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Patients with primary thrombophilia on anticoagulation face increased mortality, thromboembolic events, and neurologic complications after laminectomy: a propensity-matched analysis

Naasik Syed[1]*, Zuhair Zaidi[1], Muaz Wahid[1], Ronak Desai[2]

[1]Medical School, UT Southwestern Medical Center, TX, USA
[2]Department of Orthopedic Surgery, UT Southwestern Medical Center, TX, USA

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***Correspondence:**
Dr. Naasik Syed,
E-mail: naasik.syed@utsouthwestern.edu

ABSTRACT

Background: Inherited thrombophilias are known risk factors for venous thromboembolism (VTE). The impact of thrombophilia on spine surgery outcomes, especially laminectomy, remains poorly defined, particularly in the context of chronic anticoagulation.
Methods: Using the TriNetX Research Network, we conducted a retrospective cohort study of adult patients undergoing laminectomy between 2004–2025. Patients with primary thrombophilia on ≥6 months of anticoagulation prior to surgery were identified and propensity score–matched 1:1 to controls without thrombophilia or anticoagulation. Outcomes assessed at 90 days and 1 year included mortality, VTE, systemic complications, neurologic deficits, and hospitalizations.
Results: A total of 3,812 matched patients were analyzed. At 90 days, thrombophilia patients had significantly higher rates of mortality, deep vein thrombosis (DVT), sepsis, and hospitalizations. At 1 year, rates of mortality remained elevated, as did DVT, hospitalizations, and new or worsening neurologic deficits. Other systemic and surgical complications were not significantly different.
Conclusion: Patients with inherited thrombophilia on anticoagulation face substantially higher risks of morbidity and mortality following laminectomy. These findings underscore the need for careful perioperative anticoagulation strategies and individualized risk assessment in this high-risk surgical population.

Keywords: Thrombophilia, Laminectomy, Spinal fusion, Anticoagulants, Venous thromboembolism, Postoperative complications

INTRODUCTION

Inherited (primary) thrombophilias, including factor V Leiden, prothrombin G20210A, and deficiencies of protein C, protein S, and antithrombin III, are well-recognized risk factors for venous thromboembolism (VTE).[1] Although relatively uncommon, their presence poses important challenges in patients undergoing spine surgery, where the balance between preventing thromboembolic events and avoiding bleeding complications is especially delicate. Procedures such as lumbar fusion are associated with higher VTE risk than decompression alone, yet the use of anticoagulation raises concerns for spinal epidural hematoma and neurologic injury.[2-5]

In orthopedics, carriers of inherited thrombophilia have consistently demonstrated increased postoperative VTE risk, including deep vein thrombosis (DVT) and pulmonary embolism (PE), particularly after major procedures like hip or knee arthroplasty.[6] Within spine surgery, studies have largely focused on general risk factors such as advanced age, comorbidities, and surgical

complexity, with limited direct investigation of thrombophilia. Current evidence suggests chemoprophylaxis can reduce thrombotic events without substantially increasing bleeding risk, but guidelines remain cautious and nonspecific for patients with inherited hypercoagulability.[5-7]

In this context, our study evaluates systemic outcomes in patients with inherited thrombophilia on anticoagulation undergoing lumbar spinal fusion. By leveraging a large, multicenter national database, we capture these relatively rare disorders at scale and provide new insights into the risks faced by this high-stakes surgical population.

METHODS

This retrospective cohort study was conducted using the TriNetX Research Network, a federated database that aggregates de-identified electronic health records from over 70 healthcare organizations across the United States. Adult patients, defined as those aged 18 years and older, who underwent laminectomy procedures between 01 January 2004, and 13 September 2025, were identified using current procedural terminology (CPT) codes for lumbar, thoracic, and cervical decompression. Specifically, the following CPT codes were employed to capture laminectomy procedures: 63005 (laminectomy, partial, for decompression of spinal cord, single vertebral segment, cervical), 63012 (laminectomy, cervical, two or more vertebral segments), 63017 (laminectomy, thoracic), 63020 (laminotomy, with decompression of nerve root, single interspace, cervical), and 63030 (laminotomy, lumbar, single interspace). Additional codes, including 63035 (each additional lumbar interspace) and 63047 (laminectomy, lumbar, single vertebral segment), were also included to ensure comprehensive capture of the laminectomy cohort.

