

# Management of Splanchnic Venous Thrombosis: An Update

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## Abstract

This review provides an updated and comprehensive overview of the diagnosis and management of splanchnic venous thrombosis (SVT), a rare but clinically important manifestation of venous thrombosis involving the portal, mesenteric, splenic, and hepatic veins. The aim of this article is to summarize and critically appraise recent advances in the evidence base for anticoagulant therapy, prognosis, and clinical decision-making across the diverse clinical settings in which SVT occurs, including the most important subgroups of liver disease, myeloproliferative neoplasms, and solid malignancy. The review integrates findings from prospective studies, meta-analyses, and registry data and highlights the evolving role of direct oral anticoagulants (DOACs) as an alternative to vitamin K antagonists and low-molecular-weight heparins. Meta-analyses demonstrate that anticoagulation increases the likelihood of vessel recanalization and improves survival in cirrhotic portal vein thrombosis (PVT) without increasing major bleeding. Pooled data suggest that DOACs achieve at least comparable efficacy to conventional anticoagulant drugs, with lower rates of thrombus growth and major bleeding, while low-dose rivaroxaban has proven effective for secondary prevention in chronic non-cirrhotic PVT. In cancer-associated SVT, anticoagulation reduces recurrence with acceptable bleeding risk, and in Budd–Chiari syndrome, DOACs show promising safety and efficacy in selected patients. Despite major progress in recent years, most evidence still remains observational, and randomized trials, pediatric data, and studies on long-term secondary prevention are urgently needed to further refine and standardize SVT management.

## Keywords

- ▶ anticoagulants
- ▶ Budd–Chiari syndrome
- ▶ mesenteric vein thrombosis
- ▶ portal vein thrombosis
- ▶ splenic vein thrombosis

## Why Splanchnic Venous Thrombosis Matters?

Splanchnic venous thrombosis (SVT) is a rare but clinically important manifestation of venous thrombosis, including thrombosis of the portal, splenic, and mesenteric veins as well as Budd–Chiari syndrome (BCS). Its relevance stems from the complexity of the underlying risk factors, the high risk of complications such as intestinal ischemia and portal

hypertension, and the delicate balance between thrombosis and bleeding that complicates management. In contrast to deep vein thrombosis and pulmonary embolism, SVT often occurs in the setting of local predisposing conditions such as cirrhosis, malignancy, or pancreatitis, as well as systemic disorders such as myeloproliferative neoplasms (MPNs).<sup>1,2</sup>

SVT is rare in the general population, with incidence rates for portal vein thrombosis (PVT) of approximately 2 to 3 per 100,000 person-years and BCS of approximately 1 to 2 per

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million individuals annually.<sup>1,3</sup> However, prevalence is much higher in selected risk groups, for example, around 10% in patients with gastrointestinal malignancies.<sup>1,4</sup> Multivessel thrombosis is present in around 40% of patients. The sex and age distribution varies: PVT is more frequent in middle-aged men, whereas BCS shows female predominance, typically in younger adults.<sup>2</sup> In MPN, the prevalence of SVT is disproportionately high, and SVT may even be the first manifestation leading to MPN diagnosis.<sup>5</sup>

The therapeutic goal in SVT is to prevent thrombus extension, promote recanalization, and avoid recurrent thrombosis, while minimizing bleeding complications. The past decade has seen an expansion of evidence on anticoagulation in SVT, including prospective studies and meta-analyses, as well as the increasing role of direct oral anticoagulants (DOACs). At the same time, large registries and systematic reviews have clarified the natural history of SVT in different risk groups and underscored the importance of early and sustained treatment.<sup>6,7</sup>

## General Considerations in Diagnosis and Management

The diagnosis of SVT relies on imaging rather than laboratory testing. D-dimer lacks specificity in this setting, as it is frequently elevated in liver disease, cancer, or inflammation.<sup>8</sup> According to current guideline-based diagnostic algorithms, color Doppler ultrasound is the first-line modality for portal and hepatic veins, while cross-sectional imaging with computed tomography or magnetic resonance (MR) venography is recommended for complete evaluation of the splanchnic venous system, including detection of collateral circulation, and assessment of complications such as bowel ischemia.<sup>8,9</sup> Differentiating acute from chronic thrombosis is clinically important. Direct MR thrombus imaging can help distinguish fresh thrombus from chronic occlusion.<sup>9</sup> Endoscopic screening for varices is mandatory in cirrhotic patients prior to or in parallel with initiation of anticoagulation.<sup>10</sup>

