Optimizing Anti-Thrombotic Therapy After Catheter-Directed Interventions for DVT and PTS

Recommendations for anti-thrombotic and anti-inflammatory regimens after deep venous disease interventions based on patient presentation, comorbidities, and type of intervention.

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Acute deep vein thrombosis (DVT) affects 300,000 to 600,000 people in the United States each year.[1] Nearly half of patients with acute DVT may eventually develop postthrombotic syndrome (PTS), characterized by chronic lower extremity swelling, heaviness, discomfort, color changes, and ulceration, with one-quarter of patients developing moderate to severe symptoms.[2] Acute and chronic venous disease have major negative health and socioeconomic consequences.[2-4]

Catheter-based interventions, including endovascular reconstruction, are being effectively used to relieve venous obstruction, with corresponding improvements in quality of life. Long-term venous and stent patency is a necessary consideration to maintain these outcomes, and appropriate anti-thrombotic regimens are a key ingredient for successful outcomes.

The role of the interventionalist in the acute and chronic DVT setting includes procedural and periprocedural management. Endovascular specialists who are consulted for these patients should stay updated on the procedural and clinical aspects and closely follow the patient's anticoagulation (AC) plan if not initiating it themselves. Close collaboration with hematology and/or vascular medicine specialists is necessary for continuity of care and optimizing patient outcomes.

Discussions with patients about their AC plans are best initiated prior to planned procedures so that patients can ask questions and be prepared to follow through. This is especially true in chronic venous disease because patients are seen and procedures typically planned in outpatient clinic settings. Postintervention discussions with the patient should be conducted prior to discharge, with a clearly communicated AC course and follow-up plan. Scheduling a postdischarge follow-up visit with the proceduralist, hematologist, and/or vascular medicine specialist, ideally prior to discharge, is necessary for managing AC, addressing patient concerns, encouraging medication and compression adherence, and monitoring symptoms over time.

ACUTE DVT

Algorithms for AC after acute DVT vary between venous disease experts. In a 2018 international survey of interventional radiologists, vascular surgeons, and hematologists, the majority of practitioners preferred initial use of low-molecular-weight heparin (LMWH) for 2 to 6 weeks and 6 to 12 months of AC, with approximately half including antiplatelet agents in the treatment regimen.[5] Patients with major ongoing risk factors or unprovoked DVT may benefit from longer durations of therapy.

Retrospective evidence suggests that AC with or without the addition of an antiplatelet agent is appropriate for postintervention management in acute

thrombotic disease. Rivaroxaban was found to have similar safety and effectiveness as vitamin K antagonists (VKAs) after venous stent placement, and overall 2-year primary and secondary patency rates were 82% and 95%, respectively.[6] Despite decreased patency of venous stents in chronic occlusions, clinicians can still expect primary patency rates of approximately 75% at 3 months, 64% at 1 year, and 59% at 3 years, as demonstrated in a recent retrospective review.[7] Additionally, LMWH for at least 10 days (usually 4 weeks) postprocedure reduced the odds of early thrombosis.[7] LMWH for longer durations may be of most benefit in patients with underlying high inflammatory states. Antiplatelet therapy may also be helpful in this setting. One study showed that triple therapy (one anticoagulant and two antiplatelet agents) reduced the odds of in-stent thrombosis compared to dual antiplatelet therapy (DAPT) alone.[8] There was no difference in major or minor bleeding risk among the four treatment groups, including triple therapy, DAPT, AC with a single antiplatelet agent, and AC only. In a study of 100 patients who underwent iliocaval stent placement followed by treatment with a direct oral anticoagulant (DOAC), a subgroup analysis demonstrated 12-month primary patency rates of 92% for acute thrombotic and 93% for chronic thrombotic venous disease.[9]

CHRONIC VENOUS DISEASE

There is a paucity of data on anti-thrombotic management after interventions for patients with chronic venous disease, who typically present with PTS. The 2018 international survey found that in the setting of PTS with chronic thrombosis, the majority of practitioners prescribe lifelong AC. The consensus recommended lifelong AC with or without an antiplatelet agent for those with a history of multiple prior venous thromboembolism (VTE) events who underwent venous stent placement.[5]

ANTI-THROMBOTIC THERAPIES

Anticoagulants

Anticoagulants are the basis of anti-thrombotic therapy after venous intervention, and they act by interrupting the coagulation cascade.

