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Deep vein thrombosis and pulmonary embolism in neurosurgical patients

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Abstract

Objective: Venous thromboembolism (VTE), comprised of deep vein thrombosis (DVT) and pulmonary embolism (PE), poses a major risk of illness and death in neurosurgical patients. This study aims to determine the incidence of VTE in neurosurgical patients, investigate the risk factors for their development. *Methods:* All admission records of neurosurgical patients between 1st January 2023 and 31st December 2023 to our centre were reviewed retrospectively to identify cases of VTE. Patient demographics, surgical details, postoperative immobilization, and risk factors were recorded and analysed using logistic regression analyses. *Results:* A total of 1,000 neurosurgical patients were included, with 27 (2.7%) developing VTE, comprising 5 (0.5%) DVT and 22 (2.2%) PE. Univariate analysis revealed hypotension (OR: 84.43, $p < 0.001$), tachycardia (OR: 68.43, $p < 0.001$), SpO2 <90% (OR: 19.38, $p < 0.001$), respiratory rate >30 per minute (OR: 9.65, $p = 0.005$), surgery during admission (OR: 3.77, $p = 0.002$), prolonged hospital stay (OR: 1.04, $p < 0.001$), and low Glasgow Coma Scale (GCS) upon admission (OR: 0.84, $p < 0.001$) as significant predictors of VTE. Multivariate analysis determined three independent risk factors: tachycardia (adjusted OR: 28.61, $p < 0.001$), prolonged hospital stay (adjusted OR: 1.03, $p < 0.001$), and paralysis or lower extremity immobilization (adjusted OR: 5.53, $p = 0.007$).
Conclusion: Paralysis or lower extremity immobilization, prolonged hospital stays, and tachycardia are independent predictors of VTE in neurosurgical patients. Identifying high risk patients based on risk factors and implementing individualized thromboprophylaxis strategies is crucial to reduce morbidity and mortality.

Keywords: Venous thromboembolism (VTE), deep vein thrombosis (DVT), pulmonary embolism (PE), Neurosurgery, neurocritical care, thromboprophylaxis

INTRODUCTION

Venous thromboembolism (VTE), comprised of two distinct but similar pathologies: deep vein thrombosis (DVT) and pulmonary embolism (PE), can be devastating among neurosurgical patients.[1,2] Classically, Virchow's triad explained the pathogenesis of VTE, including blood stasis, endothelial injury and hypercoagulability. Neurosurgery patients are vulnerable to VTE due to multifactorial risks, in which the main contributor is immobility and bed rest after prolonged operation, which causes venous stasis.[3] Additional risk factors include advanced age, malignancy, prolonged operative duration, neurological deficits, and corticosteroid use.[3-6]

Reported incidence varies widely depending on diagnostic approach. Clinical evaluation suggests VTE rates of 4.0% without prophylaxis, 2.0% with pharmacological prophylaxis, 12.9% with mechanical prophylaxis, and 10.6% with combined methods.[7] A meta-analysis of neurosurgical patients using the American College of Surgeons' National Surgical Quality Improvement Program (NSQIP) database determined a VTE rate of 1.7%, while the prevalence rate was from 1.3% - 1.8%.[1] The Registro Informatizado de Enfermedad Tromboembólica registry reported a 0.96% incidence of VTE within 60 days post-neurosurgery.[8] Routine serial doppler ultrasonography screening leads to

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higher incidence of DVT including silent or asymptomatic types, the clinical significance of managing silent DVT still remains uncertain.[9]

Neurosurgeons must balance the risks of prevention of VTE with the dangers of haemorrhagic complications, particularly intracranial bleeding associated with pharmacological prophylaxis.[4] Pharmacologic thromboprophylaxis, including low-molecular-weight heparin (LMWH) and unfractionated heparin (UFH), has demonstrated efficacy in reducing VTE incidence, but concerns about bleeding risk including intracranial hemorrhage limit its use, particularly in the early postoperative period.[4,10] In the other hand, mechanical prophylaxis methods, such as intermittent pneumatic compression (IPC) and Thromboembolism-Deterrent (TED) stockings, offer safer but less effective alternatives as compared to chemoprophylaxis.[5,11] Thus, neurosurgeons must balance the risks of prevention of VTE with the dangers of intracranial bleeding.

