

Anticoagulant treatment in patients with internal jugular vein thrombosis: a case series

Arianna Pannunzio,¹ Ilaria Maria Palumbo,¹ Luca Tretola,² Elena Ciambella,² Valentina Castellani,³ Maddalena Ragazzo,⁴ Pasquale Pignatelli,² Emanuele Valeriani¹

Departments of ¹General Surgery and Surgical Specialty Paride Stefanini, ²Medical and Cardiovascular Sciences, ³Translational and Precision Medicine, Sapienza University of Rome; ⁴Physician, *Alessandrino Acismon* Long-term Care Facility, Rome, Italy

ABSTRACT

Internal jugular vein thrombosis (IJVT) is an uncommon form of venous thrombosis that may be associated with either transient or persistent risk factors. According to current international guidelines, vitamin K antagonists (VKAs) represent the first-line anticoagulant therapy; however, data on the use of direct oral anticoagulants (DOACs) in this setting remain limited. The aim of this case series was to describe the clinical characteristics and to compare the efficacy and safety outcomes of VKAs, DOACs, and parenteral anticoagulants in patients with IJVT. In this retrospective single-center study, we consecutively enrolled 29 patients, 17 were treated with VKAs, 10 with DOACs and 2 with parenteral anticoagulants. Among five patients who experienced recurrence of thrombosis, three cases occurred following discontinuation of anticoagulant therapy. The remaining two recurrences were observed during ongoing treatment. Five bleeding events were reported, with no difference among the three groups and none classified as major bleeding. These findings suggest that DOACs may be a safe and effective option for anticoagulant treatment in IJVT, as evidenced by low recurrence rates during therapy and the absence of major bleeding events.

Corresponding author: Arianna Pannunzio, Department of General Surgery and Surgical Specialty Paride Stefanini, Sapienza University of Rome, 00161 Roma (RM), Italy.
E-mail: arianna.pannunzio@uniroma1.it

Conflict of interest: the authors declare that they have no competing interests.

Funding: none.

Contributions: AP and IMP share co-first authorship; EV and PP share co-senior authorship. All the authors read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Ethics approval and consent to participate: the study was approved by the local Ethics Committee (no. 6573). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their enrollment in the study.

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

Received: 6 December 2025.

Accepted: 28 August 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; 5:422

doi:10.4081/btvb.2026.422

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

Key words: atypical thrombosis, anticoagulation, jugular vein.

Introduction

Internal jugular vein thrombosis (IJVT) is classified as atypical thrombosis of the upper extremity deep vein thrombosis (UEDVT). The prevalence of this form is between 0.5% and 3.47% of patients with deep vein thrombosis (DVT).^{1,2} The most common clinical presentation is arm edema and pain, but it can also be asymptomatic.²

In IJVT, transient mechanical factors are among the most frequent causes, including central venous catheter (CVC) placement and lateral cervical infections, such as Lemierre's syndrome.^{1,3} Persistent risk factors may also contribute, including active cancer and inherited or acquired thrombophilia.^{4,5} Notably, in cases of IJVT unrelated to CVC use, thrombus formation may represent the first manifestation of an occult malignancy, which should therefore be investigated.^{4,6} The prevalence of coagulation abnormalities in patients with UEDVT ranges from 8% to 61%.⁷ In a literature review, coagulation disorders accounted for 12.7% of IJVT cases, with factor V Leiden mutation being the most frequently identified abnormality.²

The main complications of UEDVT include pulmonary embolism (PE), post-thrombotic syndrome (PTS), and death. The reported incidence of PE is approximately 30%, while PTS occurs in 7-44% of patients.^{8,9} Although the risk of PE in UEDVT varies across studies, current evidence suggests that the risk of recurrent venous thromboembolism (VTE) and PE is comparable between UEDVT and lower extremity DVT (LEDVT). Consequently, the management of IJVT generally parallels that of other venous thromboembolic events, with anticoagulation representing the mainstay of treatment.¹⁰⁻¹²

In the absence of dedicated studies, current recommendations (grade 2C) support the use of anticoagulation in this setting.¹⁰ Ex-

isting guidelines suggest similar initial and long-term management strategies for patients with UEDVT and LEDVT.¹³ However, atypical thromboses are traditionally treated with vitamin K antagonists (VKAs), and evidence supporting the use of direct oral anticoagulants (DOACs) remains limited.¹⁴ As a result, the optimal therapeutic approach and the balance between efficacy and safety are still under debate. In addition, patients treated with VKAs require regular monitoring of the international normalized ratio (INR) to maintain therapeutic levels; however, poor adherence is common and may lead to suboptimal disease control.