Patients were divided into two groups on the basis of thrombophilia status. The exposure cohort consisted of patients with primary thrombophilia as identified by International Classification of Diseases, Tenth Revision (ICD-10) codes D68.5 (primary thrombophilia), D68.6 (other thrombophilia), D68.8 (other specified coagulation defects), and D68.9 (coagulation defect, unspecified). Only patients with documentation of anticoagulation use for at least six consecutive months prior to the index laminectomy were included in this cohort, ensuring that the study population represented individuals with clinically significant hypercoagulable states requiring long-term pharmacologic treatment. Anticoagulants were identified using medication records within TriNetX corresponding to warfarin, low-molecular-weight heparins, direct oral anticoagulants (apixaban, rivaroxaban, dabigatran, edoxaban), and fondaparinux. The comparator cohort included patients undergoing laminectomy without any diagnosis codes for thrombophilia and without long-term anticoagulation therapy during the same study period.

Exclusion criteria for both cohorts included age under 18 years, prior spine surgery within one year preceding the index procedure, known secondary or acquired hypercoagulable states (including malignancy-associated thrombosis or antiphospholipid syndrome when identifiable), missing demographic data, and lack of follow-up beyond the index hospitalization. Among 1,980 patients with primary thrombophilia initially identified as undergoing elective laminectomy, 1,754 met inclusion criteria after exclusions and confirmation of anticoagulation use. A total of 155,467 patients without thrombophilia undergoing elective laminectomy were identified as potential controls.

Given the retrospective nature of the TriNetX database, no a priori sample size calculation was performed. Instead, a consecutive sampling approach was used, whereby all eligible patients meeting predefined inclusion and exclusion criteria during the study period were included. This approach is standard for large database studies and maximizes statistical power while minimizing selection bias.

To account for baseline differences between groups, we performed 1:1 propensity score matching using logistic regression. Matching variables included demographic characteristics such as age, sex, race, and ethnicity; comorbid conditions including diabetes mellitus (ICD-10 E08–E13), hypertension (I10), ischemic and non-ischemic cardiovascular disease (I20–I25, I50), chronic kidney disease (N18), chronic liver disease (K70–K77), and obesity (E66); and relevant clinical factors, including body mass index categories (<18.5, 18.5–24.9, 25–29.9, and ≥30 kg/m²) and hemoglobin A1c levels (<5.7%, 5.7–6.4%, 6.5–10%, and >10%). Medication exposure was also incorporated into the matching algorithm and included opioid analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), gabapentinoids, and antidepressants. This matching strategy was designed to minimize confounding and create balanced cohorts for comparative analysis. Following propensity score matching, the final matched cohort consisted of 1,563 patients per group, as seen in Figure 1.

The primary outcomes of interest were postoperative complications occurring within 90 days and within 1 year of surgery. Ninety-day outcomes included all-cause mortality, myocardial infarction (ICD-10 I21–I22), pulmonary embolism (I26), deep vein thrombosis (I82), phlebitis and thrombophlebitis (I80), arterial embolism and thrombosis (I74), stroke (I63–I64), sepsis (A41), and all-cause hospitalization as recorded in TriNetX encounter data. One-year outcomes extended this assessment to include systemic and surgical complications.

In addition to mortality, myocardial infarction, pulmonary embolism, deep vein thrombosis, phlebitis/ thrombophlebitis, arterial embolism/thrombosis, stroke, sepsis, and hospitalizations, we also evaluated new or worsening neurologic deficits, acute kidney injury (ICD-

10 N17), hardware infection (T84.5), wound disruption (T81.3), pseudoarthrosis (M96.0), hardware failure (T84.0–T84.4), vertebral fracture (M80–M84), delirium (F05), and dementia (F01–F03). These comprehensive outcomes framework allowed for an evaluation of both systemic thromboembolic risk and neurologic and surgical sequelae in this high-risk patient population. All analyses were performed using the TriNetX analytics platform. Risk ratios (RRs) with 95% confidence intervals (CIs) were automatically calculated for each outcome. Comparisons between groups were conducted using Chi-squared testing for categorical variables and independent Student's t-tests for continuous variables. The success of propensity score matching was verified by ensuring standardized mean differences of less than 0.1 across matched variables. Statistical significance was defined as a two-sided $p<0.05$.

