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Incidence, Risk Factors, and Prevention of Deep Vein Thrombosis in Acute Ischemic Stroke Patients (IRIS-DVT Study): A Systematic Review and Meta-Analysis

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Abstract

Background: Deep vein thrombosis (DVT) is a serious thromboinflammatory complication of acute ischemic stroke (AIS). The true incidence, mechanistic risk factors, and optimal prophylactic strategies remain uncertain, particularly in the era of reperfusion therapy. Methods: This systematic review and meta-analysis (IRIS-DVT) searched PubMed, Embase, Cochrane, Scopus, and Web of Science for studies reporting DVT incidence, risk factors, or prophylaxis in AIS (2004–2025). Random-effects models were used to generate pooled prevalence and effect estimates, and the certainty of evidence was graded using the GRADE framework. Results: Forty-two studies (n = 6,051,729 patients) were included. The pooled prevalence of DVT was 7% (95% CI, 6–9%), approximately seventy-fold higher than in the general population, with wide heterogeneity influenced by screening timing and diagnostic modality. Pathophysiological risk factors included higher stroke severity (NIHSS; SMD 0.41; 95% CI, 0.38–0.43), older age (SMD 0.32; 95% CI, 0.18–0.46), elevated D-dimer (SMD 0.55; 95% CI, 0.38–0.72), female sex (OR 1.33; 95% CI, 1.19–1.50), and malignancy (OR 2.69; 95% CI, 1.56–5.22), supported by moderate-certainty evidence. Respiratory infection and admission hyperglycemia showed weaker, low-certainty associations. Traditional vascular risk factors (hypertension, diabetes, atrial fibrillation, dyslipidemia) were not significantly related to DVT risk. Evidence for prophylaxis with low-molecular-weight heparin, direct oral anticoagulants, or intermittent pneumatic compression was limited and graded very low certainty. Conclusions: DVT complicates approximately one in fourteen AIS cases, reflecting a distinct thromboinflammatory process driven more by acute neurological severity, systemic hypercoagulability, and malignancy than by conventional vascular risk factors. Early systematic screening (≤72 h) and consistent use of mechanical prophylaxis are warranted. Dedicated AIS-specific mechanistic and interventional trials are urgently needed to refine prevention strategies and improve post-stroke outcomes.

Keywords: acute ischemic stroke; deep vein thrombosis; thromboinflammation; pathophysiology; risk factors; prophylaxis; D-dimer; NIHSS

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1. Introduction

Deep vein thrombosis (DVT) is a serious and potentially life-threatening complication of acute ischemic stroke (AIS) [1]. It forms part of the broader spectrum of venous thromboembolism (VTE) [2], in which embolization to the pulmonary circulation may result in pulmonary embolism (PE)—a major contributor to post-stroke morbidity and mortality [3]. While PE is the most overtly fatal manifestation, its origins often lie in unrecognized distal or proximal DVT, highlighting the importance of early detection and prevention within stroke care pathways [4].

Over the past two decades, the advent of intravenous thrombolysis (IVT) and endovascular thrombectomy (EVT) has markedly improved neurological outcomes after AIS. Yet the risk of thromboembolic events has not declined proportionally [5–7]. Stroke-related immobility, systemic inflammation, endothelial dysfunction, and delayed initiation of prophylaxis collectively sustain a pro-thrombotic milieu even in modern, protocol-driven units [8]. Reported incidence rates of DVT in AIS vary widely across studies, ranging from below 1% to over 20% [8–13], reflecting methodological heterogeneity in screening protocols, imaging sensitivity, and regional prophylaxis practices rather than true biological variation [14,15].

Despite its clinical relevance, current knowledge of DVT after AIS remains fragmented. Most available studies are limited by retrospective or single-center designs, small sample sizes, or by extrapolating findings from non-stroke medical populations. As a result, the true burden of DVT in contemporary stroke care—and its complex interaction with reperfusion therapies, systemic inflammatory factors, and prophylactic strategies—remains uncertain. The comparative effectiveness of pharmacological prophylaxis (e.g., low-molecular-weight heparin, direct oral anticoagulants) [16–19] and mechanical approaches (e.g., intermittent pneumatic compression, inferior vena cava filters) [20–24] in AIS patients has not been comprehensively evaluated in a stroke-specific context. These limitations have hindered accurate risk stratification [4] and contributed to the ongoing inconsistency of international guideline recommendations, leaving clinicians without a unified, evidence-based prevention framework [25–27].

The *Investigating the Incidence, Risk Factors, and Prophylactic Strategies for Deep Vein Thrombosis in Acute Ischemic Stroke Patients* (IRIS-DVT) study was designed to address this critical evidence gap. It systematically synthesizes global data to define the incidence, determinants, and preventive strategies for DVT after AIS; examines how modern reperfusion therapies such as IVT and EVT alter thromboembolic risk through changes in mobility, procedural factors, and timing of prophylaxis; and evaluates the comparative efficacy and certainty of both pharmacological and mechanical interventions [5,28]. Beyond quantifying incidence and risk, the IRIS-DVT framework reconceptualizes DVT as a surrogate marker of systemic thromboinflammatory activation predisposing to PE and other VTE-related outcomes [1,29]. The IRIS-DVT study sought to generate evidence-based insights that can guide early risk assessment, inform precision-based prophylactic strategies, harmonize guideline recommendations, and improve long-term outcomes for patients with AIS.

2. Materials and Methods

2.1. Literature Search and Study Selection

We conducted a comprehensive systematic search of PubMed, Embase, Cochrane Library, Scopus, and Web of Science from January 2004 to May 2025. Search strategies combined terms related to "*deep vein thrombosis*" (DVT), "*acute ischemic stroke*" (AIS), "*risk factors*", and "*prophylactic interventions*", with filters for human studies in adults (≥18 years). A detailed search strategy is provided in the Supplementary Information. References of relevant reviews and meta-analyses were also screened to identify additional eligible studies.

Titles and abstracts were independently screened by two reviewers, with full-text reviews performed for potentially eligible studies. Discrepancies were resolved by consensus. A PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow diagram illustrates the selection process (Figure 1). This study was registered in Open Science Framework (OSF; registration ID: buxr8).

2.2. Eligibility Criteria

Studies were included if they: (1) enrolled adult patients with AIS; (2) reported the incidence or prevalence of DVT, risk factors for DVT after AIS, or the use and effectiveness of prophylactic interventions (pharmacological or mechanical); and (3) were designed as randomized controlled trials (RCTs), cohort studies, case–control studies, or other observational designs. Systematic reviews and meta-analyses were screened for relevant primary studies but were not pooled directly to avoid duplication. Case reports, small case series (<20 patients), pediatric populations, and non-English publications were excluded.

