

Cancer-associated superficial vein thrombosis: clinical outcomes and management

Adrian Joseph Michel Bailey,^{1*} Owen Dan Luo,^{2*} Philip Stephen Wells³

¹Faculty of Medicine, University of Ottawa, Ontario; ²Faculty of Medicine and Health Sciences, McGill University, Montréal, Quebec; ³Department of Medicine, University of Ottawa, and the Ottawa Hospital Research Institute, Ottawa, Ontario, Canada

*These authors contributed equally to this review.

ABSTRACT

Venous thromboembolism (VTE) is a major cause of morbidity and noncancer mortality in patients with active malignancy, and management frameworks for cancer-associated deep vein thrombosis (DVT) and pulmonary embolism (PE) are well developed. In contrast, there exists controversy in the management of isolated superficial vein thrombosis (iSVT) in patients with active malignancies, as the guideline recommended treatment regimens for iSVT are based on randomized trials that largely excluded patients with active cancer. Furthermore, patients with cancer have a higher risk of bleeding and thrombosis complicating anticoagulant management. This review synthesizes evidence on outcomes and management of cancer-associated iSVT. The evidence indicates there is a high rate of subsequent VTE incidence after cancer-associated iSVT despite anticoagulation, and comparisons of patients with active malignancy to noncancer-associated iSVT demonstrated a substantially higher VTE risk with cancer, with low rates of major bleeding. This suggests that patients with cancer-associated iSVT may benefit from higher doses of anticoagulation and/or extended therapy. However, the data suffers from heterogeneity

and limited numbers emphasizing the need for randomized controlled trials to assess the optimal anticoagulant intensity/duration for patients with cancer-associated iSVT.

Corresponding author: Dr Philip Wells, Suite 2255, 501 Smyth Rd, Ottawa, ON, K1H 8L6, Canada.
E-mail: pwells@toh.ca

Contributions: AJMB, concept and design, analysis and/or interpretation of data, critical writing of the manuscript, and final approval of the version to be published; ODL, concept and design, analysis and/or interpretation of data, critical writing of the manuscript, and final approval of the version to be published; PSW, critical revising the intellectual content and final approval of the version to be published.

Conflict of interest: the authors declare no competing interests and confirm accuracy.

Availability of data and materials: the datasets used and/or analyzed during the current study are available upon reasonable request from the corresponding author.

Acknowledgments: the authors thank Risa Shorr, Librarian at The Ottawa Hospital, for designing the study's comprehensive systematic review search strategy for Medline, Embase, Cochrane Central, and Web of Science.

Received: 9 January 2026.
Accepted: 6 March 2026.

Publisher's note: all claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article or claim that may be made by its manufacturer is not guaranteed or endorsed by the publisher.

©Copyright: the Author(s), 2026
Licensee PAGEPress, Italy
Bleeding, Thrombosis and Vascular Biology 2026; 5(s1):446
doi:10.4081/btvb.2026.446

This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License (CC BY-NC 4.0).

Key words: neoplasms; systematic review; venous thromboembolism; venous thrombosis; anticoagulants.

Introduction

Venous thromboembolism (VTE), comprising deep vein thrombosis (DVT) and pulmonary embolism (PE), is a major source of morbidity and mortality among patients with active malignancy and remains a leading cause of noncancer death in oncology populations.¹ Population-based studies consistently demonstrate that patients with active cancer have substantially increased VTE risk compared with individuals without cancer, as a result of tumor-driven hypercoagulability, host inflammation, endothelial perturbation, and thrombogenic exposures intrinsic to cancer care (surgery, systemic therapy, indwelling venous access, and immobility).^{2,3} A recently published network meta-analysis found that 3.1% of patients experienced VTE within 1 year of cancer diagnosis, with cancer specific rates ranging from 0.7-7.4%.⁴ These realities underpin the guideline recommendation for patients with active cancer and VTEs to receive indefinite anticoagulation for secondary prophylaxis while the malignancy remains active.⁵