Anticoagulation remains the cornerstone of SVT management. Its main goals are to (1) prevent thrombus growth and bowel ischemia, (2) promote recanalization, and (3) prevent recurrence. Early initiation of anticoagulation is associated with higher recanalization rates and fewer complications related to portal hypertension.<sup>11</sup> Therapeutic options include low-molecular-weight heparin (LMWH), vitamin K antagonists (VKAs), and DOACs. LMWH is typically used in the acute phase, particularly in cirrhosis or malignancy, whereas long-term therapy increasingly relies on DOACs. Evidence from a prospective study and recent meta-analyses shows that DOACs provide at least comparable efficacy and a favorable bleeding profile compared with VKA in non-cirrhotic SVT.<sup>12–14</sup> However, careful patient selection remains crucial, especially in advanced hepatic dysfunction, severe renal impairment, or triple-positive antiphospholipid syndrome. The recommended duration of anticoagulation depends on the persistence of underlying risk factors. Three to six months are recommended for transient triggers such as pancreatitis and indefinite therapy for permanent conditions such as cirrhosis,

active malignancy, or MPN.<sup>15,16</sup> Reduced-dose DOAC regimens for extended secondary prevention are extrapolated from venous thromboembolism (VTE) trials, and SVT-specific evidence remains limited. Thrombolysis or thrombectomy should be reserved for cases with intestinal ischemia not controlled by anticoagulation, and interventional approaches such as transjugular intrahepatic portosystemic shunt (TIPS) implantation may be considered in selected cirrhotic patients with progressive thrombosis or as a bridge to transplantation.<sup>10</sup>

With the widespread use of abdominal imaging, a growing proportion of splanchnic vein thromboses are detected incidentally, particularly in patients undergoing screening for liver disease or cancer. Cohort studies estimate that up to 30 to 60% of SVTs are now diagnosed without clinical symptoms.<sup>17</sup> Importantly, incidentally detected SVTs carry a prognosis comparable to symptomatic events. An individual patient data meta-analysis including nearly 500 cases found similar risks of major bleeding, higher recurrence rates, and lower mortality compared with symptomatic SVT.<sup>18</sup> These findings support the use of anticoagulation even in asymptomatic cases. However, registry data demonstrate that incidental SVTs are undertreated. In the Registro Informatizado de Enfermedad TromboEmbólica (RIETE) registry, anticoagulation was given less frequently to asymptomatic than to symptomatic patients.<sup>19</sup> Guidelines therefore recommend that incidental and symptomatic SVT be managed similarly, with anticoagulation indicated in the absence of contraindications.<sup>16</sup> Key recent studies and meta-analyses informing anticoagulant management across SVT populations are summarized in [Table 1](#).

## Myeloproliferative Neoplasms and Splanchnic Venous Thrombosis

MPNs represent the leading systemic cause of SVT, particularly BCS and non-cirrhotic, non-malignant PVT. Recent meta-analyses indicate that Philadelphia-negative MPNs account for approximately one-third to one-half of BCS cases and for about one-fifth of idiopathic, non-cirrhotic PVT.<sup>1,20</sup> The *JAK2-V617F* mutation is found in more than 75% of patients with MPN-associated SVT.<sup>21</sup> The pronounced prothrombotic state in MPN-associated SVT is further reflected by the presence of free thrombin in the circulation.<sup>22</sup>