2.3. Data Extraction

All article titles and abstracts were initially reviewed in Endnote (Clarivate Analytics, London, UK) to exclude studies that did not meet the eligibility criteria. The remaining articles underwent full-text examination to confirm suitability for inclusion in the systematic review or meta-analysis. Data extraction was performed using a standardized sheet, capturing study-level demographics (author, country, publication year, registry or trial name, study design, number of centers), intervention characteristics (IVT, EVT, or both), though treatment-specific data were limited and inconsistently reported, and patient demographics (age, sex). Clinical and biological predictors collected included hypertension, diabetes mellitus, hyperlipidaemia, obesity, atrial fibrillation, tobacco and alcohol use, drug abuse, coronary artery disease, malignancy, respiratory infection, stroke severity measured by the National Institutes of Health Stroke Scale (NIHSS), admission glucose, D-dimer, low-density lipoprotein, and fibrinogen. Clinical outcomes were classified as patients who developed DVT versus those without DVT. When covariates or outcomes were incompletely reported, analyses were restricted to available cases; no imputation was undertaken for missing predictor variables, and such studies were excluded from specific pooled analyses.

2.4. Methodological Quality Assessment of Included Studies

The methodological quality assessment of included studies was conducted using the modified Jadad analysis (MJA) [30], completed independently by the primary researcher (Table S3). The risk of biases in results due to funding was also evaluated, based on the declaration of funding sources and conflicts of interest, extracted from each individual study (Table S4).

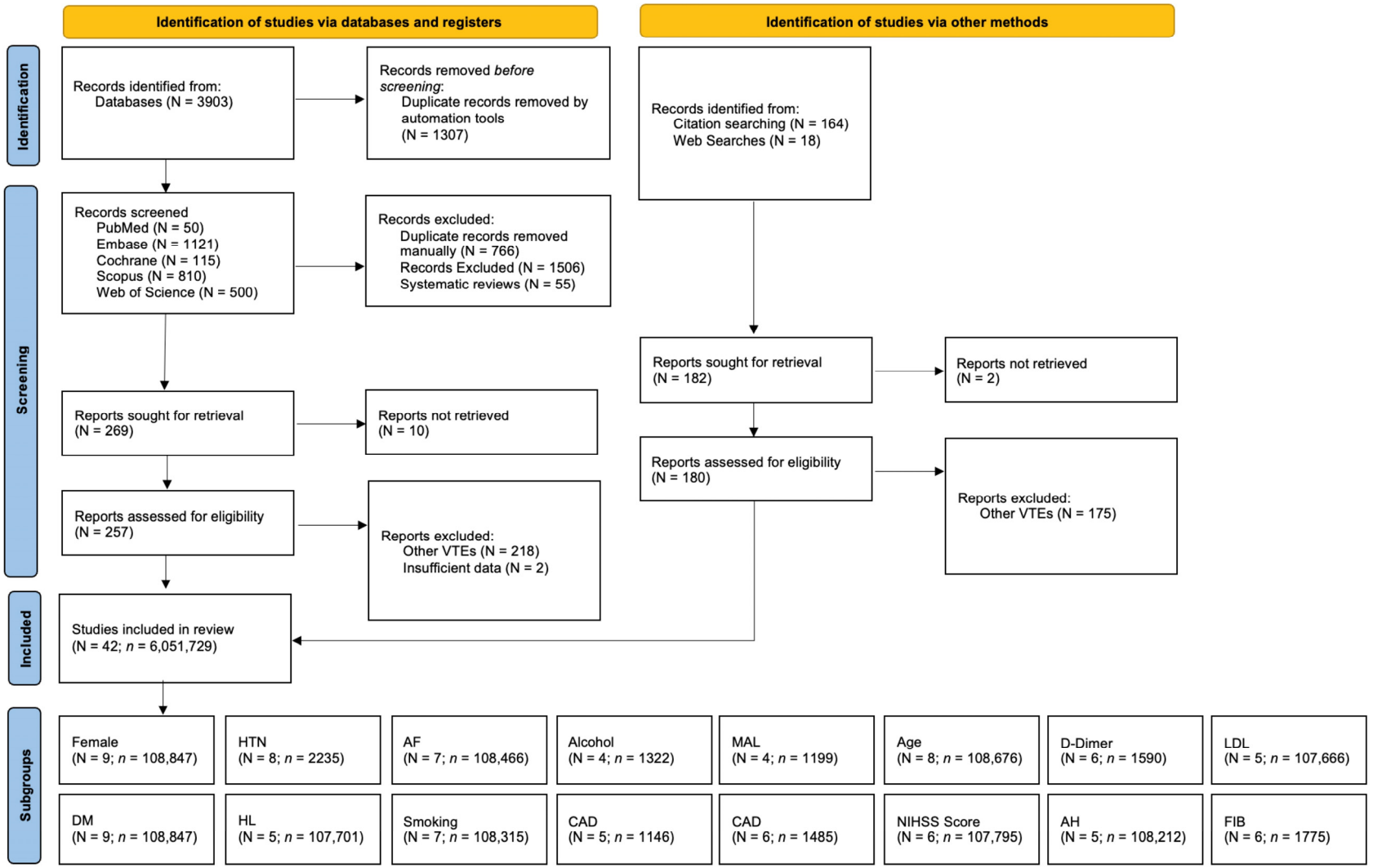

Figure 1. PRISMA flow diagram of study selection for the IRIS-DVT meta-analysis. Abbreviations: DVT: deep vein thrombosis; VTE: venous thromboembolism; HTN: hypertension; DM: diabetes mellitus; HL: hyperlipidemia; AF: atrial fibrillation; CAD: coronary artery disease; MAL: malignancy; NIHSS: National Institute of Health Stroke Scale; LDL: low density lipoprotein; AH: admission hyperglycemia; FIB: fibrinogen.

2.5. Certainty of Evidence Assessment (Grading)

The certainty of evidence across outcomes was assessed using the GRADE (Grading of Recommendations, Assessment, Development, and Evaluation) framework. The outcomes: incidence of DVT, risk factors, and prophylactic strategies were independently evaluated across five domains: risk of bias, inconsistency, indirectness, imprecision, and publication bias. Randomized controlled trials were initially rated as high certainty, whereas observational studies were rated as low certainty; evidence was subsequently downgraded for methodological limitations, heterogeneity, or imprecision, and upgraded in cases of large effect sizes, consistent associations, or dose–response relationships. Final ratings were categorized as high, moderate, low or very low.

2.6. Statistical Analyses

All statistical analyses were performed using STATA v13.0 (StataCorp, College Station, TX, USA). Baseline characteristics of included cohorts were extracted from each study. Where necessary, means and standard deviations (SDs) were estimated from medians and interquartile ranges (IQRs) using the method of Wan et al. [31], and combined with Bessel's correction to ensure unbiased SD estimates. The pooled prevalence of DVT among patients with AIS was calculated using the metaprop command, applying a random-effects meta-analysis of proportions with exact 95% confidence intervals (CIs) obtained using the cimethod (exact) and ftt options. Associations between clinical or biological factors and DVT were synthesized using DerSimonian and Laird (DL) random-effects models, generating pooled odds ratios (ORs) for categorical variables and standardized mean differences (SMDs) for continuous variables.