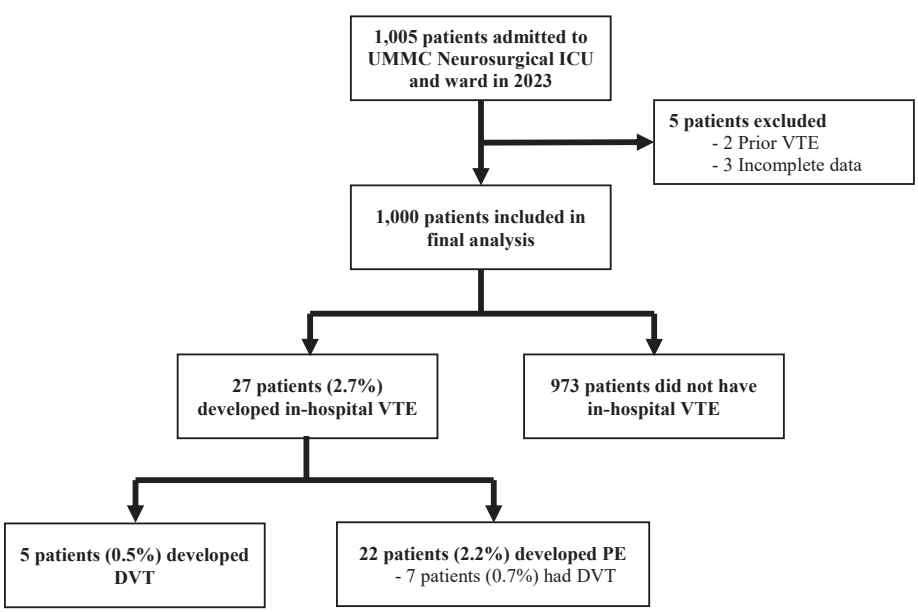
Given that PE accounts for up to 9–50% mortality in neurosurgical patients[12], this study aims to determine the incidence of VTE and identifying significant risk factors in neurosurgical patients in University of Malaya Medical Centre (UMMC), a tertiary teaching hospital, located in Kuala Lumpur, the capital of Malaysia.

METHODS

This was a retrospective cross-sectional study conducted at a single academic centre. Ethical approval was obtained from UMMC Ethics Committee (20231218-13145). The retrospective analysis utilized data from the electronic medical records (EMR) of neurosurgical patients admitted to the University of Malaya Medical Centre (UMMC) from 1st January 2023 to 31st December 2023.

Inclusion criteria were all neurosurgical patients aged 18 years or older who were admitted to UMMC NeuroICU and neurosurgical ward during the study period. Patients with a documented history of VTE prior admission or incomplete medical records were excluded. A total of 1,005 patients were screened, of whom 5 were excluded (2 with prior VTE and 3 with incomplete records). The final study cohort consisted of 1,000 patients (Figure 1).

Clinical and demographic data included patient age, sex, body mass index (BMI), Glasgow Coma Scale (GCS) upon presentation to Emergency Department, hospital length of stay and comorbidities such as cancer, cardiovascular disease. Surgical details were recorded, including type, duration, and timing of procedures. The clinical parameters including vital signs and neurological status were extracted from EMR. The diagnoses of DVT and PE were confirmed



UMMC = University of Malaya Medical Center; ICU = Intensive Care Unit; VTE = Venous Thromboembolism; DVT=Deep Vein Thrombosis; PE= Pulmonary Embolism

Figure 1: Flow diagram of patient selection for the study.

using Doppler ultrasonography and computed tomography pulmonary angiography (CTPA) respectively. Laboratory data such as D-dimer levels was included. Risk stratification was performed retrospectively using the Wells criteria for DVT/PE and the Pulmonary Embolism Severity Index (PESI) for PE.

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 29.0.1 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize patient demographics and clinical characteristics. Continuous variables were presented as medians with interquartile ranges in view of non-parametric data, while categorical variables were expressed as percentages. Between-group comparisons were analysed using Mann-Whitney U test for non-parametric data, and Chi-squared test for categorical variables or Fischer's exact test. Univariate logistic regression was applied to identify potential predictors of VTE, and significant variables ($p < 0.05$) were entered into a multivariate logistic regression. Odds ratios with 95% confidence intervals were calculated for each risk factor. All tests were two tailed with a significant level of 0.05. Multicollinearity among predictors was assessed by variance inflation factors (VIF); VIF ≥ 5 (tolerance ≤ 0.20) indicated unacceptable collinearity.