Warfarin. An oral VKA, warfarin inhibits the epoxide reductase complex through competitive inhibition, decreasing the intracellular vitamin K form needed for factor II, VII, IX, and X as well as protein C and S production. A 5-day AC bridge is administered to reach therapeutic levels. Frequent laboratory testing while on warfarin is needed to titrate dosage for an optimum international normalized ratio (INR) of 2 to 3. INR fluctuations, drug interactions, and the need for regular lab draws can make this option inconvenient. This class is the oldest oral anticoagulant still used.[10]

Unfractionated heparin (UFH). UFH is a glycosaminoglycan that binds to anti-thrombin, inactivates thrombin and factor Xa, and inhibits thrombin production, platelet activation, and factor V and VIII activation.[11] Heparin is widely used in inpatient and periprocedural settings and as a bridge to other anticoagulants.

LMWH. Including enoxaparin, tinzaparin, and dalteparin, LMWHs share a similar mode of action as UFH.[12] Studies have shown that LMWH outperforms warfarin and factor Xa inhibitors in treating acute lower extremity DVT in native veins, with higher vein recanalization rates at 6 and 12 months.[7,13] Marston et al found that LMWH for > 10 days after intervention helps reduce in-stent rethrombosis, which may reduce PTS.[7] In another study, long-term LMWH (tinzaparin) prevented PTS better than a LWMH bridge to warfarin in 480 patients with iliac and/or noniliac DVT.[14] Of note, enoxaparin requires a twice-daily dosing for therapeutic effect and is only available as subcutaneous injection, which can be a deterrent to some patients.

DOACs. DOACs used for treating VTE include oral factor Xa inhibitors (eg, rivaroxaban, apixaban, edoxaban) and direct thrombin inhibitors (eg, dabigatran). These are convenient because they do not require lab monitoring or titration. According to the EINSTEIN-DVT, EINSTEIN-PE, and AMPLIFY studies, rivaroxaban and apixaban are noninferior to warfarin in VTE prevention and treatment.[15-17] Additionally, apixaban has a lower bleeding risk than warfarin. Due to their safety profile and ease of administration, the International Society on Thrombosis and Haemostasis recommends DOACs over VKAs for outpatient VTE prevention in patients with active cancer except luminal gastrointestinal and genitourinary cancers.[18] Patients and prescribers also tend to prefer DOACs because they have consistent regimens that are easier to prescribe and explain.

Fondaparinux. This synthetic factor Xa inhibitor binds anti-thrombin and inhibits factor Xa, similar to heparinoids. It works similarly to enoxaparin or UFH for DVT in perioperative prophylaxis for orthopedic, gastrointestinal, and acute coronary syndromes.[19]

Argatroban and bivalirudin. Argatroban and bivalirudin are parenteral direct thrombin inhibitors. These drugs are usually administered in the inpatient setting and used for AC in heparin-induced thrombocytopenia.[20] Argatroban is liver metabolized and bivalirudin is renally eliminated, so dosages should be adjusted in liver and kidney disease, respectively. Little data are available on these agents after deep venous procedures and stenting.

Antiplatelet Agents

Evidence suggests that there may be a benefit to adding an antiplatelet medication to an anticoagulant after venous stenting.[21] Clopidogrel and acetylsalicylic acid (ASA) are the mainstays of antiplatelet therapy in venous disease therapy. In 62 patients with thrombotic and nonthrombotic venous illness, antiplatelet agents with AC after iliocaval venous stent placement increased primary patency compared to AC alone.[21] However, bleeding incidence increased with the addition of the antiplatelet agent. In another study of 87 patients with inferior vena cava or iliofemoral stents, there was no significant change in in-stent stenosis or thrombosis rates with antiplatelet drugs in the anti-thrombotic regimen.[8]