Subgroup analyses were conducted according to reperfusion therapy type (IVT, EVT, or both), stroke territory (anterior, posterior, mixed), study design (retrospective, prospective, or mixed), diagnostic modality, timing of DVT screening, geographical region, and mid-point year of data collection. Pharmacological prophylaxis (low-molecular-weight heparin, unfractionated heparin, direct oral anticoagulants) and mechanical prophylaxis (intermittent pneumatic compression, compression stockings, inferior vena cava filters) were grouped according to reported use, and pooled effect sizes were estimated using random-effects models where sufficient data were available. Temporal trend heterogeneity was assessed by stratifying prevalence according to midpoint year of data collection. Apparent fluctuations were interpreted primarily as methodological variation (diagnostic sensitivity, coding practices, and prophylaxis availability) rather than true secular shifts.

Forest plots were generated to display pooled effect sizes, weights, and heterogeneity estimates. Sensitivity analyses were performed using the metaninf command to assess the influence of individual studies on overall estimates. Between-study heterogeneity was quantified using the I^2 statistic, with thresholds of <30% (low), 30–50% (moderate), 50–75% (substantial), and >75% (severe). Cochran's Q test and Tau^2 were additionally reported. Publication bias was assessed using funnel plots and Egger's regression *test* (metabias and metafunnel commands). For selected predictors with consistent reporting, diagnostic accuracy was further assessed using summary receiver operating characteristic (SROC) curves and Fagan's nomograms to illustrate discriminative performance and post-test probability. Funnel plot asymmetry was interpreted in conjunction with Egger's *p*-values to evaluate bias risk. All statistical tests were two-tailed, and significance was set at $p < 0.05$. Detailed outputs of sensitivity analyses, funnel plots, SROC curves, and Fagan's nomograms are provided in Figures S11–S45. Pooled summary estimates for prophylactic interventions and continuous biomarkers are presented in Tables S7 and S8. Heterogeneity metrics (I^2, τ^2) and study weights are displayed in Figures 2 and 3.

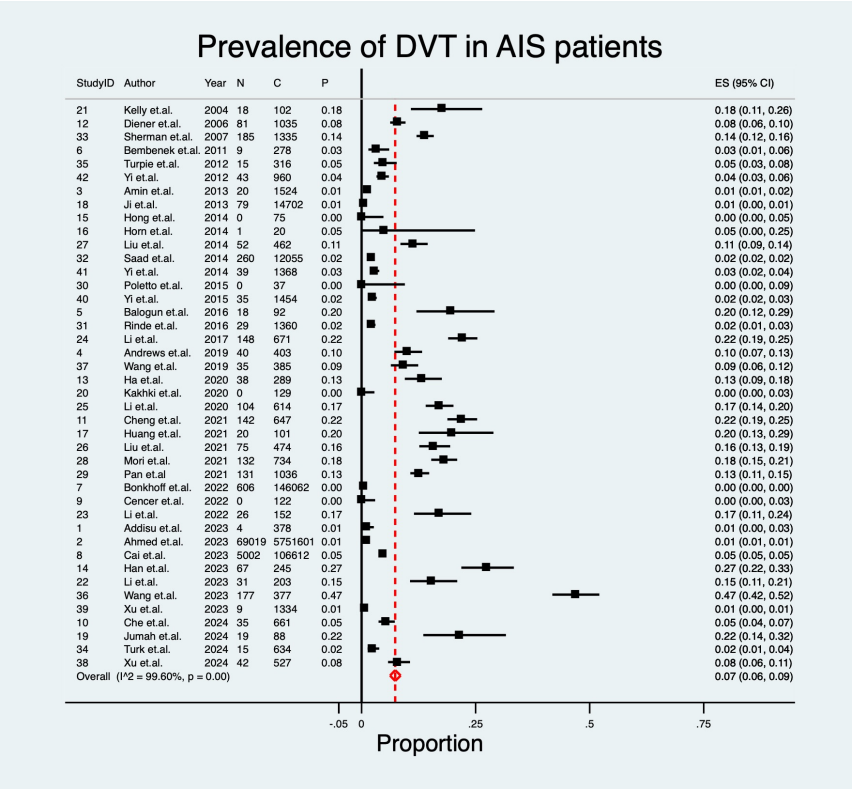

Figure 2. Forest plot of pooled prevalence of deep vein thrombosis in acute ischemic stroke patients.

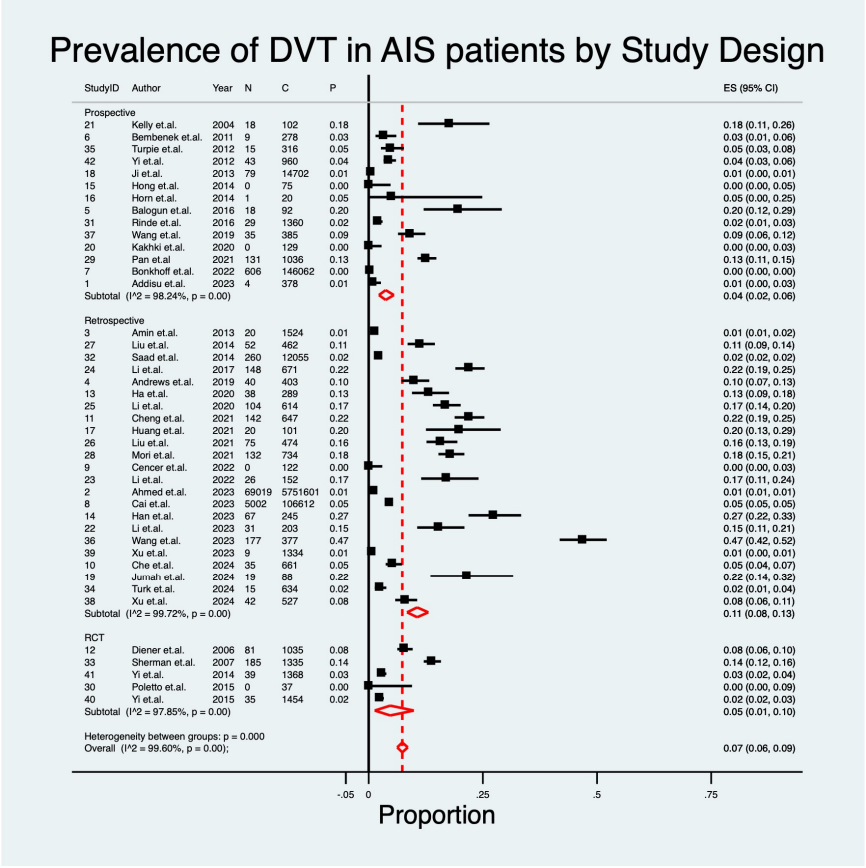

Figure 3. Forest plot of pooled prevalence of deep vein thrombosis stratified by study design. Abbreviation: DVT: Deep venous thrombosis; AIS: Acute ischemic stroke.