RESULTS

We identified 1,005 neurosurgical cases admitted to our centre from 1st January 2023 to 31st December 2023, 2 cases were excluded in view of underlying diagnosis of VTE, 3 cases were excluded due to incomplete data. The final cohort comprised 1,000 patients (Figure 1).

Over the 12-month study duration, 27 of 1,000 neurosurgical admissions developed in-hospital VTE, a cumulative incidence of 2.7%. DVT occurred in 5/1,000 patients (0.5%), and PE in 22/1,000 (2.2%). Among VTE events, 81.5% presented as PE (22/27). Among patients with PE, 7 patients (31.8%) had concomitant ultrasound-confirmed DVT.

Baseline clinical and demographic characteristics by VTE status are shown in Table 1. Compared with non-VTE patients, the VTE group showed statistically significant differences (all $p < 0.05$) in: weight, GCS at admission, presence of paralysis/paresis or recent plaster immobilisation of the lower limb, LOS, surgery during admission, time to ambulation, heart rate (HR) > 100 bpm, systolic blood pressure (SBP) < 100 mmHg, respiratory rate (RR) > 30/min, and oxygen saturation (SpO_2) < 90% (Table 1). Mechanical prophylaxis (IPC/TED stockings) was used in 24/27 (88.9%) VTE patients vs 228/973 (23.4%) non-VTE patients. Pharmacological prophylaxis (Low-Molecular-Weight Heparin or unfractionated heparin) was given to 23/27 (85.2%) VTE patients vs 40/973 (4.1%) non-VTE patients.

The risk stratification tools, including Well's scores (for DVT and PE) and PESI were significantly higher in the VTE group, supporting their utility for risk stratification in neurosurgical patients (Table 1).

In univariate logistic regression (Table 2), the strongest predictors of developing VTE were SBP <100 mmHg (OR: 84.43; 95% CI: 14.72–484.44; $p < 0.001$), HR >100 beats per minute (OR: 68.43; 95% CI: 27.31–171.42; $p < 0.001$) and SpO_2 <90% (OR: 19.38; 95% CI: 3.39–110.77; $p < 0.001$). Other significant predictors of VTE included RR >30 per min (OR: 9.65, 95% CI: 1.95–47.78, p=0.005), surgery during admission (OR: 3.77, 95% CI: 1.63–8.70, p=0.002), longer LOS (OR: 1.04, 95% CI: 1.03–1.05, p<0.001) and lower GCS upon admission (OR: 0.84, 95% CI: 0.78–0.91, p<0.001).

In multivariable logistic regression (Table 2; forest plot in Figure 2), three variables remained independently associated with VTE, including HR >100 bpm (adjusted OR: 28.61, 95% CI: 8.58–95.34; $p < 0.001$), LOS (adjusted OR: 1.03, 95% CI: 1.01–1.04; $p < 0.001$), and paralysis, paresis or recent plaster immobilisation of the lower extremity (adjusted OR: 5.53; 95% CI: 1.59–19.17; $p = 0.007$). Multicollinearity was low (VIF 1.13–1.61; tolerance > 0.60), as shown in Table 2. Global goodness-of-fit was acceptable (Pearson $\chi^2(398) = 373.59$, $p > 0.05$). The logistic regression equation is $\text{Logit}(P) = -6.674 + 0.013\mathbf{X}_2 + 0.028\mathbf{X}_3 - 1.027\mathbf{X}_4 + 0.992\mathbf{X}_6 + 1.709\mathbf{X}_7 + 3.354\mathbf{X}_8 + 0.973\mathbf{X}_9 - 0.686\mathbf{X}_{10} + 0.132\mathbf{X}_{11}$.