Aspirin. Aspirin, or ASA, is a widely used medicine for arterial and venous thromboembolic disease. A cyclooxygenase (COX) antagonist, ASA inactivates COX-1 and COX-2, reducing prostaglandin metabolism and thromboxane A2 production. ASA preferentially inhibits COX-1, preventing platelet aggregation at low doses.[22] In a postanalysis study of a trial comparing endovenous intervention and AC for lower extremity acute DVT, it was found that ASA use reduced PTS, suggesting a role for antiplatelet therapy.[23]

P2Y12 inhibitors (clopidogrel, ticagrelor, prasugrel). Clopidogrel, an adenosine diphosphate (ADP) receptor antagonist, stops platelet aggregation by binding to the ADP platelet receptor (P2Y12) and activating the glycoprotein IIb/IIIa complex. Ticagrelor and prasugrel are not well-studied in venous disease. In coronary trials, ticagrelor has a higher bleeding risk than clopidogrel but better platelet inhibition and lower arterial stent restenosis/thrombosis rates.[24,25] Prasugrel has a higher bleeding risk than clopidogrel despite equivalent clinical benefit in acute coronary syndromes.[26]

ANTI-INFLAMMATORY THERAPY IN EVOLUTION

In 1856, Rudolf Virchow introduced the triad of endothelial injury, hypercoagulability, and venous stasis, widely recognized in the etiology of thrombosis.[27] Today, the role of inflammation is increasingly being recognized. Common proinflammatory states include obesity, infection, cancer, and the postoperative period.[28] Many cytokines and chemokines mediate inflammatory and anti-inflammatory responses that both form and resolve thrombus.[29] Endothelial cells, platelets, and leukocytes can activate tissue factor and trigger the coagulation cascade, resulting in thrombosis even in otherwise normal blood vessels.[30] The potential application of anti-inflammatory agents and venoactive substances are increasingly acknowledged in the management of DVT and may have a role in management after venous stenting.

Statins have shown mixed results in addressing the inflammatory component of thrombosis. A prospective randomized study of 234 patients with DVT showed a significant decrease in incidence of PTS in the group treated with LMWH plus rosuvastatin compared to LWMH only.[31] Studies have found that rosuvastatin can decrease the occurrence of symptomatic venous thrombosis in patients with elevated C-reactive protein.[32] Animal models have suggested that statins can reduce platelet aggregation, cytokine levels, and vein wall scarring.[33,34] Conversely, a recent multicenter randomized controlled pilot study was performed evaluating the role of rosuvastatin in the development of PTS at 6 months in patients with a newly diagnosed DVT. The study randomized 312 patients to anticoagulation plus 20 mg rosuvastatin or anticoagulation alone for 180 days. The study did not find a reduction in PTS incidence or improvement of Villalta score. We agree with the authors of the study that further studies with longer duration of rosuvastatin (and other agents) are needed.[35]

E-selectin inhibition of iliac vein thrombosis in a primate animal model resulted in improved vein recanalization, decreased vein wall inflammation, and intimal thickness and fibrosis compared to E-selectin inhibitor plus LMWH and the untreated control group.[36]

Preclinical studies of factor XI inhibitors show promise, with reduction of VTE occurrences by 41% when compared to LMWH.[37] Additionally, a meta-analysis revealed a 59% decrease in bleeding risk in individuals receiving a factor XI inhibitor as opposed to enoxaparin.[37]

Venoactive drugs (VADs), including flavonoids, saponins, calcium dobesilate, and plant extracts offer significant relief for symptoms of chronic venous disease by improving venous tone, enhancing microcirculation, and reducing capillary permeability. These actions alleviate symptoms such as leg heaviness, swelling, and pain. Additionally, VADs exhibit anti-inflammatory and antioxidant properties, reducing oxidative stress and supporting endothelial function. Micronized purified flavonoid fraction, which contains both diosmin and hesperidin, is highly effective in reducing edema, alleviating pain, and accelerating venous ulcer healing, especially when paired with compression therapy.[38,39]

These agents are gaining traction in the United States, where they are available primarily as nutritional supplements. Ongoing randomized controlled trials may help further define the role of these agents in PTS.[40] However, at this time, the authors of this article frequently recommend these substances to patients for symptomatic relief of PTS.