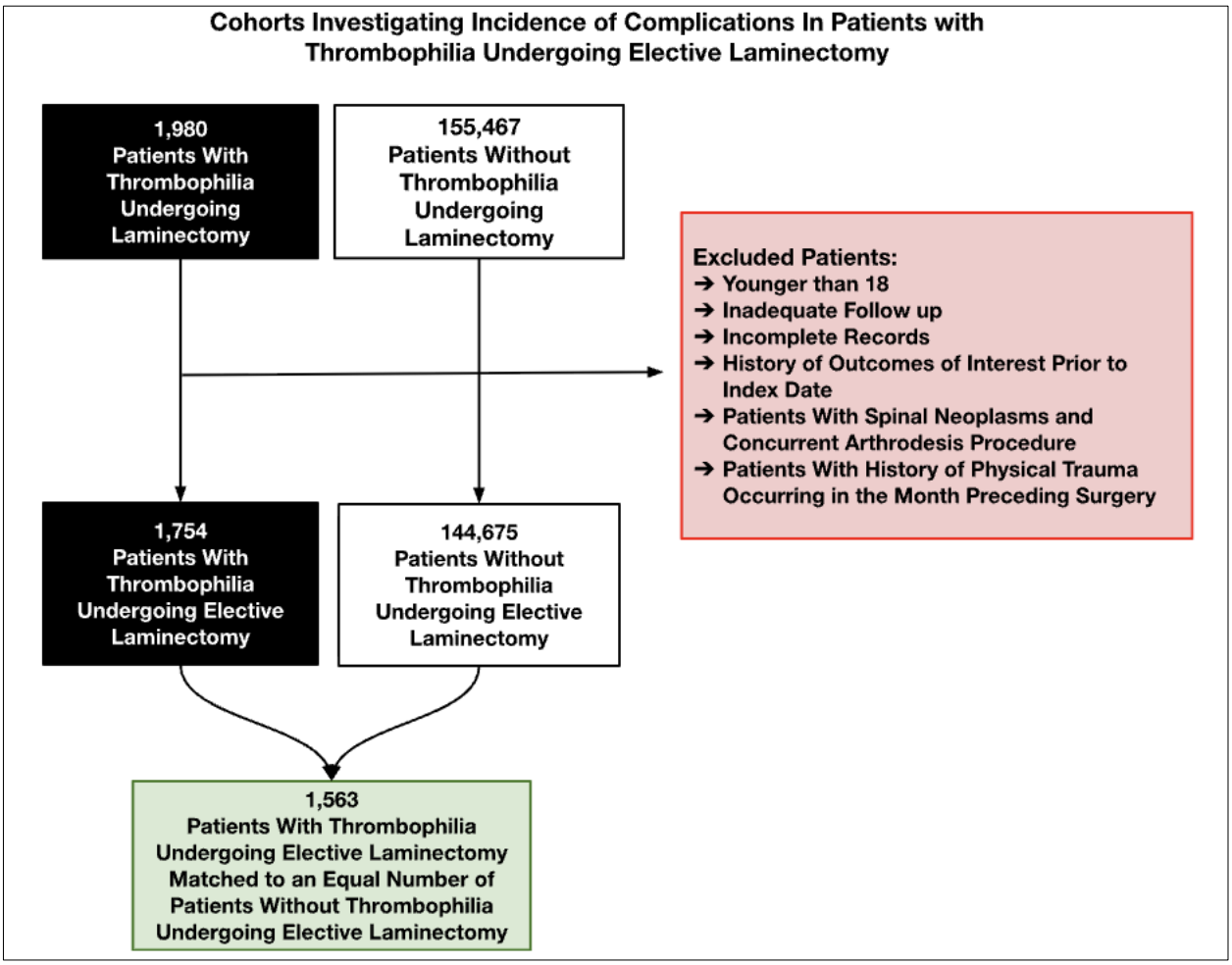

Figure 1: Flow diagram depicting cohort construction.

RESULTS

Prior to propensity score matching, the Thrombophilia cohort was significantly older than the control group, presenting with a mean age of 55.3±14.3 years compared to 53.7±15.4 years ($p<0.0001$), alongside notable disparities in racial distribution and comorbidity prevalence. Following the matching process, these imbalances were effectively neutralized, resulting in comparable mean ages of 55.2±14.3 years for the Thrombophilia group and 55.5±14.7 years for the control group ($p=0.7$). This equilibration extended to all other demographic and clinical characteristics, including sex, race, and key diagnoses like hypertension and diabetes, which showed no statistically significant differences between the cohorts after matching (Table 1).