DISCUSSION

In this 12-month, single-centre cohort, the clinically detected incidence of VTE was 2.7% (DVT 0.5%; PE 2.2%), similar to NSQIP database with overall VTE rate of 1.7% in neurosurgical patient[8] but comparatively lower to previous studies based on clinical evaluation, with incidence of 4.0% – 12.9%.[7,13,14] Some studies applied systemic screening method with routine surveillance imaging (doppler

Table 1: Clinical and demographic characteristics of patients with and without venous thromboembolism (VTE)

Clinical and demographic characteristics	VTE (n=27)	Non-VTE (n=973)	Total (n=1000)	*P*-value
Age (years), median (IQR)	47.0 (34.0)	56.0 (29.0)	55.5 (29.0)	0.242[b]
Gender, n (%)				
Male	15 (55.6)	611 (62.8)	626.0 (62.6)	0.546
Female	12 (44.4)	362 (37.2)	374 (37.4)	
Weight (kg), median (IQR)	70.0 (18.0)	65.0 (20.0)	65.0 (20.2)	0.023[b]
Height (m), median (IQR)	1.7 (0.1)	1.6 (0.1)	1.6 (0.1)	0.385[b]
BMI (kg/m2), median (IQR)	28.2 (11.6)	24.6 (5.8)	24.6 (5.9)	0.080[b]
Ethnic, n (%)				
Malay	10 (37.0)	323 (33.2)	333 (33.3)	0.668[a]
Chinese	8 (29.6)	392 (40.3)	400 (40.0)	
Indian	8 (29.6)	200 (20.6)	208 (20.8)	
Others	0 (0)	16 (1.6)	16 (1.6)	
Foreigner	1 (4.3)	42 (3.7)	43 (4.3)	
Smoking, n (%)	0 (0)	67 (6.9)	67 (6.7)	0.363[a]
Drinking, n (%)	2 (7.4)	43 (4.4)	45 (4.5)	0.345
History of Heart Failure, n (%)	0 (0)	9 (0.9)	9 (0.9)	1.000
History of Chronic Lung Disease, n (%)	0 (0)	7 (0.7)	7 (0.7)	1.000
Active Cancer, n (%)	5 (18.5)	104 (10.7)	109 (10.9)	0.204
History of Chemotherapy, n (%)	2 (7.4)	62 (6.4)	64 (6.4)	0.689
History of Radiotherapy, n (%)	4 (14.8)	70 (7.2)	74 (7.4)	0.133
Hormone Use, n (%)	0 (0)	3 (0.3)	3 (0.3)	1.000
GCS upon Admission, median (IQR)	10 (8)	15 (1)	15 (1)	<0.001[b]
Paralysis, Paresis or Recent Plaster Immobilization of The Lower Extremity, n (%)	21 (77.8)	172 (17.7)	193 (19.3)	<0.001[a]
Length of hospital stay(days), median (IQR)	44.0 (54.0)	4.0 (6.0)	4.0 (7.0)	<0.001[b]
Surgery during admission, n (%)	19 (70.4)	376 (38.6)	395 (39.5)	0.001
Duration of Operation (mins), median (IQR)	145.0 (198.0)	110.0 (126.0)	115.0 (128.5)	0.416[b]
Duration to ambulation (days), median (IQR)	4.0 (19.3)	0 (1.0)	0 (1.0)	0.023[b]
TED Stocking, n (%)	22 (81.5)	223 (22.9)	245 (24.5)	<0.001
Anticoagulant, n (%)	23 (85.2)	40 (4.1)	63 (6.3)	<0.001
D-dimer (ng/ml), median (IQR)	2521.5 (9391.8)	2691.5 (4956.5)	2650 (4779)	0.220 [b]
Subspecialty for admission, n (%)				
Trauma	7 (25.9)	336 (34.6)	343 (34.3)	0.836[a]
Oncology	6 (22.2)	205 (21.1)	211 (21.1)	
Vascular	5 (18.5)	168 (17.3)	173 (17.3)	
Spine	3 (11.1)	122 (12.6)	125 (12.5)	
Skullbase	3 (11.1)	56 (5.8)	59 (5.9)	
Others	3 (11.1)	85 (8.7)	88 (8.8)	
Types of hospital admission, n (%)				
Emergency	23 (85.2)	672 (69.1)	695 (69.6)	0.090
Elective	4 (14.8)	300 (30.9)	304 (30.4)	
Vital signs, n (%)				
Heart rate >100 beats per min	20 (74.1)	39 (4.0)	59 (5.9)	<0.001[a]
Systolic BP <100mmHg	4 (14.8)	2 (0.2)	6 (0.6)	<0.001
Respiratory Rate >30 per min	2 (7.4)	8 (0.8)	8 (0.8)	0.028
Temperature <36°C/96.8°F	0 (0)	2 (0.2)	2 (0.2)	1.000
Oxygen saturation <90%	2 (7.4)	4 (0.4)	4 (0.4)	0.010
Clinical score and risk assessment, median (IQR)				
Well score for DVT	2.0 (1.0)	1.0 (1.0)	1.0 (1.0)	<0.001[b]
Well score for PE	3.0 (0)	1.5 (1.5)	1.5 (1.5)	<0.001[b]
PESI	116.0 (45.0)	75.0 (47.5)	75.0 (52.0)	<0.001[b]