Superficial vein thrombosis (SVT) is defined as a partial or total thrombotic obstruction with associated inflammation of a superficial vein of the lower or upper extremities and presents clinically as a red, swollen, and painful cord along the course of the affected superficial vein. SVT can occur in the lower extremity (classically great or small saphenous veins and tributaries) or upper extremity (often cephalic/basilic veins), the latter frequently associated with peripheral intravenous cannulation or central venous access devices. Compression ultrasound is used to diagnose SVT, exclude the presence of concomitant DVT, and determine

its length and proximity to the deep venous system. SVTs are prevalent and historically perceived as a benign, self-limiting condition that can be treated conservatively with warm compresses and topical anti-inflammatory agents. The incidence of SVT is estimated to be 1.31 per 1000 person-years in a population-based study of primary care patients, with an annual diagnosis rate of 0.64% in a separate community-based sample of general adult patients.^{6,7} Although there is no published incidence data of SVT in patients with active cancer, the prevalence of malignancy in a cohort of patients with SVT was 8.7% emphasizing the significantly increased risk compared to patients without cancer.⁸

In noncancer populations, contemporary guidelines recommend therapeutic-dose anticoagulation when the SVT is junction-threatening (typically within 3 cm of the deep venous junction) or when concurrent DVT/PE is present, whereas prophylactic-dose fondaparinux or rivaroxaban, or prophylactic/intermediate-dose low-molecular-weight heparin (LMWH), for ~45 days is recommended for extensive isolated SVT (≥ 5 cm in length) away from the deep venous junctions.⁹ These recommendations are grounded in randomized evidence. In the “Comparison of Arixtra in Lower Limb Superficial Vein Thrombosis with Placebo” (CALISTO) randomized controlled trial, 45 days of fondaparinux 2.5 mg daily reduced the development of the composite outcome of death from any cause or symptomatic PE, symptomatic DVT, or symptomatic extension/recurrence of SVT compared with placebo without increasing the incidence of bleeding, but patients treated for cancer within the prior 6 months were excluded, which limits applicability to patients with active malignancy.¹⁰ In the “Superficial Phlebitis Treated for Forty-five Days with Rivaroxaban versus Fondaparinux” (SURPRISE) randomized controlled trial, rivaroxaban 10 mg daily was noninferior to fondaparinux 2.5 mg daily for 45 days in the treatment of a higher-risk SVT population and was not associated with more major bleeding, but patients with cancer represented only a small subgroup (around 10%) of the study sample.¹¹

Active malignancy complicates the management of SVT by simultaneously amplifying the risk of thrombotic extension and bleeding. It remains controversial whether cancer-associated isolated SVT (iSVT; SVT without baseline concomitant DVT/PE) behaves as a lower-risk superficial phenotype amenable to standard 45-day SVT regimens, or whether longer or more intensive therapy is warranted in selected patients with active malignancy.

The present review provides an outcomes-focused synthesis for clinicians managing cancer-associated iSVT that is based on the findings reported by our recently published systematic review and meta-analysis.¹² We summarize absolute risks of subsequent VTE, bleeding, hospitalization, and death after cancer-associated iSVT; compare cancer-associated iSVT clinical outcomes with patients without active cancer and iSVT; and delineate key controversies and research priorities, including optimal anticoagulant intensity/duration for cancer-associated iSVT.

Clinical outcomes after cancer-associated isolated SVT

To directly address this evidence gap, Bailey and colleagues conducted a systematic review and random-effects meta-analysis (IROVAM-iSVT) to quantify the risk of subsequent VTE after iSVT in adults with active malignancy.¹² The prespecified protocol

was registered (PROSPERO CRD42024550983), the review followed PRISMA reporting principles, and the search encompassed Medline, Embase, Web of Science, and Cochrane from inception to December 2, 2024, without language or geographic restrictions. In an updated search using the same search strategy and eligibility criteria through December 9, 2025 performed by the review authors, no additional full-text studies that met the inclusion criteria were identified.

From 839 screened records, 29 full-text articles were assessed and 8 studies met eligibility criteria, comprising four prospective and four retrospective cohorts from more than six countries and spanning patient data from 1991 to 2021.¹⁴⁻²¹ This review was unable to find any randomized, controlled data that focused only on the treatment of iSVT in active malignancy. Collectively, the studies we included enrolled 5,998 patients with objectively confirmed iSVT, including 448 patients meeting the ISTH-informed definition of active malignancy (ongoing anticancer treatment, metastatic disease, diagnosis within 6 months, or recurrent malignancy). Follow-up was most often reported after 3 months of treatment, but some cohorts extended to 6 months, 1 year, or longer. Person-years were estimated, when not reported, by multiplying cohort size by mean/median follow-up duration. The overall quality of the studies was high with a median of 7.5 (IQR 6.5-9) as per the Newcastle-Ottawa Quality Assessment Scale for Cohort Studies.

