [9] In patients with significant renal impairment treated with DOACs, anti-Xa level monitoring may be considered where available, though validated therapeutic ranges specific to RVT are lacking. The persistence of the provoking factor generally guides duration of therapy: anticoagulation is typically continued for as long as nephrotic-range proteinuria persists in NS-associated RVT, for a finite course (typically 3–6 months) in provoked, transient scenarios, and indefinitely in patients with recurrent thrombosis or an unresolved hypercoagulable state. [10,12]

Suggested anticoagulant approach in RVT according to renal function.

Catheter-Directed and Surgical Therapy

In patients with acute, extensive RVT and rapidly deteriorating renal function in whom systemic anticoagulation alone appears inadequate, catheter-directed thrombectomy and/or thrombolysis has been described. In a single-center experience, percutaneous catheter-directed treatment of acute RVT was feasible in both native and allograft kidneys. It was associated with improvement in renal function and a low incidence of morbidity. However, the evidence base comprises small, non-randomized case series and must be regarded as hypothesis-generating. [8] In RCC with venous tumor extension, surgical thrombectomy combined with nephrectomy is the mainstay of treatment for resectable, non-metastatic disease. [4,5]

Etiology-Directed Treatment

Treating the underlying cause is integral to management. In NS, control of the glomerular disease and proteinuria reduces the ongoing thrombotic drive. [2,10] In RCC, oncologic therapy addresses the tumor itself. In COVID-19–associated RVT, management of the infection and its coagulopathy accompanies anticoagulation. [6,7]

Primary Thromboprophylaxis in NS

Because the thrombotic risk in NS is high and is concentrated in patients with membranous nephropathy and severe hypoalbuminemia, primary thromboprophylaxis has been proposed as a means of preventing VTE, including RVT, before it occurs. [2,10] No randomized controlled trial has established the efficacy or safety of prophylactic anticoagulation in this setting, so current recommendations rest on observational data and on estimates of the balance between thrombotic and bleeding risk. [3,17] In a retrospective cohort, nephrotic patients who received prophylactic anticoagulation experienced fewer thromboembolic events than those who did not, without a prohibitive excess of major bleeding. [18] A systematic review of this literature reached a similar conclusion—that prophylaxis can reduce thromboembolic events in selected high-risk patients—while emphasizing the low certainty of the underlying evidence. [3] In practice, the decision is individualized, commonly guided by the degree of hypoalbuminemia, the underlying glomerular lesion, and competing bleeding risk; emerging pharmacokinetic data for DOACs in NS may help refine agent selection, although prospective trials remain necessary. [16,17]

Emerging Therapeutic Approaches

Factor XI inhibitors represent a mechanistically attractive class because they aim to uncouple antithrombotic efficacy from bleeding risk. Several agents—including the monoclonal antibody abelacimab and the small molecules asundexian and milvexian—are in clinical development. In a phase 2 trial in patients undergoing total knee arthroplasty, abelacimab produced a dose-dependent reduction in postoperative VTE relative to enoxaparin (4% versus 22% at the highest dose) with a low incidence of bleeding. [19] The antibody agents in this class are of particular interest in renal impairment because they are cleared largely by the reticuloendothelial system rather than the kidney. [20] Results across the class have been mixed, however: the phase 3 OCEANIC-AF trial of the oral factor XIa inhibitor asundexian in atrial fibrillation was stopped early for efficacy inferior to apixaban, albeit with less bleeding. [21] Definitive efficacy and safety data in renal populations, and in RVT specifically, are not yet available.

Etiology-directed oncologic therapy continues to evolve in the management of RCC-associated venous tumor thrombus. Neoadjuvant tyrosine kinase inhibition has been evaluated to reduce the extent of venous tumor thrombus before surgery, with the NAXIVA study providing prospective data on neoadjuvant axitinib in clear cell RCC with venous invasion. [22] Such approaches may, in selected patients, facilitate less morbid surgery, and the role of anticoagulation alongside oncologic treatment is being formally appraised. [13]

ROLE OF ARTIFICIAL INTELLIGENCE

Interest in AI across thrombosis care is growing, but a central caveat governs this entire section: there are currently no AI tools validated specifically for RVT. All evidence presented here derives from general VTE, DVT, or PE contexts, and its application to RVT is, at present, conjectural. We therefore frame these findings as a methodological template that could in future be applied to RVT, not as established RVT evidence. [23,24]

Risk Prediction

Machine-learning (ML) models that integrate electronic health record data, laboratory values, and clinical variables have been developed for VTE risk stratification. A systematic review and meta-analysis of ML models for VTE prediction pooled 27 studies comprising more than half a million patients and reported a pooled sensitivity of approximately 0.79 and specificity of approximately 0.82; importantly, the majority of included studies were judged to be at high risk of bias, chiefly because of methodological limitations and a lack of external validation. [25] If trained and validated on RVT-specific data—which do not currently exist—such models might in principle identify high-risk nephrotic or RCC patients, but this remains entirely theoretical.