IQR = Interquartile Range; BMI = Body Mass Index; GCS = Glasgow Coma Scale; BP = Blood Pressure; TED = Thromboembolic Deterrent; PESI = Pulmonary Embolism Severity Index

[a] *P*-values from Chi-squared test

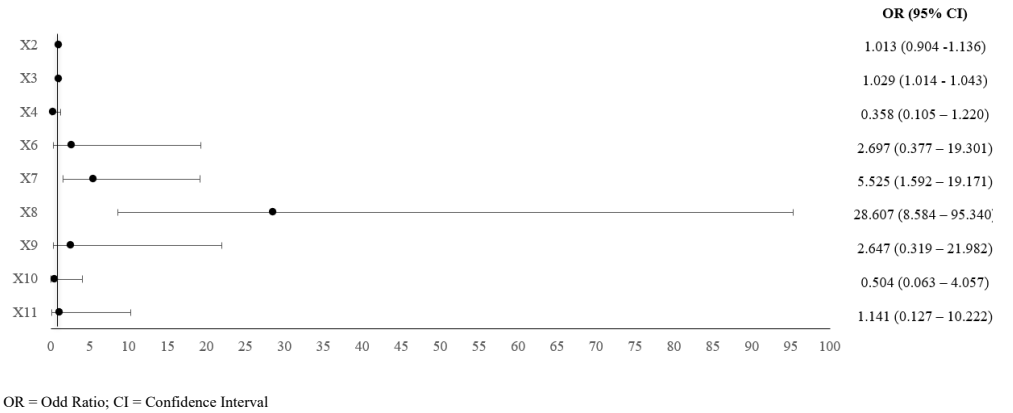
[b] *P*-values from Mann-Whitney U test

Other *P*-values from Fisher's exact test

Table 2: Univariate and multivariate logistic regression analysis of studied risk factors for venous thromboembolism (VTE)

	Risk Factors	Univariate				Multivariate				VIF
		B	P-value	OR	95% CI	B	P-value	OR	95% CI	
X_0	Intercept					-6.674	<0.001	0.001		
X_1	Weight	0.005	0.279	1.005	0.996 - 1.013					
X_2	GCS upon Admission	-0.174	<0.001	0.84	0.779 - 0.906	0.013	0.824	1.013	0.904 - 1.136	1.322
X_3	Length of Hospital Stay	0.037	<0.001	1.037	1.028 - 1.048	0.028	<0.001	1.029	1.014 - 1.043	1.388
X_4	Surgery during Admission	1.327	0.002	3.771	1.634 - 8.701	-1.027	0.101	0.358	0.105 - 1.220	1.133
X_5	Duration to Ambulation	0.005	0.677	1.005	0.982 - 1.028					
X_6	Immobilization >3 days	2.818	<0.001	16.737	3.942 - 71.056	0.992	0.323	2.697	0.377 - 19.301	1.608
X_7	Paralysis, Paresis or Recent Plaster Immobilization of The Lower Extremity	2.791	<0.001	16.299	6.482 - 40.987	1.709	0.007	5.525	1.592 - 19.171	1.445
X_8	Heart rate >100 beats per min	4.226	<0.001	68.425	27.312 - 171.424	3.354	<0.001	28.607	8.584 - 95.340	1.499
X_9	Systolic BP <100mmHg	4.436	<0.001	84.435	14.716 - 484.442	0.973	0.367	2.647	0.319 - 21.982	1.152
X_{10}	Respiratory Rate >30 per min	2.267	0.005	9.65	1.949 - 47.775	-0.686	0.520	0.504	0.063 - 4.057	1.179
X_{11}	Oxygen saturation <90%	2.964	<0.001	19.38	3.391 - 110.767	0.132	0.906	1.141	0.127 - 10.222	1.132