The included cancer-iSVT populations were clinically heterogeneous but broadly representative of oncology practice. Across studies, the proportion of male patients ranged from 30% to 67% and mean/median age ranged from 65.2 to 72.0 years. Malignancy types were diverse including breast, lung, gastrointestinal, genitourinary, hematological, central nervous system, melanoma, thyroid and oropharyngeal with a metastatic disease prevalence that ranged from 11.8% to 44% across the studies. Most studies reported common VTE risk factors (e.g., prior VTE, varicose veins), but definitions of “symptomatic” vs “asymptomatic” outcome events were inconsistent: four studies specified that subsequent VTE events were symptomatic, while four did not specify symptom status. This distinction is clinically important because incidentally detected VTE can differ from symptomatic VTE in prognosis and management. Most of the studies included only patients with lower extremity iSVTs except for Ghenassia-Fouillet *et al.* and Bell *et al.*, which included both upper and lower extremity iSVTs.^{16,19}

Quantitatively, IROVAM-iSVT demonstrated that cancer-associated iSVT is followed by substantial subsequent VTE burden despite anticoagulation. The pooled overall incidence of VTE after cancer-associated iSVT was 18.2 events per 100 patient-years (95% CI, 5.2–31.2; $I^2=76\%$; $n=448$). When PE and DVT were reported separately (four studies; $n=315$), the incidence of PE was 9.6 events per 100 patient-years (95% CI, 1.8–17.4; $I^2=0\%$) and the incidence of DVT was 20.1 events per 100 patient-years (95% CI, 0–44.4; $I^2=61\%$).^{14,16-18} Notably, one study reported a median time to subsequent DVT/PE of 792 days (IQR, 484–1583) in the overall iSVT population (including patients with and without active malignancy), highlighting the potential for late events and the limitation of short follow-up windows to fully characterize VTE extension risk trajectories.²¹

Comparative analyses further emphasized malignancy as a strong modifier of iSVT prognosis. Across five of the included studies, with a total of 288 patients with active cancer and iSVT and 2,913 patients with no cancer and iSVT, active malignancy

was associated with a substantially higher risk of subsequent PE/DVT (risk ratio 2.6; 95% CI, 1.8–3.7; $I^2=0\%$; $p<0.001$).^{14,15,17,19,21} Importantly, these studies adjusted for known confounders for VTE progression (e.g., age, sex, varicose vein status, cardiovascular risk factors), strengthening the inference that active cancer independently increases VTE risk after iSVT. The review also captured clinically salient non-thrombotic outcomes and healthcare utilization: only one study reported hospitalization rates, and in that study malignancy was associated with a markedly higher risk of VTE-related hospitalization (HR 8.9; 95% CI, 2.1–37.1; $p=0.003$), with 3.9% requiring hospitalization.¹⁴

Four studies reported on the incidence of bleeding and mortality outcomes in patients with active cancer and iSVT.^{14–17} The incidence of major bleeding was low at 2.0 events per 100 patient-years (95% CI, 0–6.7; $I^2=59\%$) and the incidence of death was 22.8 events per 100 patient-years (95% CI, 0–58.7; $I^2=73\%$). Clinically relevant nonmajor bleeding (CRNMB) was reported in only one study (2 events among 77 patients; 2.6 events per 100 patient-years).¹⁴ VTE-specific mortality was reported in only two studies, which found that fatal VTE events were uncommon (e.g., RIETE reported 1 fatal PE [0.9%] among 110 cancer-iSVT patients vs 2 fatal PEs [0.2%] among 1,030 noncancer-iSVT patients).^{15,17}

Among anticoagulated patients with active cancer and iSVT, LMWH was most used (65%), followed by fondaparinux (26%), vitamin K antagonists (5%), and direct oral anticoagulants (4%). Across the four studies that reported the relative proportions of patients with iSVT treated with anticoagulation, there was no statistically significant difference in the proportion treated (OR 1.9; 95% CI 0.7–5.4; $I^2=46\%$; p =non-significant result [NR]) or duration treated (mean difference of 0.2 days; 95% CI 4.4–4.8; $I^2=0\%$; P =NR) between cancer-iSVT and noncancer-iSVT, implying that many cancer patients are treated with broadly similar anticoagulation regimens as noncancer iSVT despite higher recurrence risk.^{14,15,17,18} Compared to patients with active cancer and DVT, cancer-associated iSVT patients were less likely to receive anticoagulation (76% vs 96%) and received shorter courses (median 45.5 days vs 120 days) in the OPTIMEV-SFMV, while in RIETE cancer-iSVT patients received lower LMWH doses (118±60 vs 152±41 IU/kg/day) and shorter durations (125±155 vs 201±213 days) than cancer-DVT patients (all $p<0.001$).^{15,17}