Thrombosis associated with transient risk factors is treated with anticoagulation for 3 months once the provoking factor has been removed, whereas extended treatment (≥ 6 months) is considered in other cases.¹⁵ Removal of the main risk factor — such as a CVC — may reduce the risk of recurrence; however, this is often not feasible due to the ongoing need for central venous access.⁷ Current recommendations suggest that the catheter should not be removed if it remains functional and clinically necessary. In patients with CVC-related UEDVT in whom the catheter can be safely removed, a 3-month course of anticoagulation is generally recommended rather than extended therapy.¹⁵

Given the limited data on the use of DOACs in IJVT, the aim of this case series was to describe the clinical characteristics and to evaluate the efficacy and safety outcomes of VKAs, DOACs, and parenteral anticoagulants in patients with IJVT.

Case Report

We consecutively enrolled all patients diagnosed with IJVT who were referred to the Centre of Anticoagulant Therapies at Policlinico Umberto I, Rome, between 2014 and 2024.

The aim of this study was to describe the clinical characteristics of patients with IJVT and to identify potential differences among anticoagulant treatment regimens. We collected clinical

data including age, sex, prior anticoagulant or antiplatelet therapy, family history or previous episodes of venous thromboembolism (VTE), duration of anticoagulant treatment from the time of IJVT diagnosis, type of anticoagulant, and dosage.

All patients were screened for inherited and acquired thrombophilia, including factor II G20210A and factor V G1691A mutations, plasminogen activator inhibitor-1 (PAI-1) polymorphisms, MTHFR C677T and A1298C mutations, protein C and protein S levels, lupus anticoagulant (LAC), antithrombin III (ATIII) levels, anticardiolipin antibodies, and anti- $\beta 2$ -glycoprotein I antibodies.

The diagnosis of IJVT was established by color Doppler ultrasound. During follow-up, patients were evaluated at 3, 6, and 12 months, and annually thereafter the initiation of anticoagulant therapy. At each visit, anthropometric parameters, laboratory tests, and clinical outcomes were reassessed. Major bleeding and clinically relevant non-major bleeding (CRNMB) were defined according to the International Society on Thrombosis and Hemostasis (ISTH) criteria.^{16,17}

A total of 29 patients were included: 10 were treated with DOACs, 17 with VKAs, and 2 with parenteral anticoagulants. The mean age was 58 ± 13 years, with no significant differences among groups in terms of age or sex (44.8% women). The most frequent etiologies were CVC-related thrombosis (37.9%), cancer (20.7%) and Lemierre's syndrome (13.8%) (Table 1). All patients with Lemierre's syndrome were treated with VKAs, patients with cancer were preferentially treated with DOACs or parenteral anticoagulants, with a statistically significant difference ($p=0.001$).

No significant differences were observed among the three groups regarding thrombophilia, major comorbidities (e.g., history of cancer, type 2 diabetes mellitus, ischemic heart disease), or concomitant antiplatelet therapy. Although 51.7% of cases were classified as provoked, this did not translate into a significant difference in the duration of anticoagulation, which had a mean length of 37 months across all groups. This may be explained by the coexistence of persistent risk factors in many pa-

Table 1. Clinical characteristics of patients with jugular vein thrombosis.

	Total (n=29)	DOACs (n=10)	VKAs (n=17)	Parenteral (n=2)	P
Sex (F), %	13 (44.8)	4 (40.0)	8 (47.0)	1 (50)	0.927
Age, mean $\pm$ SD	58 $\pm$ 13	59 $\pm$ 14	59 $\pm$ 13	50 $\pm$ 20	0.725
Symptoms* %	10 (24.5)	4 (40.0)	6 (35.3)	0	0.470
Causes* %					
Lemierre's syndrome	4 (13.8)	0	4 (23.5)	0	0.001
CVC	11 (37.9)	4 (40.0)	6 (35.3)	1 (50)	
Cancer	6 (20.7)	2 (20.0)	3 (17.6)	1 (50)	
Cancer, %	11 (37.9)	4 (40.0)	5 (29.4)	1 (50)	0.148
Type2 DM, %	3 (10.3)	1 (10.0)	2 (11.7)	0	0.874
Ischaemic heart disease, %	4 (13.8)	0	4 (23.5)	0	0.194
Hypertension, %	9 (31.0)	4 (40.0)	5 (29.4)	0	0.523
CKD, %	5 (17.2)	0	5 (29.4)	0	0.119
Dyslipidemia, %	8 (27.6)	3 (30.0)	5 (29.4)	0	0.664
Antiplatelet, %	2 (6.9)	1 (10.0)	1 (5.9)	0	0.850
Thrombophilia, %	7 (24.2)	3 (30.0)	4 (23.5)	0	0.661
Length of treatment, mean $\pm$ SD	37 $\pm$ 36	39 $\pm$ 36	39 $\pm$ 38	18 $\pm$ 8	0.732

DOACs, direct oral anticoagulants; VKAs, vitamin K antagonists; SD, standard deviation; CVC, central venous catheter; DM, diabetes mellitus; CKD, chronic kidney disease.