Anticoagulation is the cornerstone of therapy, and indefinite treatment is generally required due to the persistent prothrombotic stimulus. Long-term follow-up demonstrates that VKAs reduce the risk of recurrence by approximately 50%, although the residual risk remains substantial.<sup>21</sup> More recently, DOACs have been used in this population. Pooled analyses and meta-analyses suggest comparable safety and efficacy of DOACs compared with VKAs, although prospective evidence is sparse.<sup>14</sup> The additional use of cytoreductive drugs is the second key element of MPN-associated SVT. In patients with polycythemia vera or essential thrombocythemia, treatment with hydroxyurea or interferon  $\alpha$  may reduce thrombotic risk, although data in the SVT setting are largely observational.<sup>5</sup>

As summarized in recent reviews, BCS is strongly associated with MPN, representing the prototypical manifestation of MPN-related SVT and accounting for the majority of systemic

**Table 1** Selected recent key studies on splanchnic venous thrombosis management

Study	Population/Design	Main finding	Key message
Valeriani et al, 2021 <sup>33</sup>	Prospective IRSVT registry; 264 cancer patients (132 SVT vs. 132 VTE)	Similar VTE recurrence (4.7% vs. 5.5%) and major bleeding (2.3% vs. 4.7%)	Cancer-associated SVT has outcomes comparable to typical-site VTE
Ageno et al, 2022 <sup>12</sup>	Prospective interventional single-arm trial; 100 adults with acute non-cirrhotic SVT treated 6 months with rivaroxaban	Major bleeding 2%; recurrent SVT 2%; complete recanalization 47%, good overall safety and efficacy	Rivaroxaban is a feasible alternative to LMWHs/VKAs for acute non-cirrhotic SVT
Plessier et al, 2022 <sup>41</sup> (RIPORT)	RCT, 111 patients with non-cirrhotic chronic PVT; rivaroxaban 15 mg/day versus no anticoagulation	Thrombosis incidence 0/100 PY with rivaroxaban versus 19.7/100 PY without; major bleeding 2 versus 1 cases	Low-dose rivaroxaban prevented recurrent VTE in non-cirrhotic PVT without increased major bleeding
Cohen et al, 2023 <sup>44</sup>	Systematic review and meta-analysis, 17 studies, 506 pediatric SVT patients	Recanalization 55% anticoagulated versus 29% untreated; major bleeding 3.8%; mortality 10%	Anticoagulation in pediatric SVT achieves moderate recanalization with low bleeding; limited evidence
Guerrero et al, 2023 <sup>28</sup> (IMPORTAL)	Individual patient data meta-analysis; 5 studies, 500 cirrhotic PVT patients; 205 anticoagulated, 295 untreated	Anticoagulation ↓ mortality (aSHR 0.59), ↑ recanalization (OR 3.45), no ↑ PH-related bleeding	In cirrhotic PVT, anticoagulation improves survival independent of recanalization
Semmler et al, 2023 <sup>25</sup>	Retrospective multicenter study; 47 BCS patients, 22 on DOACs, median follow-up 82 months	Major bleeding 18% (8.8/100 PY); 5-year transplant-free survival 91.6%	DOACs show good efficacy and acceptable safety in BCS, limited evidence
Calcaterra et al, 2024 <sup>14</sup>	Pooled analysis and meta-analysis; 16 studies, 648 SVT patients on DOACs	Any recanalization 60%; full 52%; recurrence 2.8%; major bleeding 5.8%	DOACs show favorable safety and efficacy in SVT, limited evidence
Wan et al, 2024 <sup>13</sup>	Systematic review and meta-analysis; 9 observational studies, DOACs versus VKAs in SVT	↑ recanalization (71% vs. 55%), less SVT growth (OR 0.12), ↓ major bleeding (OR 0.27) with DOACs	DOACs outperform VKAs in safety and some efficacy endpoints

Abbreviations: aSHR, adjusted subdistribution hazard ratio; BCS, Budd–Chiari syndrome; DOAC, direct oral anticoagulant; IRSVT, International Registry on Splanchnic Vein Thrombosis; LMWH, low-molecular-weight heparin; OR, odds ratio; PH, portal hypertension; PVT, portal vein thrombosis; PY, patient years; RIPORT, Rivaroxaban Prophylaxis in Portal Vein Thrombosis; RCT, randomized controlled trial; SVT, splanchnic venous thrombosis; VKA, vitamin K antagonist; VTE, venous thromboembolism.