3. Results

3.1. Description of Included Studies

A total of 3903 records were initially identified through database searches (PubMed, Embase, Cochrane, Scopus, and Web of Science) and an additional 183 records via citation searching and web searches. After removing 1307 duplicates automatically and 766 manually, 2013 records were screened. Of these, 1561 were excluded for irrelevance or being systematic reviews. A further 12 reports were not retrieved. Finally, 438 full-text articles were assessed for eligibility, and 393 were excluded (391 for focusing on other VTEs and 2 for insufficient data).

Ultimately, 42 studies, encompassing 6,051,729 AIS patients, were included in the meta-analysis (PRISMA flow diagram, Figure 1). These studies varied in design, including retrospective cohorts and randomized controlled trials, and spanned a wide geographical and temporal range. Further details regarding study characteristics, sample sizes, and outcome measures are summarized. Baseline characteristics of the included studies are summarized in Table 1, with detailed study-level data presented in Table S6. Discrete and continuous risk factors for deep vein thrombosis (DVT) following acute ischemic stroke are summarized in Table 2 and Table 3, respectively. Table 4 presents the key predictors of DVT after stroke, while Table 5 outlines the effectiveness of various prophylactic interventions in preventing DVT among patients with acute ischemic stroke. A comprehensive summary of the certainty and quality of evidence across all key outcomes is provided in Table 6.

3.2. Pooled Prevalence of DVT in AIS

A total of 42 studies [5,10,32–72] reporting on the prevalence of DVT in patients with AIS, encompassing 6,051,729 patients, were included. The overall pooled prevalence of DVT was 7% (95% CI: 0.06–0.09; $p < 0.001$), with very high heterogeneity (I^2 = 99.6%) (Table 3; Figure 2). Early systematic screening within 72 h identified DVT in up to 23% of patients, underscoring the importance of detection timing. Subgroup analyses revealed considerable variability in the reported prevalence of DVT among AIS patients. When stratified by study design, retrospective studies (N = 23) [5,33–35,39–42,44,47,49,52–58,62,64,66,68,69] showed the highest pooled prevalence of 11% (95% CI: 0.08–0.13; I^2 = 99.7%), whereas randomized controlled trials (N = 5) [10,43,60,63,70,72] and prospective studies (N = 14) [32,36–38,45,46,48,50,51,59,61,65,67,71] reported lower estimates of 5% (95% CI: 0.01–0.10; I^2 = 97.9%) and 4% (95% CI: 0.02–0.06; I^2 = 97.8%), respectively (Table 3; Figures 3 and S4). Temporal stratification demonstrated fluctuations across years of data collection, with more recent studies in 2023 [49,68] estimating a prevalence of 9% (95% CI: 0.07–0.12), compared to higher values in 2022 [41,52,64,66] (14%, 95% CI: 0.01–0.36) and much lower rates in earlier years, such as 2012 [45,46,60,72] (1%) and 2013 [70] (2%). Older studies from 2004 [51] and 2006 [63,65] reported markedly higher prevalence rates of 18% and 12%, respectively (Table 3; Figure S3). These temporal differences likely reflect heterogeneity in design, population size, diagnostic methods, and prophylaxis practices, rather than true secular trends (Figure S46). Prevalence estimates fluctuated markedly across time, reflecting methodological variability rather than true secular trends. The unexpectedly low prevalence observed in 2012–2013 (≈1–2%) likely reflects methodological artifacts, including reliance on administrative coding rather than systematic imaging, regional differences in reporting, and smaller cohort sizes in those years, rather than a true secular decline in thrombotic risk.

For clarity, 'early systematic screening' refers to active ultrasound or imaging surveillance performed within 72 h of stroke onset, irrespective of symptoms.

Regional analysis also highlighted variation, with studies from Asia [5,39,41,42,44,45,47,48,52,53,55–59,66–72] (N = 22) reporting a pooled prevalence of 10% (95% CI: 0.07–0.14),

compared to 6% in Europe [36–38,51,61] (N = 6, 95% CI: 0.02–0.12) and 5% in North America [33–35,40,46,49,54,62,64] (N = 9, 95% CI: 0.03–0.07), while single studies from the Middle East [50], South America [60], and Africa [32] yielded very low prevalence estimates of 0–1% (Table 3; Figures 4 and S2). The consistently higher prevalence reported in Asian cohorts may reflect systematic early ultrasound screening, inclusion of more severe stroke populations, and smaller single-center designs, whereas North American and European estimates often relied on record-based surveillance with less sensitive methods. Thus, geographic variation is more likely methodological than biological, though differences in thromboprophylaxis protocols and patient demographics may contribute. The timing of DVT screening further influenced estimates, with studies performing early screening within 72 h of stroke [49,54,57,58,66] reporting the highest pooled prevalence (23%, 95% CI: 0.12–0.35), compared with intermediate windows of 1–2 weeks [5,36,37,42–44,47,55,56,59,63,70–73] (12–13%) and routine in-hospital surveillance without systematic screening (2%, 95% CI: 0.02–0.03) (Table 3; Figure 5, Figures S5 and S47) [32–35,38–41,45,46,48,50,53,61,62,67–69]. Diagnostic modality also proved influential: magnetic resonance direct thrombus imaging (MRDTI) [51] detected the highest prevalence (18%, 95% CI: 0.11–0.26), followed by compression Doppler ultrasonography [5,10,36,42–44,52,56,58,59,61,63,66,67] (15%, 95% CI: 0.08–0.24) and color Doppler ultrasound [37,39,41,45,47,54,55,57,65,68] (9%, 95% CI: 0.05–0.13). In contrast, duplex ultrasound alone [64,70–72] identified a prevalence of 3% (95% CI: 0.02–0.04), and studies relying on clinical records [32–35,38,40,46,48–50,53,60,62,69] reported the lowest prevalence (2%, 95% CI: 0.01–0.02) (Table 3; Figure S6).