POSTINTERVENTION ANTI-THROMBOTIC THERAPY RECOMMENDATIONS

In 2022, a multidisciplinary expert panel of venous experts published recommendations on postvenous intervention regimens, dividing patients into high and low risk for postprocedure in-stent thrombosis.[27] High-risk patients were those with underlying thrombophilia, a malignant obstruction or active cancer, history of unprovoked or recurrent DVTs, or prior stent thrombosis. Procedural factors resulting in high-thrombotic-risk grouping included poor venous inflow or outflow, stent compression or kinking, and other stent-related factors, such as length and diameter.

The expert panel recommendations included postintervention anticoagulant only and anticoagulant plus antiplatelet regimens, stratified by low versus high risk. All regimens included initial use of therapeutic LMWH for 30 days, with the option to add an antiplatelet agent in high-thrombotic-risk cases.

In the AC-alone regimen for high-thrombotic-risk patients, LMWH (typically enoxaparin) was continued for 1 to 6 months. In all other cases, the anticoagulant was switched to a DOAC for this period, with the option to add an antiplatelet agent in high-thrombotic-risk cases. After 6 months, DOAC was stopped or reduced in all low-thrombotic-risk cases and continued in all high-thrombotic-risk cases, with the option to add an antiplatelet agent.

Authors' Recommendations

For acute DVT, we recommend 2 to 6 weeks of LMWH with or without an antiplatelet agent, followed by DOAC with or without an antiplatelet agent for up to 6 months. The long-term plan depends on the type of thrombosis (provoked vs unprovoked) and the patient's overall hypercoagulable state.

For chronic DVT, we recommend indefinite (often lifelong) AC, with or without an antiplatelet agent provided a tolerable bleeding risk.

CONCLUSION AND FUTURE DIRECTIONS

The optimal anti-thrombotic regimen and duration are based on patient presentation, comorbidities, and types of venous intervention. Generally, LMWH is preferred in the initial postprocedural setting.

Current larger randomized controlled trials are investigating outcomes of venous stenting after deep venous thrombosis, including the C-TRACT trial and ARIVA trials. Findings from these studies and many other ongoing multidisciplinary efforts will hopefully guide optimal anti-thrombotic guidelines after deep venous interventions. ■