Finally, the review synthesized the limited direct comparisons between cancer-iSVT and cancer-DVT/PE outcomes. Two studies suggested that subsequent VTE risk after cancer-iSVT may be similar to that after cancer-associated DVT/PE (OR 1.8; 95% CI, 0.8–3.9; and RR 1.5; 95% CI, 0.4–5.8; both p =NR), raising the clinically consequential hypothesis that a subset of cancer-iSVT may share recurrence biology with cancer-associated VTE.^{15,17}

This systematic review and meta-analysis of studies reported the clinical outcomes of cancer-iSVT and found a high pooled rate of subsequent VTE (18.2 events per 100 patient-years; 9.6 PEs per 100 patient-years and 20.1 DVTs per 100 patient-years) and a relatively low pooled rate of major bleeding events (2.0 events per 100 patient-years). This is a significantly greater rate of subsequent VTE when compared to another systematic review and meta-analysis of a predominantly non-cancer sample of iSVT (4% prevalence of active cancer in their pooled analysis) which found a pooled rate of subsequent VTE of 8 events per 100 patient-years (1.4 PEs per 100 patient-years and 7.8 DVTs per 100

patient-years) and major bleeding rate of 0.3 events per 100 patient-years among patients with iSVT that were treated with the guideline recommended duration of anticoagulation.²² In addition, our systematic review and meta-analysis identified that active malignancy was associated with a substantially higher risk of subsequent PE/DVT (RR 2.6) despite broadly similar anticoagulation regimens utilized for iSVT treatment between patients with and without active malignancy. Taken together, these findings suggest that cancer-associated iSVT is not benign as the VTE risk remains high despite guideline-concordant SVT therapy.

This highlights that the standard SVT treatment paradigm of 45 days of prophylactic dose anticoagulation is insufficient for at least a subset of cancer patients with iSVT and there is a potential role for longer term and higher intensity anticoagulation treatment for these patients, perhaps at least 3 months on treatment doses. There are some previously published randomized trials exploring the use of therapeutic anticoagulation for iSVT. The “Superficial Thrombophlebitis Treated by Enoxaparin” STENOX randomized controlled study identified that prophylactic (40 mg SC daily) and therapeutic dose (1.5 mg/kg SC daily) enoxaparin as well as oral nonsteroidal anti-inflammatory agent (tenoxicam 20 mg PO daily) for 8–12 days all significantly reduced the incidence of DVT or SVT extension by 12 days in patients with lower extremity iSVT with no major hemorrhage, but only included a total of 6 patients with cancer (1.4% of the total sample).²³ In another randomized trial, the VESALIO investigators found similar rates of SVT extension and incident VTE in 3 months between patients with iSVT treated with prophylactic or therapeutic doses of nadroparin for 1 month with no major bleeding; however, they excluded patients with active cancer.²⁴ Furthermore, given the persistent incidence of VTE events beyond 3 months in cancer-associated iSVT highlighted by the studies with longer follow-up, indefinite anticoagulation with prophylactic dose anticoagulation should be considered for select patients with iSVT as long as the malignancy remains active. It may be helpful to employ validated risk-stratification tools for cancer-related VTE such as the Khorana score alongside with consideration of the patient’s bleeding risk to determine optimal candidates for indefinite prophylactic anticoagulation.²⁵ There is already randomized controlled evidence through the AVERT trial that ambulatory patients with cancer at intermediate-to-high risk of VTE (Khorana score ≥ 2) who were starting chemotherapy will benefit from low-dose apixaban 2.5 mg PO BID.²⁶ Recently, we demonstrated that risk is further increased in patient with common thrombophilias.²⁷