causes identified in this setting.<sup>1</sup> The clinical spectrum ranges from fulminant liver failure to indolent chronic disease with portal hypertension. Anticoagulation is typically initiated with LMWH, followed by long-term oral anticoagulation. Outcomes have improved significantly with a stepwise therapeutic approach combining anticoagulation, endovascular interventions, TIPS, and ultimately liver transplantation in refractory cases. With this strategy, 5-year survival now exceeds 80%.<sup>23,24</sup> Data on DOACs in BCS are limited but growing. Retrospective series suggest comparable efficacy to VKAs, although bleeding risk appears elevated in patients with advanced cirrhosis. In a multicenter study, DOACs achieved high stent patency rates but were associated with major bleeding in 18% of patients, half of whom had Child-Pugh B/C disease.<sup>25</sup> Thus, DOACs may be considered in selected cases but require cautious use.

In summary, MPN-associated SVT is characterized by early onset, frequent multivessel involvement, and high risks of recurrence and bleeding. Lifelong anticoagulation is warranted, with VKAs and DOACs as viable options. Optimal integration of cytoreduction and antithrombotic therapy remains an unmet need and is a priority for future research.

## Splanchnic Venous Thrombosis in Liver Disease

Liver disease is the most frequent local risk factor for SVT, particularly PVT. Its pathophysiology combines reduced portal blood flow, endothelial dysfunction, and systemic prothrombotic changes, resulting in a high thrombotic risk despite the traditionally perceived bleeding tendency of these patients.<sup>8,26</sup>

Anticoagulation has consistently been shown to improve outcomes in cirrhotic SVT. In a randomized controlled trial, early initiation of LMWH increased rates of partial or complete recanalization and reduced decompensation events.<sup>11</sup> A recent meta-analysis showed that anticoagulation in cirrhotic SVT approximately triples the likelihood of recanalization and reduces thrombus progression and mortality without a significant increase in major bleeding.<sup>27</sup> In addition, a competing-risk meta-analysis demonstrated that anticoagulation improves transplant-free survival irrespective of baseline thrombus extension.<sup>28</sup> Historically, VKAs were used, but DOACs are now increasingly adopted for long-term anticoagulant treatment. Recent meta-analyses

and large observational studies have shown that DOACs provide comparable or superior efficacy to VKAs for recanalization and prevention of recurrent thrombosis, with similar or lower rates of major bleeding.<sup>13,14</sup> Their use, however, requires careful patient selection, particularly in advanced liver disease, as bleeding risk increases in the setting of portal hypertension.<sup>29</sup> Rivaroxaban remains contraindicated in Child-Pugh B, and all DOACs are contraindicated in Child-Pugh C.

Guideline recommendations have shifted accordingly. International and national guidelines now advise early initiation of LMWH followed by VKAs or, in eligible patients, DOACs.<sup>8,16</sup> The Baveno VII consensus recommends anticoagulation for  $\geq 50\%$  occlusion of the portal trunk, for progression on short-term imaging, or in transplant candidates.<sup>10</sup> Duration of therapy is generally indefinite, since cirrhosis is a permanent risk factor. Anticoagulation should be continued until vessel recanalization or lifelong in transplant candidates.<sup>26</sup> While reduced-dose DOACs are established in extended VTE treatment, data in cirrhotic SVT remain limited.<sup>15</sup>

In summary, while cirrhotic SVT carries both high thrombotic and bleeding risks, evidence now supports systematic anticoagulation in most patients. The shift from cautious avoidance to proactive therapy represents a major change in the management paradigm of cirrhotic SVT.