Imaging and Diagnosis

AI-guided point-of-care compression ultrasound systems allow non-expert operators to acquire diagnostic-quality images for DVT assessment, with remote expert review. Prospective evaluation of such a pathway demonstrated that a substantial proportion of patients with suspected DVT could be assessed by non-specialists with high diagnostic-quality scan rates and favorable sensitivity, raising the possibility of expanding access to venous imaging in resource-limited settings. [26] Whether comparable systems could be adapted to detect RVT—a deeper, technically more demanding target—has not been studied, and would require dedicated development and validation.

Anticoagulation Dosing and Monitoring

AI-based dosing tools that incorporate pharmacokinetic, renal-function, and pharmacogenomic data have been explored to personalize anticoagulant therapy, an application of obvious relevance to patients with impaired and fluctuating renal clearance. A recognized limitation of many such models is restricted ethnic generalizability, since models derived in predominantly European or North American cohorts may transfer poorly to other populations—a concern that is amplified for pharmacogenomically dependent algorithms and that is directly relevant to a condition such as RVT that occurs worldwide. [23,24] Natural-language-processing tools and patient-facing applications have likewise been studied in general VTE care but not in RVT. [23]

Methodological and Translational Requirements

Realizing any of this potential will require RVT-specific training data, prospective external validation, attention to algorithmic bias and data privacy, and adherence to established reporting standards for AI interventions, such as the SPIRIT-AI guidance for trial protocols. [27] The foundational literature on machine learning in clinical medicine emphasizes that performance demonstrated in one setting cannot be assumed to transfer to another, particularly for rare diseases. [23,24] AI is therefore best regarded, in the context of RVT, as a promising direction that has not yet generated usable disease-specific evidence.

Future Directions

Several priorities follow from this synthesis. First, multicenter cohorts dedicated to RVT are needed to generate disease-specific data on natural history, optimal anticoagulant choice and duration, and the role of catheter-directed therapy. Second, the comparative effectiveness of DOACs versus VKAs warrants formal study across the spectrum of renal function, including dialysis-dependent patients, in whom current evidence is weakest. [9] Third, factor XI inhibitors merit dedicated evaluation in renal populations given their potentially favorable safety profile. [20] Finally, any AI tool proposed for RVT should be developed and validated prospectively in RVT-specific datasets, with ethnically diverse training data and pre-specified, guideline-concordant evaluation. [25,27]

Given the rarity of RVT, future research should also address the cost-effectiveness of diagnostic and management strategies—for example, comparing selective, risk-stratified screening imaging against routine screening in high-risk NS populations, and evaluating whether DOACs offer cost advantages over traditional anticoagulation once monitoring and complication costs are considered. Formal health economic analyses in this area are currently lacking.

Limitations

This is a narrative review and does not employ the systematic search, dual independent screening, formal risk-of-bias appraisal, or quantitative pooling required of a systematic review or meta-analysis; selection of sources therefore carries a risk of bias. No randomized trials specific to RVT were identified, and much of the evidence cited is extrapolated from broader VTE and CKD populations that share mechanisms with, but are not equivalent to, RVT. A portion of the RVT-specific literature comprises case reports and small series. The AI literature discussed is drawn from DVT, PE, and general VTE contexts and has not been validated in RVT. Restriction to English-language sources may have omitted relevant literature.

CONCLUSIONS

RVT is a rare but consequential disorder arising principally from NS, malignancy, thrombophilia, and, more recently, COVID-19–associated coagulopathy. Its pathophysiology reflects Virchow's triad, with hypercoagulability central to the nephrotic state. Anticoagulation remains the cornerstone of treatment; DOACs are a reasonable first choice in non-dialysis patients, with VKAs reserved for situations in which DOACs are contraindicated, and management individualized according to renal function and bleeding risk. Catheter-directed therapy, etiology-directed oncologic treatment, and factor XI inhibitors are promising adjuncts that require further study. AI offers a credible long-term opportunity to improve risk stratification, diagnosis, and dosing in RVT, but at present no AI evidence is specific to this condition; bridging that gap is the essential next step.

AUTHORS' CONTRIBUTIONS

All authors contributed substantially to the conception and design of the study, data acquisition and analysis, drafting and revising of the manuscript, final approval of the version to be published, and are accountable for all aspects of the work.

DISCLOSURE OF ARTIFICIAL INTELLIGENCE ASSISTANCE:

The authors disclose the use of AI technologies in the preparation of this manuscript, specifically for linguistic refinement, content restructuring, and figure creation. Despite this assistance, the intellectual framework, analysis, and scholarly conclusions remain the exclusive original work of the authors.

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None.

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