At 90 days (Table 2), patients with thrombophilia on anticoagulation demonstrated significantly higher risks of adverse outcomes compared with non-thrombophilia patients. Mortality was increased more than fourfold (3.5% versus 0.8%; RR 4.36, 95% CI 2.45–7.76; $p<0.001$), as was deep vein thrombosis (2.4% versus 0.6%; RR 4.08, 95% CI 2.00–8.34; $p<0.001$) and sepsis (2.0% versus 0.6%; RR 3.28, 95% CI 1.61–6.67; $p<0.001$). Hospitalization within 90 days was also significantly more common (28.7% versus 18.7%; RR 1.53, 95% CI 1.36–1.73; $p<0.001$). In contrast, risks of myocardial infarction, pulmonary embolism, phlebitis, arterial embolism, and stroke were not significantly different between groups (all $p>0.05$).

At 1 year (Table 3), patients with thrombophilia on anticoagulation experienced significantly worse outcomes compared with non-thrombophilia patients. Mortality was more than threefold higher (5.9% versus 1.6%; RR 3.60, 95% CI 1.80–7.19; p<0.001), deep vein thrombosis risk was doubled (3.8% versus 1.7%; RR 2.25, 95% CI 1.04–4.86; p = 0.03), and hospitalizations were more frequent (29.9% versus 22.4%; RR 1.33, 95% CI 1.10–1.61; p=0.003). Neurologic complications were also elevated, with new or worsening neurologic deficits occurring in 13.8% of thrombophilia patients versus 8.2% of controls (RR 1.68, 95% CI 1.05–2.67; p=0.03). Other systemic complications, including myocardial infarction, pulmonary embolism, sepsis, phlebitis, arterial thrombosis, and stroke, as well as surgical complications such as acute kidney injury, hardware failure or infection, wound disruption, pseudoarthrosis, vertebral fracture, delirium, and dementia, showed no significant differences between groups (p>0.05).

Table 1: Baseline patient characteristics before and after propensity score matching.

Characteristics	Before matching			After matching		
	Thrombophilia cohort	Controls	P value	Thrombophilia cohort	Controls	P value
Demographics						
Age at index (mean±SD)	55.3±14.3	53.7±15.4	<0.0001	55.2±14.3	55.5±14.7	0.7
White (%)	69.46	83.66	<0.0001	86.94	85.89	0.79
Male (%)	53.53	59.82	<0.0001	53.10	51.24	0.29
Female (%)	46.46	40.11	<0.0001	46.89	48.75	0.29
Black or African-American (%)	7.18	7.58	0.53	7.16	7.48	0.73
Hispanic or Latino (%)	4.79	5.13	0.54	4.79	4.67	0.75
Asian (%)	0.96	2.53	<0.0001	0.95	1.21	0.49
Diagnoses						
Essential hypertension (I10) (%)	71.16	43.59	<0.0001	71.14	72.23	0.5
Hyperlipidemia (E78.5) (%)	57.31	30.05	<0.0001	57.26	56.04	0.48
Anxiety disorders (F40-F48) (%)	43.29	18.06	<0.0001	43.25	42.28	0.054
Overweight (E66) (%)	41.59	18.51	<0.0001	41.45	40.17	0.47
Body mass index (Z68) (%)	34.82	16.93	<0.0001	34.74	33.46	0.45
Chronic pain (R52) (%)	31.54	10.49	<0.0001	31.60	30.71	0.62
Type 2 diabetes (E11) (%)	28.45	17.70	<0.0001	29.49	30.90	0.39
Other hypothyroidism (E03) (%)	23.36	10.23	<0.0001	22.13	21.75	0.8
Chronic kidney disease (N18) (%)	21.34	7.20	<0.0001	21.30	21.62	0.83
Nicotine dependence (F17) (%)	20.69	11.83	<0.0001	20.85	21.81	0.51
Diseases of musculoskeletal (M00-M99) (%)	18.72	6.44	<0.0001	18.61	18.74	0.93
Dorsalgia (M54) (%)	12.23	2.51	<0.0001	11.64	11.00	0.61
Major depressive disorder (F32) (%)	10.16	3.57	<0.0001	10.17	9.35	0.35
Alcohol related disorders (F10) (%)	6.26	3.09	<0.0001	6.27	6.78	0.56
Type 1 diabetes (E10) (%)	4.92	1.87	<0.0001	4.92	3.45	0.054
Polyosteoarthritis (M15) (%)	0.95	0.40	0.0007	0.95	0.76	0.56
Labs						
BMI (mean±SD)	31.2±6.91	28.2±6.26	<0.0001	31.2±6.91	31.2±6.27	0.96
Hemoglobin A1c (mean±SD)	6.07±1.19	6.14±1.15	0.04	6.07±1.19	6.06±1.09	0.99
Medications						
Opioids (CN101) (%)	94.31	57.47	<0.0001	94.30	95.13	0.3
Anticoagulants (BL110) (%)	81.46	26.50	<0.0001	81.38	81.18	0.89
Gabapentinoids (N02BF) (%)	66.83	18.59	<0.0001	66.79	65.06	0.31
Skeletal muscle relaxants (M03B) (%)	64.99	20.10	<0.0001	64.94	63.94	0.52
Antidepressants (N06A) (%)	53.29	27.22	<0.0001	53.22	51.95	0.47
Antipsychotics (N05A) (%)	39.49	11.17	<0.0001	39.53	37.03	0.11
Non-steroidal anti-inflammatories (M01A) (%)	32.35	10.33	<0.0001	32.18	31.03	0.47