The current evidence base for cancer-associated iSVT is dominated by observational cohorts with heterogeneity in SVT phenotype definitions, symptom-status reporting, anticoagulant intensity/duration, and follow-up windows. Key evidence gaps include: i) absence of published randomized controlled trials exploring the treatment of iSVT in patients with active malignancy; ii) limited reporting of clinically relevant nonmajor bleeding and other patient-centered outcomes (hospitalization, VTE-specific mortality); iii) inadequate length of follow-up beyond 3 months, despite signals of both early clustering and late events; and iv) whether additional biomarkers or thrombophilia testing may help select the highest risk patients.^{28,29,30} These observations provide a strong impetus for randomized controlled trials in cancer-iSVT comparing standard SVT regimens (45 days) against intensified and/or extended-duration anticoagulation strategies (e.g., ≥ 3 months, or

longer prophylactic-intensity secondary prevention), with stratification by cancer status (metastatic vs nonmetastatic), bleeding risk modifiers (intraluminal GI/GU tumor sites, baseline thrombocytopenia/coagulopathy), and anatomic SVT phenotype (junction distance, length, multifocality, upper or lower extremity, catheter-associated).³¹ As a final point, the vast majority of patients we have discussed had lower extremity SVT. We believe upper-extremity SVT, especially catheter-related, should be studied separately.

Conclusions

Cancer-associated iSVT is often not benign. Observational evidence synthesized in the recently published systematic review and meta-analysis IROVAM-iSVT with an updated systematic literature search up to December 2025 indicates substantial subsequent VTE risk despite guideline-appropriate anticoagulation. Although, it seems clear that VTE recurrences in cancer-associated iSVT are much higher than noncancer-associated iSVT, and indeed seem very clinically relevant one can argue the conclusions on bleeding, deaths, and comparisons to patients with cancer-associated DVT/PE are limited by relatively few patients informing the outcomes and incomplete reporting on anticoagulation usage. There is a need for randomized controlled trials to define the optimal intensity and duration for the treatment of iSVT in patients with active malignancy.

References

- Sorensen HT, Pedersen L, van Es N, et al. Impact of venous thromboembolism on the mortality in patients with cancer: a population-based cohort study. *Lancet Reg Health Eur* 2023; 34:100739.
- Grilz E, Posch F, Nopp S, et al. Relative risk of arterial and venous thromboembolism in persons with cancer vs. persons without cancer: a nationwide analysis. *Eur Heart J* 2021;42:2 299-307.
- Khorana AA, Mackman N, Falanga A, et al. Cancer-associated venous thromboembolism. *Nat Rev Dis Primers* 2022;8:11.
- Betts MB, Liu X, Junqueira DR, et al. Risk of venous thromboembolism by cancer type: a network meta-analysis. *Semin Thromb Hemost* 2024;50:328-41.
- Lyman GH, Carrier M, Ay C, et al. American Society of Hematology 2021 guidelines for management of venous thromboembolism: prevention and treatment in patients with cancer. *Blood Adv* 2021;5:927-74.
- Geersing GJ, Cazemier S, Rutten F, et al. Incidence of superficial venous thrombosis in primary care and risk of subsequent venous thromboembolic sequelae: a retrospective cohort study performed with routine healthcare data from the Netherlands. *BMJ Open* 2018;8:e019967.
- Frappe P, Buchmuller-Cordier A, Bertolotti L, et al. Annual diagnosis rate of superficial vein thrombosis of the lower limbs: the STEPH community-based study. *J Thromb Haemost* 2014;12:831-8.
- Hirmerova J, Seidlerova J, Subrt I, Hajsmanova Z. Prevalence of cancer in patients with superficial vein thrombosis and its clinical importance. *J Vasc Surg Venous Lymphat Disord* 2022;10:26-32.
- Stevens SM, Woller SC, Baumann Kreuziger L, et al. Antithrombotic therapy for VTE disease: compendium and review of CHEST guidelines 2012-2021. *Chest* 2024;166: 388-404.
- Decousus H, Prandoni P, Mismetti P, et al. Fondaparinux for the treatment of superficial-vein thrombosis in the legs. *N Engl J Med* 2010;363:1222-32.
- Beyer-Westendorf J, Schellong SM, Gerlach H, et al. Prevention of thromboembolic complications in patients with superficial-vein thrombosis given rivaroxaban or fondaparinux: the open-label, randomised, non-inferiority SURPRISE phase 3b trial. *Lancet Haematol* 2017;4: e105-13.
- Bailey AJM, Luo OD, Zhou SQ, Wells PS. The incidence and risk of venous thromboembolism in patients with active malignancy and isolated superficial venous thrombosis: a systematic review and meta-analysis (the IROVAM-iSVT review). *J Thromb Haemost* 2025;23:1824-37.
- DerSimonian R, Laird N. Meta-analysis in clinical trials. *Control Clin Trials* 1986;7:177-88.
- Langer F, Gerlach HE, Schimke A, et al. Clinical outcomes of cancer-associated isolated superficial vein thrombosis in daily practice. *Thromb Res* 2022;220:145-52.
- Galanaud JP, Blaise S, Sevestre MA, et al. Long-term outcomes of isolated superficial vein thrombosis in patients with active cancer. *Thromb Res* 2018;171:179-86.
- Ghenassia-Fouillet L, Morel A, Frappe P, et al. Management of superficial venous thrombosis in unevaluated situations: Cancer, severe renal impairment, pregnancy and post-partum. *Phlebology* 2021;36:464-72.
- Debourdeau P, Bertolotti L, Font C, et al. Three-month outcomes in cancer patients with superficial or deep vein thrombosis in the lower limbs: results from the RIETE registry. *Cancers (Basel)* 2023;15:2034.
- Diez-Vidal A, Gomez Lopez J, Rodriguez Fuertes P, et al. Superficial vein thrombosis and its relationship with malignancies: a prospective observational study. *J Thromb Thrombolysis* 2024;57:650-7.
- Bell LN, Berg RL, Schmelzer JR, et al. Thromboembolic complications following a first isolated episode of superficial vein thrombosis: a cross-sectional retrospective study. *J Thromb Thrombolysis* 2017;43:31-7.
- Beddar Chaib F, Jimenez Hernandez S, Pedrajas Navas JM, et al. Impact of emergency department management of isolated superficial vein thrombosis of the lower limbs: a secondary analysis of data from the ALTAMIRA study. *Emergencias* 2023;35:109-16.
- Barco S, Pomero F, Di Minno MND, et al. Clinical course of patients with symptomatic isolated superficial vein thrombosis: the ICARO follow-up study. *J Thromb Haemost* 2017;15:2176-83.
- Lobastov K, Dubar E, Schastlivtsev I, Bargandzhiya A. A systematic review and meta-analysis for the association between duration of anticoagulation therapy and the risk of venous thromboembolism in patients with lower limb superficial venous thrombosis. *J Vasc Surg Venous Lymphat Disord* 2024;12:101726.
- Superficial Thrombophlebitis Treated By Enoxaparin Study