### Intra-Abdominal Malignancies and Inflammatory Diseases

SVT is a frequent complication in patients with intra-abdominal malignancies, particularly hepatocellular carcinoma (HCC) and pancreatic cancer, where both tumor-related vascular invasion and systemic hypercoagulability contribute to thrombosis.<sup>30,31</sup> Up to one-third of patients with HCC present with portal vein involvement, often requiring differentiation between bland and tumor thrombus.<sup>30</sup> Like HCC, gastrointestinal malignancies are also associated with incidental or symptomatic SVT, sometimes as the first manifestation of occult cancer.<sup>32</sup> The clinical course of cancer-associated SVT is marked by high overall mortality, which is largely determined by the underlying tumor biology rather than thrombosis itself. In the International Registry on Splanchnic Vein Thrombosis, patients with cancer-associated SVT had similar rates of recurrence and major bleeding compared with patients with VTE, but higher mortality due to cancer progression.<sup>33</sup> A large analysis confirmed worse survival in gastrointestinal cancers with concurrent SVT.<sup>34</sup>

Anticoagulation is generally recommended when not contraindicated, as it reduces recurrent VTE and promotes recanalization. In a retrospective multicenter cohort of 203 patients with solid cancer-associated SVT, approximately 50% received anticoagulants, with low rates of major bleeding and recurrent events within 30 days.<sup>35</sup> However, incidental SVT is often undertreated, despite evidence that prognosis parallels that of symptomatic cases.<sup>17,19</sup> The choice of anticoagulant requires consideration of tumor location and bleeding risk. LMWHs have long been preferred

in treatment, particularly in gastrointestinal cancers with mucosal involvement, where DOACs may increase bleeding risk. However, recent studies suggest that DOACs are effective and safe in selected patients without luminal disease or drug interactions.<sup>30,36</sup> Retrospective analyses of DOACs use in unusual-site VTE, including SVT, have shown encouraging outcomes, but prospective evidence remains limited. Overall, cancer-associated SVT requires individualized management. Anticoagulation is beneficial in most patients, but tumor-related factors, bleeding risk, and drug interactions determine the optimal regimen. Integration of anticoagulant therapy into multidisciplinary cancer care is essential, with LMWHs preferred in high-risk gastrointestinal cancers and DOACs increasingly considered in other settings.

Acute pancreatitis is another frequent local trigger of SVT, most commonly involving the splenic vein due to its anatomical proximity to the pancreas. Approximately 12% of patients with acute pancreatitis develop SVT, and the splenic vein is affected in more than 90% of cases, either in isolation or in combination with PVT or mesenteric vein thrombosis (MVT).<sup>37</sup> The clinical significance of pancreatitis-associated SVT is heterogeneous. While isolated splenic vein thrombosis may remain asymptomatic or spontaneously recanalize, extension into the portal or mesenteric veins increases the risk of complications such as portal hypertension and intestinal ischemia. In a cohort study, spontaneous resolution occurred in 44% of untreated splenic vein thromboses, compared with none of the untreated PVT or MVT, highlighting the differential prognosis of venous segments.<sup>37</sup> Two systematic reviews and meta-analyses have addressed the role of anticoagulation in this setting. Anticoagulated patients had higher recanalization rates, but no clear reduction in mortality compared with non-anticoagulated patients.<sup>38,39</sup> Anticoagulation is therefore preferred in extensive thrombosis, in mesenteric vein involvement, or when ischemia is suspected, while isolated splenic vein thrombosis in mild pancreatitis may be managed conservatively.

### Chronic Splanchnic Venous Thrombosis

Chronic SVT is characterized by cavernous transformation of the portal vein or extensive collateralization. By convention, thromboses persisting for more than 6 months are considered chronic.<sup>26</sup> Management decisions are challenging, as the balance between recurrence prevention and bleeding risk is delicate. Observational studies suggest that anticoagulation may prevent recurrent thrombosis and intestinal ischemia, particularly when mesenteric veins are involved or an underlying prothrombotic state is present.<sup>40</sup> In patients with non-cirrhotic chronic PVT, the RIPORT randomized trial showed that prophylactic-dose rivaroxaban reduced recurrence compared with no anticoagulation, without excess major bleeding.<sup>41</sup> In cirrhotic patients with chronic PVT, recanalization is less likely, but anticoagulation may still improve transplant outcomes and prevent progression.<sup>42</sup> The decision must be individualized, guided by risk factors and vessel involvement.