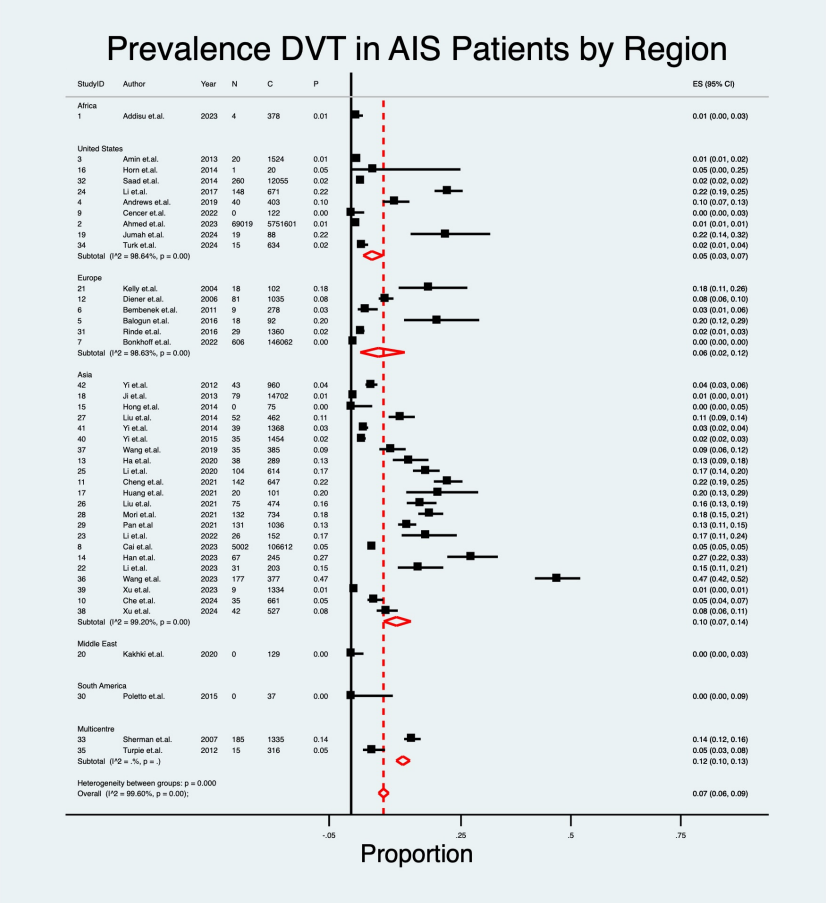

Figure 4. Forest plot of pooled prevalence of deep vein thrombosis stratified by geographical region. Abbreviation: DVT: Deep venous thrombosis; AIS: Acute ischemic stroke.

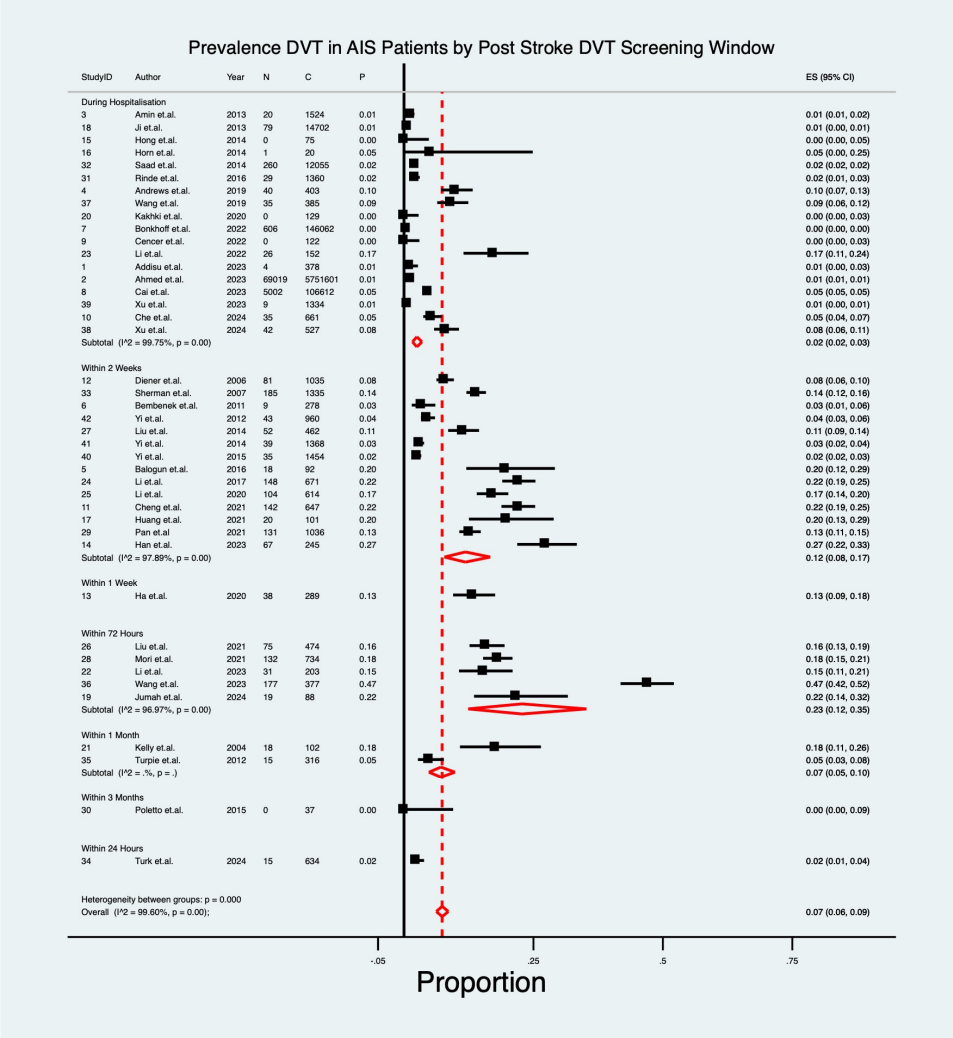

Figure 5. Forest plot of pooled prevalence of deep vein thrombosis stratified by timing of post-stroke screening. Abbreviation: DVT: Deep venous thrombosis; AIS: Acute ischemic stroke.