Table 2: 90-day outcomes.

Outcome	Thrombophilia risk (%)	Non-thrombophilia risk (%)	Risk difference (%)	Risk ratio (95% CI)	P value
Death	3.5	0.8	2.7	4.36 (2.45–7.76)	<0.001
Myocardial infarction	0.64	0.62	0.02	1.04 (0.43–2.48)	0.94
Pulmonary embolism	1.05	0.59	0.46	1.77 (0.78–4.03)	0.17
Deep vein thrombosis	2.44	0.6	1.84	4.08 (2.00–8.34)	<0.001
Phlebitis/thrombophlebitis	0.64	0.59	0.06	1.10 (0.46–2.64)	0.83
Hospitalization	28.72	18.73	9.99	1.53 (1.36–1.73)	<0.001
Arterial embolism/thrombosis	0.61	0.58	0.03	1.05 (0.44–2.51)	0.91
Stroke	0.69	0.67	0.02	1.04 (0.43–2.48)	0.94
Sepsis	1.98	0.61	1.38	3.28 (1.61–6.67)	<0.001

Table 3: 1-year outcomes.

Outcome	Thrombophilia risk (%)	Non-thrombophilia risk (%)	Risk difference (%)	Risk ratio (95% CI)	P value
Death	5.85	1.63	4.23	3.60 (1.80–7.19)	<0.001
Myocardial infarction	1.85	1.77	0.08	1.05 (0.44–2.50)	0.92
Pulmonary embolism	2.19	1.69	0.5	1.30 (0.54–3.09)	0.55
Deep vein thrombosis	3.8	1.69	2.11	2.25 (1.04–4.86)	0.03
Phlebitis/thrombophlebitis	1.75	1.65	0.11	1.07 (0.45–2.54)	0.89
Hospitalization	29.92	22.44	7.48	1.33 (1.10–1.61)	0.003
Arterial embolism/thrombosis	1.72	1.64	0.09	1.05 (0.42–2.40)	0.91
Stroke	1.88	1.83	0.05	1.03 (0.42–2.49)	0.95
Sepsis	1.81	2.39	-0.58	0.76 (0.34–1.69)	0.50
New/worsening neurologic deficit	13.78	8.22	5.56	1.68 (1.05–2.67)	0.03
Acute kidney injury	4.18	3.45	0.73	1.21 (0.79–1.88)	0.38
Hardware infection	2.93	2.65	0.28	1.11 (0.69–1.77)	0.68
Wound disruption	2.0	2.12	-0.12	0.94 (0.54–1.63)	0.83
Pseudoarthrosis	1.0	0.92	0.09	1.09 (0.48–2.47)	0.83
Hardware failure	0.81	0.81	0.0	1.00 (0.42–2.40)	0.99
Vertebral fracture	0.93	0.83	0.1	1.12 (0.48–2.62)	0.80
Delirium	1.51	0.83	0.68	1.81 (0.84–3.91)	0.12
Dementia	0.82	0.81	0.01	1.01 (0.42–2.42)	0.98