GCS = Glasgow Coma Scale; BP = Blood Pressure; VIF = Variance Inflation Factor



OR = Odd Ratio; CI = Confidence Interval

Figure 2. Forest plot of multivariate logistic regression output with X-axis showing odds ratio with 95% CI

ultrasonography ± screening CTPA) consistently find higher incidence rate (7.3% – 31.1%).[7,15-17] Our incidence of 2.7 % was derived from clinically prompted investigations; imaging was obtained only when signs or symptoms raised suspicion for DVT or PE. Systematic protocols with scheduled ultrasound/CTPA detect more "silent" events (asymptomatic distal DVTs and sub-segmental PE). Hence, the difference is mainly methodological rather than biological. Our data also revealed that PE may occur without demonstrable lower-extremity DVT on doppler ultrasonography, which could be due to upper-extremity/line-associated thrombosis.[5]

Systematic D-dimer surveillance was not part of our institutional protocol; instead, the assay was ordered when VTE was clinically suspected. which introduces ascertainment bias and limits inference about D-dimer as an independent predictor. Future prospective studies should incorporate routine D-dimer measurements at prespecified postoperative time points, coupled with systematic imaging, to diagnose subclinical VTE and to determine the biomarker's predictive value in neurosurgical populations.

In our multivariable model, three independent predictors of VTE emerged: tachycardia >100 bpm, longer hospital stay, and lower-limb paralysis/paresis or recent plaster immobilisation.

In our model, tachycardia >100 bpm was independently associated with VTE. it is a recognised clinical sign of pulmonary embolism (PE) and included in risk stratification tools such as Well's score for PE and PESI. Moreover, tachycardia may be the earliest abnormal vital sign in silent PE among neurosurgical patients.[11,18] Even small embolic fragments traversing the pulmonary circulation can trigger reflex sympathetic discharge, right-ventricular pressure overload, and hypoxaemia, which can lead to persistent sinus tachycardia hours or days prior to haemodynamic collapse.[19] Concurrently, the catecholamine surge amplifies coagulation by up-regulating tissue-factor expression, releasing von Willebrand factor, increasing Factor VIII activity, and promoting platelet activation thus may trigger a hypercoagulable state and enhance the odds of overt thrombosis.[20,21] Therefore, persistent tachycardia in an immobilised neurosurgical inpatient should prompt urgent evaluation with CTPA.

Paralysis, paresis, or immobilization as one of the strong predictors of VTE, is consistent with finding from previous studies.[1,8,16,22-24] With prolonged immobilization, it can lead to stasis and flow turbulence in deep vein[25,26], causing local hypoxia, and further promoting thrombosis.[27] In our multivariable model, immobilisation ≥3 days did not show statistical significance, likely reflecting overlap with other mobility-related covariates (e.g., paralysis, length of stay). Time to ambulation showed a small effect size—approximately 0.5% higher odds of VTE per additional day (OR ≈ 1.005)—but this association was not significant on univariate analysis. These findings suggest that, in our cohort, the effect of gross motor deficit (paralysis/paresis) outweighed duration metrics of immobility (immobilization > 3 days or duration to ambulation), possibly due to limited events, collinearity with length of stay, and residual confounding.

In addition, longer hospitalisation period, also one independent risk factor for VTE is consistent with some published literatures.[3,7,12,16,28] It is

often due to complex neurosurgical procedure which required intensive monitoring and delayed postoperative mobilization, some may be caused by postoperative complications such as hospital acquired infections (HAI), intracranial hemorrhage, pressure sores and others.

In our logistic regression analysis, surgery during admission showed clinical significance in univariate analysis, however it was not an independent predictor in the multivariate model. Some studies revealed moderate risk of VTE in patient underwent a cranial neurosurgical procedure.[4,29] Those who underwent cranial surgery are more likely to develop VTE as compared to spinal surgery.[1] Prolonged immobilization (>3 days) was significantly associated with VTE in the univariate model but was not an independent predictor in the multivariate model. This could be due to diminished significance in the multivariate model due to its strong correlation with other variables, such as paralysis, duration of hospitalization, and significant motor weakness. Other non-independent predictive factors included SBP <100 mmHg, RR >30 bpm and oxygen saturation <90% as these could be a marker of other underlying conditions, such as hemodynamic instability, sepsis, or cardiopulmonary dysfunction.