3.3. Predictive Indicators of DVT

Meta-analysis of discrete risk factors revealed that female sex [5,36,39,42,44,47,52,57,68] was significantly associated with higher odds of DVT (OR 1.33, 95% CI: 1.19–1.50; $p < 0.001$; I^2 = 50.4%) (Figure S8). In contrast, traditional vascular risk factors such as hypertension [5,36,42,44,47,52,57,68,74] (OR 0.79, 95% CI: 0.52–1.21; p = 0.28; I^2 = 77.2%), diabetes mellitus [5,36,39,42,44,47,52,57,68,74] (OR 1.06, 95% CI: 0.90–1.25; p = 0.49; I^2 = 65.4%), and hyperlipidemia [36,39,44,52,57] (OR 0.99, 95% CI: 0.65–1.50; p = 0.96; I^2 = 65.2%) showed no significant associations (Figures S8 and S9). Although atrial fibrillation [5,32,36,39,42,52,57,74] suggested a trend toward increased risk (OR 1.68, 95% CI: 0.93–3.05; p = 0.09), the results did not reach statistical significance and were highly heterogeneous (I^2 = 97.7%) (Figure S9). Tobacco use [5,36,39,44,52,57,68] showed an apparent inverse association (OR 0.77, 95% CI: 0.62–0.95; p = 0.016; I^2 = 24.2%), but this was likely artefactual, graded very low certainty, and should be interpreted cautiously (Figure S10). It is also possible that tobacco users represented a younger, less comorbid subset of AIS patients, or that competing risks such as early cardiovascular mortality limited detection of DVT in this group. Nonetheless, these alternative explanations underscore the likelihood of residual confounding, and the finding should not be interpreted as biologically protective. Other factors, including alcohol use [5,52,57,68] (OR 0.79, 95% CI: 0.52–1.20; p = 0.27; I^2 = 33.1%) and coronary artery disease [5,36,47,52,57,74] (OR 1.16, 95% CI: 0.87–1.56;

p = 0.30; I^2 = 11.1%), were not significantly associated (Figure S10). By contrast, malignancy [5,42,44,52,74] (OR 2.69, 95% CI: 1.56–5.22; p = 0.022; I^2 = 48.2%) and respiratory infection [5,42,47,52,57] (OR 2.30, 95% CI: 1.17–4.53; p = 0.016; I^2 = 48.7%) emerged as strong predictors of DVT (Figure S9). The association of respiratory infection with DVT may be pathophysiologically plausible, as systemic inflammation, cytokine activation, and prolonged immobility during infection can amplify hypercoagulability in the acute stroke setting. These mechanisms could explain the nearly two-fold increase in risk observed, despite the low certainty of evidence. Discrete predictive factors are summarized in Table 2.

Continuous predictors also demonstrated important associations. Older age [5,36,39,42,44,47,52,57] was consistently linked with higher DVT risk (SMD 0.32, 95% CI: 0.18–0.46; p < 0.001; I^2 = 60.8%) (Figure S13), while stroke severity, as measured by the NIHSS [5,36,39,44,47,57], showed a particularly robust relationship (SMD 0.41, 95% CI: 0.38–0.43; p < 0.001; I^2 = 0%) (Figure S13). Laboratory markers further reinforced this pattern: elevated D-dimer levels [5,36,42,44,47,52] (SMD 0.55, 95% CI: 0.38–0.72; p < 0.001; I^2 = 34.8%) and, to a lesser extent, higher admission glucose [5,39,42,52,57] (SMD 0.07, 95% CI: 0.04–0.09; p < 0.001; I^2 = 0%) were significantly associated with DVT (Figures S13 and S14). By contrast, LDL cholesterol [5,39,47,52,57] (SMD −0.03, 95% CI: −0.12 to 0.09; p = 0.73; I^2 = 27%) and fibrinogen [5,36,42,47,52,57] (SMD 0.01, 95% CI: −0.11 to 0.13; p = 0.87; I^2 = 0%) showed no significant associations (Figure S14). Further details on pooled heterogeneity estimates for continuous predictors, including D-dimer, admission glucose, LDL, and fibrinogen, are summarized in Table S8.

Overall, the most consistent predictors of DVT after AIS were stroke severity (NIHSS score), malignancy, female sex, older age, and elevated D-dimer levels, all supported by moderate-certainty evidence. Respiratory infection and admission glucose also showed associations but with lower certainty. In contrast, traditional vascular risk factors such as hypertension, diabetes mellitus, hyperlipidemia, and atrial fibrillation were not significantly associated, and LDL cholesterol and fibrinogen showed no meaningful relationship. The observed inverse association with tobacco use was likely confounded and should not be interpreted as protective. These findings suggest that clinical focus should shift toward neurological severity, cancer status, and selected biomarkers rather than conventional vascular comorbidities when stratifying DVT risk in AIS patients (Tables 4 and 5; Figures S8–S14).

Across 11 RCTs and cohort studies reporting prophylactic interventions, intermittent pneumatic compression (IPC) reduced DVT risk (pooled OR 0.62; 95% CI 0.41–0.93), while pharmacological agents such as LMWH showed a trend toward benefit (pooled OR 0.78; 95% CI 0.55–1.11), both graded low certainty (Tables S7 and S8).

3.4. Certainty of Evidence (GRADE)

The certainty of evidence across outcomes ranged from moderate to very low (Table 6). Moderate-certainty evidence supported the association of higher NIHSS scores, increasing age, elevated D-dimer levels, female sex, and malignancy with increased risk of DVT in AIS. Respiratory infection and admission glucose were supported by low-certainty evidence due to heterogeneity and modest effect sizes. The apparent inverse association with tobacco use was graded with very low certainty, reflecting likely residual confounding and inconsistency. Similarly, LDL and fibrinogen showed no significant associations and were rated very low. Evidence on the effectiveness of prophylactic interventions (e.g., LMWH, IPC) in AIS cohorts was insufficient and highly heterogeneous, warranting a very low certainty rating. Overall, while some predictors such as NIHSS and malignancy demonstrate robust and consistent associations, most other outcomes remain supported by low- to very low-certainty evidence.

Table 1. Baseline clinical and methodological characteristics of studies included in the IRIS-DVT meta-analysis.