*70% of patients have this data.

tients, leading to a sustained risk of recurrence and consequently prolonged therapy.

Among patients treated with DOACs, 8 received apixaban (initial dose 5 mg twice daily, followed by a reduced dose of 2.5 mg twice daily), and 2 received rivaroxaban (initial dose 20 mg once daily, followed by 10 mg once daily). These dosing regimens were chosen based on current guideline recommendations for treatment of deep venous thromboembolism, which suggest an initial full anticoagulant dose for acute management followed by a reduced prophylactic dose for secondary prevention. The initial full-dose phase typically lasted 3-6 weeks according to standard practice and patient-specific factors. In the VKA group, warfarin was the most used agent (94.1%). Among patients receiving parenteral anticoagulants, one was treated with low-molecular-weight heparin and one with fondaparinux. No treatment switches (e.g., from DOACs to VKAs or vice versa) were required based on laboratory findings, clinical considerations, or patient preference.

During follow-up, complete thrombus resolution was observed in 24.1% of patients. Five bleeding events were reported, with a higher prevalence in the VKAs group (23.5% vs 10.0% in the DOACs group and 0% in the parenteral group; $p=0.534$) (Table 2). These included two genitourinary CRNMB events, one episode of epistaxis, one case of anemia of unknown origin, and one conjunctival hemorrhage. No major bleeding events were recorded.

All-cause mortality occurred in two patients, both in the parenteral anticoagulant group, due to causes unrelated to anticoagulant therapy; this difference was statistically significant ($p=0.003$). A total of five thrombotic events were observed: two pulmonary embolisms, two cerebral venous thromboses, and one recurrence of IJVT. Among patients with recurrence, two had Lemierre's syndrome and one had CVC-related thrombosis; all events occurred after discontinuation of VKAs therapy.

Discussion

The available literature on IJVT is limited, consisting of case reports and small case series focusing on specific etiologies such as Lemierre's syndrome, catheter-related thrombosis, cancer, antiphospholipid syndrome, and inherited thrombophilia. To date, no randomized controlled trials have compared different anticoagulant treatment strategies in this setting.

The optimal management of DVT, including isolated IJVT, should be individualized, considering the patient's comorbidities,

concomitant medications, bleeding risk associated with anticoagulation, and risk of VTE recurrence. Factors such as recent surgery, trauma, active bleeding, or thrombocytopenia may increase bleeding risk. Conversely, even when a transient provoking factor has resolved, persistent or minor risk factors may contribute to a sustained risk of recurrence.

Our findings support the use of anticoagulant therapy to reduce the risk of VTE recurrence and to maintain venous patency. These results are consistent with previous evidence from a study of 142 hospitalized patients with IJVT, in whom cancer was present in 29.7% of cases CVCs in all patients.¹⁶ That study supported the use of anticoagulation — specifically low-molecular-weight heparin — in the acute setting, compared with alternative strategies such as superior vena cava filter placement or serial duplex ultrasound monitoring.

In a retrospective study of 87 patients with cancer and UEDVT, both CVC-related and non-CVC-related, complication rates were higher in patients with internal jugular vein involvement compared with other sites (68.2% vs 52.2%), and in patients without CVC-related thrombosis compared with those with a CVC in place (55% vs 27.7%).¹⁷ These findings are consistent with our observations, particularly regarding the duration of anticoagulation irrespective of the type of risk factor and the occurrence of VTE recurrence after discontinuation of VKAs therapy in patients with transient risk factors.

Recurrence rates of up to 18% have been reported among patients with UEDVT and cancer after a median follow-up of 3.5 years,¹⁸ and 13% after a median follow-up of 5 years in another study.¹⁹ The recurrence rate observed in our cohort (17.2%) is consistent with these data, considering a median follow-up of 3.1 years and a cancer prevalence of 37.9%.¹⁹

The higher mortality rate due to causes unrelated to thrombotic events or bleeding complications observed in patients receiving parenteral anticoagulants may reflect poorer baseline clinical conditions in this subgroup (with 50% affected by cancer). However, the small sample size precludes definitive conclusions.

The main limitations of our study include the small sample size, as well as its retrospective and single-center design. Several questions remain unresolved. For example, the role of follow-up color Doppler ultrasound in assessing recanalization after 3-6 months of therapy and its potential impact on decisions regarding anticoagulant discontinuation remains unclear. Furthermore, comparative studies among different DOACs would be valuable to identify the most effective and safest agents in this setting.