DISCUSSION

This study aims to address the scarcity of data on the impact of thrombophilia on adverse outcomes of laminectomy. Our results demonstrate that patients with thrombophilia on anticoagulation therapy have greatly increased morbidity and mortality compared with matched non-thrombophilia patients. At 90 days, thrombophilia patients had more than four-fold increased risk of death and DVT, more than three-fold increased risk of sepsis, and 1.5-fold increased risk of hospitalization relative to controls. At 1 year, thrombophilia patients had more than three-fold increased risk of death and two-fold risk of DVT in addition to significantly increased risk of hospitalization and new or worsening neurologic deficits relative to controls. These differences in risk indicate that thrombophilia patients should be considered as a high-risk group for complications following laminectomy.

Inherited thrombophilias, such as factor V Leiden, prothrombin G20210A mutation, and protein C or S deficiency, create a hypercoagulable state through overactivity of procoagulant or underactivity of anticoagulant factors.[1] Due to the risk of VTE, including DVT and PE, thrombophilia patients are routinely placed on short-term or long-term anticoagulation therapy after individualized risk assessment.8 Long-term anticoagulation therapies, including warfarin and direct oral anticoagulants (DOACs), are associated with an increased rate of bleeding events in inherited thrombophilia.[9] In these patients, the balance between hypercoagulability and anticoagulation may contribute to complications that arise after surgery. Patients with thrombophilia are hypercoagulable at baseline. In the post-procedure environment, factors such as immobility, leading to stasis of flow, and vessel wall damage from surgery further increase risk for VTE.[10] There is a well-recognized risk of VTE following orthopedic surgeries.[11] Major lower extremity procedures, such as total hip

arthroplasty and total knee arthroplasty, carry the highest rate of symptomatic VTE of around 4%.[6] Spine procedures, such as laminectomy, have lower VTE rates ranging from 0.35 to 2.39%.[12,13] It has been thoroughly documented that thrombophilia patients have an additional risk of VTE following orthopedic procedures.[14-16] Our finding that thrombophilia increases both the short-term and long-term risk of DVT agrees with the existing literature. Although we did not find a significant increase in risk of PE, higher rates of PE within the thrombophilia cohort compared to controls may imply an existing association between thrombophilia and PE after laminectomy. The lower rate of PE after spine surgery relative to other orthopedic procedures may require more statistical power to elucidate a significant relationship.[17] We found that thrombophilia patients were at increased short-term risk of sepsis following laminectomy. While hypercoagulable states are generally not directly associated with increased risk of infection, antithrombin III deficiency has previously been associated with a higher risk of sepsis in hospitalized patients.18 On the contrary, bleeding disorders have been described on numerous occasions to be associated with an increased risk of infection and sepsis.[19-21] Due to the interplay between pathologic hypercoagulability and chronic anticoagulation therapy, thrombophilia patients may be at increased risk of infection and sepsis via multiple mechanisms. The increased risk of VTE, such as DVT, may increase the risk of sepsis acutely, via phlegmasia cerulea dolens, a near-total occlusion of major deep veins and most of the microvascular collateral veins of the extremity which causes severe venous congestion, or chronically, via venous insufficiency and ulceration leading to infection.[22,23] The patients in our thrombophilia cohort received anticoagulation therapy, which has previously been associated with sepsis in total hip arthroplasty and total knee arthroplasty.[24,25] Furthermore, both hypercoagulable states and coagulopathies have been demonstrated to have an increased risk for infection after lumbar spinal surgery.[26] Although neurologic deficits after spinal surgery are most commonly due to fluid collection, such as epidural hematoma, they may also arise due to VTE and rarer events like dural sinus thrombosis in thrombophilia patients.[3-5,7,27,28] Hematomas are particularly devastating complications of spinal surgery, as they can cause spinal cord or nerve root compression leading to pain, weakness, incontinence, and paralysis.[4] Anticoagulation has been demonstrated to increase the risk of symptomatic hematoma in elective spinal surgery, and bleeding disorders are an independent predictor of neurologic deficit in cervical laminectomy.[21] Complications related to thrombosis arise when thromboemboli impair perfusion of the central nervous system or peripheral nerves, leading to ischemic damage. The increased chronic risk of neurologic deficits observed in our cohort may reflect complications arising from a combination of both pro-thrombotic and anti-thrombotic etiologies.