1. Beckman MG, Hooper WC, Critchley SE, Ortel TL. Venous thromboembolism: a public health concern. Am J Prev Med. 2010;38(4 suppl):S495-S501. doi: 10.1016/j.amepre.2009.12.017
2. Vedantham S, Goldhaber SZ, Julian JA, et al. Pharmacomechanical catheter-directed thrombolysis for deep-vein thrombosis. N Engl J Med. 2017;377:2240-2252. doi: 10.1056/NEJMoa1615066
3. de Wolf MA, Wittens CH, Kahn SR. Incidence and risk factors of the post-thrombotic syndrome. Phlebology. 2012;27(suppl 1):85-94. doi: 10.1258/phleb.2011.012s06
4. Pesavento R, Bernardi E, Concolato A, et al. Postthrombotic syndrome. Semin Thromb Hemost. 2006;32:744-751. doi: 10.1055/s-2006-951460
5. Milinis K, Thapar A, Shalhoub J, Davies AH. Antithrombotic therapy following venous stenting: international Delphi consensus. Eur J Vasc Endovasc Surg. 2018;55:537-544. doi: 10.1016/j.ejvs.2018.01.007
6. Sebastian T, Hakki LO, Spirk D, et al. Rivaroxaban or vitamin-K antagonists following early endovascular thrombus removal and stent placement for acute iliofemoral deep vein thrombosis. Thromb Res. 2018;172:86-93. doi: 10.1016/j.thromres.2018.10.027
7. Marston WA, Browder SE, Iles K, et al. Early thrombosis after iliac stenting for venous outflow occlusion is related to disease severity and type of anticoagulation. J Vasc Surg Venous Lymphat Disord. 2021;9:1399-1407.e1. doi: 10.1016/j.jvsv.2021.02.012
8. Lin C, Martin KA, Wang M, et al. Long-term antithrombotic therapy after venous stent placement. Phlebology. 2020;35:402-408. doi: 10.1177/0268355519893819
9. Hays K, Jolly M, Silver M, et al. Outcomes of endovascular venous stenting in patients on direct oral anticoagulants and antiplatelet therapy at a tertiary referral center. J Vasc Surg Venous Lymphat Disord. 2021;9:753-759.e1. doi: 10.1016/j.jvsv.2020.08.024
10. Hirsh J, Fuster V, Ansell J, Halperin JL. American Heart Association/American College of Cardiology Foundation guide to warfarin therapy. Circulation. 2003;107:1692-1711. doi: 10.1161/01.CIR.0000063575.17904.4E
11. Hirsh J, Anand SS, Halperin JL, Fuster V. Mechanism of action and pharmacology of unfractionated heparin. Arterioscler Thromb Vasc Biol. 2001;21:1094-1096. doi: 10.1161/hq0701.093686
12. Hirsh J. Low-molecular-weight heparin. Circulation. 1998;98:1575-1582. doi: 10.1161/01.CIR.98.15.1575
13. Romera A, Cairols MA, Vila-Coll R, et al. A randomised open-label trial comparing long-term sub-cutaneous low-molecular-weight heparin compared with oral-anticoagulant therapy in the treatment of deep venous thrombosis. Eur J Vasc Endovasc Surg. 2009;37:349-356. doi: 10.1016/j.ejvs.2008.11.030
14. Hull RD, Liang J, Merali T. Effect of long-term LMWH on post-thrombotic syndrome in patients with iliac/noniliac venous thrombosis: a subanalysis from the home-LITE study. Clin Appl Thromb Hemost. 2013;19:476-481. doi: 10.1177/1076029613481845
15. Agnelli G, Buller HR, Cohen A, et al. Oral apixaban for the treatment of acute venous thromboembolism. N Engl J Med. 2013;369:799-808. doi: 10.1056/NEJMoa1302507
16. EINSTEIN Investigators; Bauersachs R, Berkowitz SD, Brenner B, et al. Oral rivaroxaban for symptomatic venous thromboembolism. N Engl J Med. 2010;363:2499-2510. doi: 10.1056/NEJMoa1007903
17. EINSTEIN–PE Investigators; Büller HR, Prins MH, Lensin AW, et al. Oral rivaroxaban for the treatment of symptomatic pulmonary embolism. N Engl J Med. 2012;366:1287-1297. doi: 10.1056/NEJMoa1113572
18. Khorana AA, Noble S, Lee AYY, et al. Role of direct oral anticoagulants in the treatment of cancer-associated venous thromboembolism: guidance from the SSC of the ISTH. J Thromb Haemost. 2018;16:1891-1894. doi: 10.1111/jth.14219
19. Turpie AG. The safety of fondaparinux for the prevention and treatment of venous thromboembolism. Expert Opin Drug Saf. 2005;4:707-721. doi: 10.1517/14740338.4.4.707
20. Cuker A, Arepally GM, Chong BH, et al. American Society of Hematology 2018 guidelines for management of venous thromboembolism: heparin-induced thrombocytopenia. Blood Adv. 2018;2:3360-3392. doi: 10.1182/bloodadvances.2018024489
21. Endo M, Jahangiri Y, Horikawa M, et al. Antiplatelet therapy is associated with stent patency after iliocaval venous stenting. Cardiovasc Intervent Radiol. 2018;41:1691-1698. doi: 10.1007/s00270-018-2062-5
22. Altenburg A, Haage P. Antiplatelet and anticoagulant drugs in interventional radiology. Cardiovasc Intervent Radiol. 2012;35:30-42. doi: 10.1007/s00270-011-0204-0
23. Sharifi M, Mehdipour M, Bay C, et al. Endovenous therapy for deep venous thrombosis: the TORPEDO trial. Catheter Cardiovasc Interv. 2010;76:316-325. doi: 10.1002/ccd.22638
24. Turgeon RD, Koshman SL, Youngson E, et al. Association of ticagrelor vs clopidogrel with major adverse coronary events in patients with acute coronary syndrome undergoing percutaneous coronary intervention. JAMA Intern Med. 2020;180:420-428. doi: 10.1001/jamainternmed.2019.6447
25. Wallentin L, Becker RC, Budaj A, et al. Ticagrelor versus clopidogrel in patients with acute coronary syndromes. N Engl J Med. 2009;361:1045-1057. doi: 10.1056/NEJMoa0904327
26. Jia M, Li Z, Chu H, et al. Novel oral P2Y12 inhibitor prasugrel vs. clopidogrel in patients with acute coronary syndrome: evidence based on 6 studies. Med Sci Monit. 2015;21:1131-1137. doi: 10.12659/MSM.893914
27. Kishore S, Khaja MS, Thornburg B, et al. Antithrombotic therapy after venous interventions: AJR expert panel narrative review. AJR Am J Roentgenol. 2022;219:175-187. doi: 10.2214/AJR.22.27413
28. Borgel D, Bianchini E, Lasne D, et al. Inflammation in deep vein thrombosis: a therapeutic target? Hematology. 2019;24:742-750. doi: 10.1080/16078454.2019.1687144
29. Najem MY, Couturaud F, Lemarié CA. Cytokine and chemokine regulation of venous thromboembolism. J Thromb Haemost. 2020;18:1009-1019. doi: 10.1111/jth.14759
30. Branchford BR, Carpenter SL. The role of inflammation in venous thromboembolism. Front Pediatr. 2018;6:142. doi: 10.3389/fped.2018.00142
31. San Norberto EM, Gastambide MV, Taylor JH, et al. Effects of rosuvastatin as an adjuvant treatment for deep vein thrombosis. Vasa. 2016;45:133-140. doi: 10.1024/0301-1526/a000507
32. Glynn RJ, Danielson E, Fonseca FA, et al. A randomized trial of rosuvastatin in the prevention of venous thromboembolism. N Engl J Med. 2009;360:1851-1861. doi: 10.1056/NEJMoa0900241
33. Feng Y, Lei B, Zhang F, et al. Anti-inflammatory effects of simvastatin during the resolution phase of experimentally formed venous thrombi. J Investig Med. 2017;65:999-1007. doi: 10.1136/jim-2017-000442
34. Kessinger CW, Kim JW, Henke PK, et al. Statins improve the resolution of established murine venous thrombosis: reductions in thrombus burden and vein wall scarring. PLoS ONE. 2015;10:e0116621. doi: 10.1371/journal.pone.0116621
35. Delluc A, Ghanima W, Kovacs MJ, et al. Prevention of post-thrombotic syndrome with rosuvastatin: A multicenter randomized controlled pilot trial (SAVER). Thromb Res. 2022;213:119-124. doi: 10.1016/j.thromres.2022.03.014
36. Myers DD, Ning J, Lester P, et al. E-selectin inhibitor is superior to low-molecular-weight heparin for the treatment of experimental venous thrombosis. J Vasc Surg Venous Lymphat Disord. 2022;10:211-220. doi: 10.1016/j.jvsv.2020.12.086
37. Nopp S, Kraemmer D, Ay C. Factor XI inhibitors for prevention and treatment of venous thromboembolism: a review on the