**Table 2** Evidence gaps and future needs in splanchnic venous thrombosis management

Topic	Current recommendation	Evidence gaps/future needs
Initiation and choice of anticoagulant	Early full-dose LMWH, transition to VKAs or DOACs if eligible; DOACs increasingly used in non-cirrhotic SVT	No randomized comparison of DOACs versus LMWH/VKAs; need for data in cirrhotic and cancer-associated SVT
Treatment duration	About 3–6 months for transient risk factors; indefinite therapy for cirrhosis, active cancer, or MPN	Optimal duration for unprovoked or chronic SVT unclear; no validation of reduced-dose DOAC regimens.
Cirrhotosis-associated SVT	Treat $\geq$ 50% portal trunk occlusion or progression; continue until recanalization or transplantation	Better risk stratification tools balancing bleeding and thrombosis; need for RCTs including Child-Pugh B/C patients
Cancer-associated SVT	LMWHs preferred for luminal GI tumors; DOACs acceptable in selected patients without mucosal disease	Lack of prospective SVT-specific cancer trials; impact of anticoagulation on survival unresolved.
MPN-associated SVT	Lifelong anticoagulation; cytoreductive therapy recommended	Optimal integration of cytoreduction and anticoagulation; unclear role of antiplatelet agents.

Abbreviations: DOAC, direct oral anticoagulant; GI, gastrointestinal; LMWH, low-molecular-weight heparin; MPN, myeloproliferative neoplasm; RCT, randomized controlled trial; SVT, splanchnic venous thrombosis; VKA, vitamin K antagonist.

## Future Directions and Unmet Needs

Despite significant advances in recent years, management of SVT remains guided mainly by observational data, meta-analyses, and extrapolation from usual-site VTE. Several important gaps persist.

First, randomized controlled trials are scarce. To date, most evidence on anticoagulation in SVT derives from retrospective cohorts and registry studies, with only a few prospective interventional trials assessing rivaroxaban in non-cirrhotic patients.<sup>12</sup> Robust randomized data comparing DOACs with VKAs or LMWHs in diverse SVT populations are urgently needed. Second, the optimal duration and intensity of anticoagulation remain unresolved. While indefinite therapy is standard in persistent risk factors such as cirrhosis, active cancer, and MPNs, the role of reduced-dose DOACs for secondary prevention is uncertain. Evidence is extrapolated from trials in VTE,<sup>15</sup> but specific validation in SVT is lacking. Third, the benefit of thrombophilia testing in SVT is limited. Apart from *JAK2-V617F* screening in suspected MPNs, hereditary thrombophilia testing rarely changes management decisions.<sup>43</sup> Future studies must clarify which patient groups may benefit from targeted diagnostics.

Finally, pediatric SVT remains an underresearched area. Most treatment is extrapolated from adult practice or from usual-site pediatric VTE studies. Tailored prospective data are needed to inform management in this subgroup.<sup>44</sup> Current evidence gaps and priorities for future research in SVT are summarized in **Table 2**.

## Conclusion

SVT represents a rare but clinically important manifestation of thrombosis, arising in complex risk constellations such as cirrhosis, cancer, and MPNs. Anticoagulation remains the cornerstone of therapy, with early initiation improving recanalization and reducing complications. Recent evidence sup-

ports the increasing use of DOACs in selected patients, while LMWHs and VKA remain essential in high-risk settings.

Management must be individualized according to underlying disease, bleeding risk, and patient-specific factors. For cirrhosis, careful assessment of liver function is crucial. In cancer, mucosal tumor location and drug interactions guide anticoagulant choice. In MPNs, lifelong anticoagulation is mandatory but insufficient alone, requiring integration with cytoreductive therapy.

While outcomes have improved substantially with structured therapeutic approaches, particularly in BCS, several open questions remain. Randomized controlled trials, pediatric studies, and better evidence on long-term secondary prevention are priorities.

In conclusion, SVT exemplifies the challenges of balancing thrombotic and hemorrhagic risks in complex patients. Evidence increasingly favors proactive anticoagulation across subgroups, but individualized, multidisciplinary management remains essential to optimize outcomes.

### Authors' Contributions

H.R. and S.R. drafted and edited the manuscript. Both authors agreed with its content and approved the submission.

### Conflict of Interest

The authors declare that they have no conflict of interest.

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