ID	Author	Year	Cohort Size	Crude Prevalence of DVT n (n%)	Country	Study Design	Primary Stroke Treatment	Immobilization Post Stroke?	DVT Diagnosis Modality	Diagnosis Days Post Thrombectomy (Median)	Chemical DVT Prophylaxis	Physical DVT Prophylaxis
1	Addisu et al. [32]	2023	378	4 (1.1)	Ethiopia	Retrospective	No Acute Reperfusion	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
2	Ahmed et al. [33]	2023	5,751,601	69,019 (1.2)	United States	Retrospective	IV tPA or MT	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
3	Amin et al. [34]	2013	1524	20 (1.3)	United States	Retrospective	NA	Unspecified	Medical Notes	During Hospitalization	Variable	Variable
4	Andrews et al. [35]	2019	403	40 (9.9)	United States	Retrospective	MT	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
5	Balogun et al. [36]	2016	92	18 (19.6)	United Kingdom	Retrospective	No Acute Reperfusion	Unspecified	CDU	Within 2 Weeks	Antiplatelet	Not Routine
6	Bembenek et al. [37]	2011	269	9 (3.2)	Poland	Prospective	Unspecified	Unspecified	Color Doppler Ultrasound	Within 2 Weeks	Variable	Unspecified
7	Bonkhoff et al. [38]	2022	146,062	606 (0.4)	Germany	Retrospective	IV tPA when possible	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
8	Cai et al. [39]	2023	106,612	5002 (4.7)	China	Retrospective	No Acute Reperfusion	Unspecified	Color Doppler Ultrasound	During Hospitalization	Variable	Variable
9	Cencer et al. [40]	2022	122	0 (0.0)	United States	Retrospective	IV tPA	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
10	Che et al. [41]	2024	661	35 (5.3)	China	Retrospective	EVT	Unspecified	CDU	During Hospitalization	Unspecified	Unspecified
11	Cheng et al. [42]	2021	431	142 (21.9)	China	Retrospective	Variable	Unspecified	CUS	Within 2 Weeks	Unspecified	Unspecified
12	Diener et al. [43]	2006	1035	81 (7.8)	Multicenter	RCT	Variable	Unspecified	CDU	Within 2 Weeks	Antiplatelet or Anticoagulant	Unspecified
13	Ha et al. [44]	2020	289	38 (13.1)	Korea	Retrospective	IV tPA when possible	Unspecified	CUS	Within 1 Week	Not Routine	Not Routine
14	Han et al. [5]	2023	245	67 (27.3)	China	Retrospective	EVT	Yes	CDU	Within 2 Weeks	Antiplatelet	IPC
15	Hong et al. [45]	2014	75	0 (0.0)	Korea	Prospective	Variable	Variable	CDU	During Hospitalization	Unspecified	Unspecified
16	Horn et al. [46]	2014	20	1 (5.0)	United States	Prospective	MT	Yes	Medical Notes	During Hospitalization	Unspecified	Unspecified
17	Huang et al. [47]	2021	101	20 (19.8)	China	Retrospective	NA	Unspecified	CDU	Within 2 Weeks	Not Routine	Unspecified
18	Ji et al. [48]	2013	14,702	79 (0.5)	China	Retrospective	Unspecified	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
19	Jumah et al. [49]	2024	88	19 (21.6)	United States	Retrospective	Variable	Unspecified	Medical Notes	Within 72 h	IV Heparin	Unspecified
20	Kakhki et al. [50]	2020	129	0 (0.0)	Iran	Retrospective	Unspecified	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
21	Kelly et al. [75]	2004	102	18 (17.6)	United Kingdom	Prospective	Unspecified	Unspecified	MRDTI	Within 1 Month	Aspirin	GCS
22	Li et al. [52]	2023	234	31 (15.3)	China	Retrospective	Unspecified	Unspecified	CUS	Within 72 h	Unspecified	Unspecified

Table 1. *Cont.*

ID	Author	Year	Cohort Size	Crude Prevalence of DVT n (n%)	Country	Study Design	Primary Stroke Treatment	Immobilization Post Stroke?	DVT Diagnosis Modality	Diagnosis Days Post Thrombectomy (Median)	Chemical DVT Prophylaxis	Physical DVT Prophylaxis
23	Li et al. [53]	2022	152	26 (17.1)	China	Retrospective	IV tPA	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
24	Li et al. [54]	2017	671	148 (22.1)	United States	Retrospective	Unspecified	Unspecified	CDU	Within 2 Weeks	None Used	None Used
25	Li et al. [55]	2020	614	104 (16.9)	China	Retrospective	MT	Unspecified	CDU	Within 2 Weeks	Unspecified	Unspecified
26	Liu et al. [57]	2021	474	75 (15.8)	China	Retrospective	IV tPA	Yes	CDU	Within 72 h	Antiplatelet	Unspecified
27	Liu et al. [56]	2014	462	52 (11.3)	China	Retrospective	Variable	Unspecified	CUS	Within 2 Weeks	Antiplatelet or Anticoagulant	Unspecified
28	Mori et al. [58]	2021	734	132 (18.0)	Japan	Retrospective	Unspecified	Unspecified	CUS	Within 72 h	Unspecified	Unspecified
29	Pan et al. [59]	2021	1036	131 (12.6)	China	Retrospective	Variable	Unspecified	CDU	Within 2 Weeks	Antiplatelet or Anticoagulant	Unspecified
30	Poletto et al. [60]	2015	37	0 (0.0)	Brazil	RCT	IV tPA when possible	Unspecified	Medical Notes	Within 3 Months	Unspecified	Unspecified
31	Rinde et al. [61]	2016	1360	29 (2.1)	Norway	Retrospective	Variable	Unspecified	CUS	During Hospitalization	Unspecified	Unspecified
32	Saad et al. [62]	2014	12,055	260 (2.2)	United States	Retrospective	MT	Unspecified	Medical Notes	During Hospitalization	Unspecified	Unspecified
33	Sherman et al. [63]	2007	1762	185 (13.9)	Multicenter	RCT	Variable	Yes	CUS	Within 2 Weeks	Antiplatelet or Anticoagulant	Unspecified
34	Turk et al. [64]	2024	634	15 (2.4)	United States	Retrospective	Unspecified	Unspecified	Duplex Ultrasonography	Within 24 h	Variable	Unspecified
35	Turpie et al. [65]	2012	316	15 (4.7)	Multicenter	Prospective	Unspecified	Unspecified	CDU	Within 1 Month	Not Routine	Unspecified
36	Wang et al. [66]	2023	377	177 (46.9)	China	Retrospective	IV tPA	Unspecified	CUS	Within 72 h	Unspecified	Unspecified
37	Wang et al. [67]	2019	385	35 (9.1)	NA	Prospective	Unspecified	Unspecified	CUS	During hospitalization	Unspecified	IPC
38	Xu et al. [68]	2024	369	42 (8.0)	China	Retrospective	Unspecified	Unspecified	CDU	During hospitalization	Unspecified	Unspecified
39	Xu et al. [69]	2023	1334	9 (0.7)	China	Retrospective	IV tPA	Unspecified	Medical Notes	During hospitalization	Unspecified	Unspecified
40	Yi et al. [70]	2015	1454	35 (2.4)	China	RCT	Unspecified	Unspecified	Duplex Ultrasonography	Within 2 Weeks	Antiplatelet	Unspecified
41	Yi et al. [72]	2014	1368	39 (2.9)	China	RCT	Unspecified	Unspecified	Duplex Ultrasonography	Within 2 Weeks	Antiplatelet or Anticoagulant	Unspecified
42	Yi et al. [71]	2012	960	43 (4.5)	China	Prospective	Unspecified	Unspecified	Duplex Ultrasonography	Within 2 Weeks	Unspecified	Unspecified

Abbreviations: NA: not available; DVT: deep vein thrombosis; MT: mechanical thrombectomy; IV tPA: intravenous tissue plasminogen activator; RCT: randomized controlled trial; IPC: intermittent pneumatic compression; CUS: compression ultrasound; CDU: color Doppler ultrasound; MRDTI: magnetic resonance direct thrombus imaging.

Table 2. Discrete risk factors for deep vein thrombosis after acute ischemic stroke: pooled effect estimates.