- G. A pilot randomized double-blind comparison of a low-molecular-weight heparin, a nonsteroidal anti-inflammatory agent, and placebo in the treatment of superficial vein thrombosis. *Arch Intern Med* 2003;163:1657-63.
24. Prandoni P, Tormene D, Pesavento R, Vesalio Investigators Group. High vs. low doses of low-molecular-weight heparin for the treatment of superficial vein thrombosis of the legs: a double-blind, randomized trial. *J Thromb Haemost* 2005;3:1152-7.
 25. Khorana AA, Kuderer NM, Culakova E, et al. Development and validation of a predictive model for chemotherapy-associated thrombosis. *Blood* 2008;111:4902-7.
 26. Carrier M, Abou-Nassar K, Mallick R, et al. Apixaban to prevent venous thromboembolism in patients with cancer. *N Engl J Med* 2019;380:711-9.
 27. Roy DC, Wang T-Z, Carrier M, et al. Thrombophilia gene mutations predict venous thromboembolism in ambulatory cancer patients receiving chemotherapy. *J Thromb Haemost* 2023;21:3184-92.
 28. Roy DC, Wang TF, Lun R, et al. Circulating blood biomarkers and risk of venous thromboembolism in cancer patients: a systematic review and meta-analysis. *Thromb Haemost* 2024;124:1117-33.
 29. Roy DC, Wang TF, Lun R, et al. Inherited thrombophilia gene mutations and risk of venous thromboembolism in patients with cancer: A systematic review and meta-analysis. *Am J Hematol* 2024;99:577-85.
 30. Roy DC, Wang TF, Lun R, et al. Inherited thrombophilia gene mutations and risk of venous thromboembolism in patients with cancer: A systematic review and meta-analysis. *Am J Hematol* 2024;99:577-85.
 31. Khan F, Tritschler T, Marx CE, Lanting V, et al. Predictors of recurrent venous thromboembolism and bleeding in patients with cancer: a meta-analysis. *Eur Heart J* 2025:ehaf453. Online ahead of print.