rationale and update on current evidence. Front Cardiovasc Med. 2022;9:903029. doi: 10.3389/fcvm.2022.903029
38. Li KX, Diendéré G, Galanaud JP, et al. Micronized purified flavonoid fraction for the treatment of chronic venous insufficiency, with a focus on postthrombotic syndrome: a narrative review. Res Pract Thromb Haemost. 2021;5:e12527. doi: 10.1002/rth2.12527
39. Gloviczki P, Lawrence PF, Wasan SM, et al. The 2023 Society for Vascular Surgery, American Venous Forum, and American Vein and Lymphatic Society clinical practice guidelines for the management of varicose veins of the lower extremities. Part II: endorsed by the Society of Interventional Radiology and the Society for Vascular Medicine. J Vasc Surg Venous Lymphat Disord. 2024;12:101670. Published correction appears in J Vasc Surg Venous Lymphat Disord. 2024;12:101923. doi: 10.1016/j.jvsv.2023.08.011.
40. Galanaud JP, Abdulrehman J, Lazo-Langner A, et al. MUFFIN-PTS trial, micronized purified flavonoid fraction for the treatment of post-thrombotic syndrome: protocol of a randomised controlled trial. BMJ Open. 2021;11:e049557. doi: 10.1136/bmjopen-2021-049557

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