Patients with greater severity of complications after surgery are at increased risk for poor outcomes, including rehospitalization and mortality. Each of the adverse outcomes discussed above has been demonstrated to be a predictor of rehospitalization in postsurgical patients, with up to 70% of VTEs being associated with rehospitalization.[29,30] Indeed, while each of these events in isolation may be sufficient to warrant readmission, it is possible for VTE, sepsis, and neurologic deficits to develop in the same patient. Anticoagulation therapy after surgery increases risk for a spinal epidural hematoma, which can progress to cause spinal cord compression or cauda equina syndrome. Hematoma also predisposes the patient to infection and sepsis, which in turn can initiate inflammatory cascades resulting in disseminated intravascular coagulation and VTE.[31] This pathway of severe complications makes it ultimately unsurprising that the thrombophilia cohort had significantly greater short and long-term risks of rehospitalization and death following laminectomy. Careful optimization of postoperative anticoagulation therapy is likely to mitigate the incidence and severity of postoperative complications in thrombophilia patients after laminectomy. The appropriate therapy regimen must be selected to minimize the complications arising from both bleeding and VTE. Maintaining this balance is challenging, and there is currently a lack of consensus on how to anticoagulate patients after spinal surgery. Pharmacological anticoagulation has been previously linked to increased rates of symptomatic hematoma requiring reoperation without decreasing rate of VTE in the general population.[4] In a study comparing DOACs with heparin/vitamin K antagonists in patients with inherited thrombophilia, it was found both DOACs and heparin/vitamin K antagonists were effective in treating VTE but also associated with bleeding.[9] Another study of the general population demonstrated a 0% bleed rate in patients with low-molecular-weight heparin prophylaxis initiated 24-36 hours after cervical and lumbar laminectomy, but a 3.8% rate of acute VTE, which is greater than the average VTE rate after spinal surgery.[7] Ultimately, we recommend that patients with thrombophilia be carefully evaluated for risk of VTE and bleeding based on guidelines, such as from the American College of Chest Physicians, and on a case-by-case basis, using factors such as age, sex, body mass index, functional status, and operative time, among others.[32,33]

The limitations of this study are inherent to its retrospective design and usage of the TriNetX database. Because ICD-10 codes are used identify diagnoses and complications, there is potential for misclassification, as these codes may not always capture the true clinical context. Furthermore, follow-ups that occur outside of health systems that participate in the TriNetX network are not included in the aggregate data. Additionally, TriNetX lacks details such as type and dose of anticoagulant, which may impact risks of bleeding. We were also unable to account for differences in thrombophilia subtypes (factor V Leiden and antithrombin III deficiency), which confer different levels of risk, or the subtype of complications like

neurologic deficit, which can exert influence over functional outcomes.[18] Social determinants of health are not captured in TriNetX, and may therefore represent unmeasured confounders. Lastly, the lack of randomization and prospective outcome tracking limits our ability to draw causal inferences. Despite these limitations, our study includes a multi-institutional dataset of a large, diverse patient population, which increases the external validity of our findings. Propensity score matching minimized confounding by the measured baseline characteristics, which increases the robustness of comparisons between thrombophilia patients on anticoagulation and matched controls. Furthermore, measuring both short-term and long-term outcomes in our analysis highlights not only the immediate vulnerability for complications, but the sustained risk of neurologic decline. Future studies should include prospective designs accounting for socioeconomic and clinical factors to allow for nuanced investigations incorporating anticoagulation protocols, thrombophilia subtypes, and functional outcomes.

CONCLUSION

In conclusion, patients with primary thrombophilia on anticoagulation therapy have significantly increased risk of DVT, sepsis, neurologic deficits, rehospitalization, and death after laminectomy relative to matched controls without thrombophilia. These complications may be mitigated by an optimized postoperative anticoagulation, and therefore should motivate individualized assessment of thrombophilia patients for VTE and bleeding risk to minimize morbidity and mortality in this high-risk population.

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