There are several publications reported malignancy as a risk factor for VTE due to the prothrombotic state induced by tumor-derived tissue factor and cytokines and disruption of the endothelium during surgery.[8,30-32] Nevertheless, presence of active cancer was not statistically significant in our study. Other established predictive risk factors, for example, age[1,8,22-24,28,32], weight[1,8], gender[8], tobacco use[8], chemotherapy[1], longer surgical duration[12] and D-dimers levels[12,16,28], are statistically not significant in our study. There is no significance in D-dimer level between VTE and Non-VTE groups, as D-dimer is a non-specific biomarker for VTE, often providing false-positive results in neurosurgical patients due to cancer, in hospitalized patients in severe infection or inflammatory disease, and during pregnancy.[33]

This study has several limitations. The low event rate (27/1,000) widens confidence intervals and increases the risk of model overfitting, so some effects may be undetected. As our centre does not have a standardised VTE-prophylaxis pathway, prophylaxis was clinician-directed. Diagnosis of VTE depended on clinical evaluation with confirmatory imaging and selectively ordered D-dimer rather than protocolised surveillance, which likely underestimates asymptomatic distal DVT and subsegmental PE. Several exposures (e.g., time to ambulation, immobilization ≥3 days) and risk scores were abstracted retrospectively, creating potential for measurement error. This single-centre, retrospective design introduces potential selection, information, and ascertainment biases due to centre-specific referral and practice patterns, documentation practices, and non-standardised VTE screening. Thus, we propose a future multicentre, prospective study with thromboprophylaxis reporting (mechanical and pharmacological), scheduled D-dimer sampling at predefined time points (e.g., admission, postoperative day 2–3, day 7), and both symptom-triggered and surveillance doppler ultrasonography or CTPA.

Our research findings suggest implementation of VTE prophylaxis protocol in individual case basis with integration of risk stratification tools: Well's score for DVT and PE, PESI and our predictive factors. Identifying high risk patient and early initiation of thromboprophylaxis are crucial to prevent development of VTE. Mechanical prophylaxis with IPC (± TED stockings) should begin on admission with the early mobilisation and used perioperatively.[34,35] Thromboprophylaxis (UFH or LMWH) should commence once haemostatic stability is documented — within 24 to 72 hours postoperatively in patient with high risk of thrombosis and 2 to 4 days in nontraumatic intracerebral haemorrhage.[34] Pharmacologic VTE prophylaxis in traumatic brain injury (TBI) should commence once intracranial hemorrhage stability is confirmed on the repeated CT scan —generally within 24 hours for low-risk nonoperative injuries and within 24–48 hours for moderate/high-risk or post-craniotomy/craniectomy cases if stable.[35] Withholding pharmacologic VTE prophylaxis is recommended when CT demonstrates expansion of intracranial bleeding until stability is confirmed on a subsequent scan in 24–48 hours.[35] LMWH is the preferred VTE prophylaxis in TBI, including patients with ICP monitors as it yields lower VTE rates and postoperative bleeding risk as compared to UFH.[34,35] Nevertheless, risk of intracranial bleeding associated with pharmacological thromboprophylaxis and benefits of VTE prophylaxis is needed to be balanced to prevent further adverse outcome.

In conclusion, the incidence rate of VTE among neurosurgical patients in our centre is 2.7%. This study highlights the independent predictors of developing VTE: paralysis or lower extremity immobilization, prolonged hospital stays and

tachycardia >100 bpm. Thromboprophylaxis such as IPC, CS and anticoagulants, plays an important role in reducing VTE incidence. Further research is required to study the optimal timing of pharmacological prophylaxis to balance the risk of bleeding upon starting anticoagulants. Therefore, clinician should identify high risk patients of developing VTE by addressing the predictive factors and carry out prophylactic strategies to reduce morbidity and mortality associated with VTE.

DISCLOSURE

Conflict of interest: None

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