Table 2. Follow-up and outcomes of patients with jugular vein thrombosis

	Total	DOACs	VKAs	Parenteral	P value
Thrombosis resolution*	7 (24.1)	3 (30.0)	3 (17.6)	1 (50.0)	0.493
Total events	13 (41.4)	1 (10.0)	10 (52.9)	2 (100.0)	0.020
Bleedings	5 (17.2)	1 (10.0)	4 (23.5)	0	0.534
Death	3 (10.3)	0	1 (5.9)	2 (100.0)	0.003
Thrombotic events	5 (17.2)	0	5 (29.4)	0	0.119
Thrombotic events after AC suspension	3 (10.3)	0	3 (17.6)	0	0.307

DOACs, direct oral anticoagulants; VKAs, vitamin K antagonists; AC, anticoagulant.

*70% of patients have this data.

Conclusions

Although definitive conclusions cannot be drawn from this small cohort, our findings suggest that patients with IJVT may benefit from anticoagulant therapy, as indicated by the low rate of treatment-related complications and bleeding events. Among the available treatment options, DOACs appear to be a safe and effective alternative to VKAs and parenteral anticoagulants.

Larger prospective studies are needed to confirm these findings and to better identify patients at higher risk of complications, thereby optimizing treatment strategies.

References

1. Drakos P, Ford BC, Labropoulos N. A systematic review on internal jugular vein thrombosis and pulmonary embolism. *J Vasc Surg Venous Lymphat Disord* 2020;8:662-6.
2. Gbaguidi X, Janvresse A, Benichou J, et al. Internal jugular vein thrombosis: outcome and risk factors. *QJM* 2011;104:209-19.
3. Lee WS, Jean SS, Chen FL, et al. Lemierre's syndrome: a forgotten and re-emerging infection. *J Microbiol Immunol Infect* 2020;53:513-17.
4. Kim AS, Khorana AA, McCrae KR. Mechanisms and biomarkers of cancer-associated thrombosis. *Transl Res* 2020;225:33-53.
5. Pastori D, Cormaci VM, Marucci S, et al. A comprehensive review of risk factors for venous thromboembolism: from epidemiology to pathophysiology. *Int J Mol Sci* 2023;24.
6. Carrington BM, Adams JE. Jugular vein thrombosis associated with distant malignancy. *Postgrad Med J* 1988;64:455-8.
7. Flinterman LE, Van Der Meer FJ, Rosendaal FR, et al. Current perspective of venous thrombosis in the upper extremity. *J Thromb Haemost* 2008;6:1262-6.
8. Hingorani A, Ascher E, Lorenson E, et al. Upper extremity deep venous thrombosis and its impact on morbidity and mortality rates in a hospital-based population. *J Vasc Surg* 1997;26:853-60.
9. Prandoni P, Bernardi E, Marchiori A, et al. The long term clinical course of acute deep vein thrombosis of the arm: prospective cohort study. *BMJ* 2004;329:484-5.
10. Tait C, Baglin T, Watson H, et al. Guidelines on the investigation and management of venous thrombosis at unusual sites. *Br J Haematol* 2012;159:28-38.
11. Muñoz FJ, Mismetti P, Poggio R, et al. Clinical outcome of patients with upper-extremity deep vein thrombosis: results from the RIETE Registry. *Chest* 2008;133:143-8.
12. Cote LP, Greenberg S, Caprini JA, et al. Comparisons between upper and lower extremity deep vein thrombosis: a review of the RIETE Registry. *Clin Appl Thromb Hemost* 2017;23:748-54.
13. Kearon C, Akl EA, Comerota AJ, et al. Antithrombotic therapy for VTE disease: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. *Chest* 2012;141:e419S-e496S.
14. Mimier MK, Janczak DT, McBane RD, et al. Thrombosis of atypical location: how to treat patients in the era of direct oral anticoagulants? *Pol Arch Intern Med* 2018;128:604-8.
15. 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.
16. Sheikh MA, Topoulos AP, Deitcher SR. Isolated internal jugular vein thrombosis: risk factors and natural history. *Vasc Med* 2002;7:177-9.
17. Mansour A, Saadeh SS, Abdel-Razeq N, et al. Clinical course and complications of catheter and non-catheter-related upper extremity deep vein thrombosis in patients with cancer. *Clin Appl Thromb Hemost* 2018;24:1234-40.
18. Isma N, Svensson PJ, Gottsater A, et al. Upper extremity deep venous thrombosis in the population-based Malmo thrombophilia study (MATS): epidemiology, risk factors, recurrence risk, and mortality. *Thromb Res* 2010;125:e335-8.
19. Bleker SM, van Es N, Kleinjan A, et al. Current management strategies and long-term clinical outcomes of upper extremity venous thrombosis. *J Thromb Haemost* 2016;14:973-81.