ID	Author	DVT	Female		HTN		DM		HL		AF		Smoking		Alcohol		CAD		MAL		RI	
		n (n%)	n (n%)		n (n%)		n (n%)		n (n%)		n (n%)		n (n%)		n (n%)		n (n%)		n (n%)		n (n%)	
			Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
1	Addisu et al. [32]	4 (1.1)	-	-	-	-	-	-	-	-	102 (27.0)	276 (73.0)	-	-	-	-	-	-	-	-	-	-
5	Balogun et al. [36]	18 (19.6)	18 (19.6)	74 (80.4)	36 (39.1)	56 (60.9)	18 (19.6)	74 (80.4)	18 (19.6)	74 (80.4)	18 (19.6)	74 (80.4)	18 (19.6)	74 (80.4)	-	-	18 (19.6)	74 (80.4)	-	-	-	-
8	Cai et al. [39]	5002 (4.7)	5002 (4.7)	101,610 (95.3)	-	-	5002 (4.7)	101,610 (95.3)	5002 (4.7)	101,610 (95.3)	5002 (4.7)	101,610 (95.3)	5002 (4.7)	101,610 (95.3)	-	-	-	-	-	-	-	-
11	Cheng et al. [42]	96 (22.3)	96 (22.3)	335 (77.7)	96 (22.3)	335 (77.7)	96 (22.3)	335 (77.7)	-	-	96 (22.3)	335 (77.7)	-	-	-	-	-	-	96 (22.3)	335 (77.7)	96 (22.3)	335 (77.7)
14	Ha et al. [44]	38 (13.1)	114 (39.4)	175 (60.6)	208 (72.0)	81 (28.0)	87 (30.1)	202 (69.9)	195 (67.5)	94 (32.5)	-	-	130 (45.0)	159 (55.0)	-	-	-	-	5 (1.7)	284 (98.3)	-	-
15	Han et al. [5]	67 (27.3)	87 (35.5)	158 (64.5)	142 (58.0)	103 (42.0)	42 (17.1)	203 (82.9)	-	-	102 (41.6)	143 (58.4)	77 (31.4)	168 (68.6)	52 (21.2)	193 (78.8)	10 (4.1)	235 (95.9)	15 (6.1)	230 (93.9)	152 (62.0)	93 (38.0)
18	Huang et al. [47]	20 (19.8)	29 (28.7)	72 (71.3)	83 (82.2)	18 (17.8)	15 (14.9)	86 (85.1)	-	-	-	-	-	-	-	-	23 (22.8)	78 (77.2)	0 (0.0)	101 (100.0)	66 (65.3)	35 (34.7)
23	Li et al. [52]	31 (15.3)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)	31 (13.2)	203 (86.8)
27	Liu et al. [57]	75 (15.8)	142 (30.0)	332 (70.0)	284 (59.9)	190 (40.1)	120 (25.3)	354 (74.7)	194 (40.9)	280 (59.1)	54 (11.4)	420 (88.6)	216 (45.6)	258 (54.4)	185 (39.0)	289 (61.0)	33 (7.0)	441 (93.0)	-	-	45 (9.5)	429 (90.5)
40	Xu et al. [68]	29 (7.9)	217 (58.8)	152 (41.2)	269 (72.9)	100 (27.1)	164 (44.4)	205 (55.6)	-	-	-	-	73 (19.8)	296 (80.2)	105 (28.5)	264 (71.5)	-	-	-	-	-	-

Abbreviations: n: number of patients; AF: atrial fibrillation; CAD: coronary artery disease; DM: diabetes mellitus; HTN: hypertension; HL: hyperlipidemia; MAL: malignancy/cancer diagnosis; RI: respiratory infection.

Table 3. Pooled prevalence of deep vein thrombosis in acute ischemic stroke: summary effects and heterogeneity across studies.

ID	Author	DVT	Age		NIHSS Score		D-Dimer		Admission Glucose		LDL		Fibrinogen	
		n (n%)	FR (Mean ± SD)		FR (Mean ± SD)		FR (Mean ± SD)		FR (Mean ± SD)		FR (Mean ± SD)		FR (Mean ± SD)	
			Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
5	Balogun et al. [36]	18 (19.6)	69.7 (13.4)	69.1 (14.5)	15.6 (7.3)	12.8 (7.3)	2.6 (1.9)	1.4 (1.3)	-	-	-	-	3.9 (1.4)	3.9 (1.4)
8	Cai et al. [39]	5002 (4.7)	69.8 (11.7)	67.2 (12.1)	7.6 (7.1)	5.3 (5.6)	-	-	6.8 (3.2)	6.6 (2.9)	2.8 (1.5)	2.8 (1.3)	-	-
11	Cheng et al. [42]	96 (22.3)	73.4 (8.4)	68.9 (12.0)	-	-	2.2 (1.9)	1.6 (1.3)	6.0 (2.4)	6.0 (2.2)	-	-	3.6 (1.6)	3.6 (1.5)
14	Ha et al. [44]	38 (13.1)	71 (12.0)	68.4 (11.2)	7.4 (5.4)	4.5 (3.8)	12 (20.4)	8.5 (13.4)	-	-	-	-	-	-
15	Han et al. [5]	67 (27.3)	72.1 (9.1)	67.08 (11.7)	16 (4.5)	14.5 (5.7)	2.8 (2.4)	1.7 (1.7)	6.6 (1.8)	6.5 (1.7)	2.1 (0.7)	2.2 (0.8)	0.032 (0.0)	0.032 (0.0)
18	Huang et al. [47]	20 (19.8)	65 (16.4)	66 (16.2)	19.7 (10.6)	16.9 (10.2)	3.1 (5.3)	1.6 (3.3)	6.8 (3.2)	6.6 (2.9)	1.9 (1.2)	2.0 (1.2)	0.0025 (0.0)	0.0029 (0.0)
23	Li et al. [52]	31 (15.3)	64.7 (11.7)	60.2 (12.0)	-	-	1.9 (1.7)	0.8 (1.3)	6.1 (1.9)	6.3 (2.3)	1.9 (0.6)	1.7 (0.6)	4.3 (1.2)	4.1 (1.3)
27	Liu et al. [57]	75 (15.8)	69.8 (9.8)	62.7 (11.6)	9.7 (5.3)	8.0 (4.7)	-	-	8.0 (2.9)	8.1 (3.1)	2.92 (0.9)	3.0 (0.8)	3.15 (0.7)	3.1 (0.7)

Abbreviations: n: number of patients; NIHSS: National Institute of Health Stroke Scale; LDL: low-density lipoprotein.

Table 4. Predictors of deep vein thrombosis after acute ischemic stroke: meta-analysis of categorical and continuous variables.

Subgroup	N	Pooled Prevalence Rate (from Meta-Analysis)	95% CI	z-Score	*p*-Value	I^2	$\tau^2 \leq$
Overall	42	7%	0.06–0.09	21.76.	$p < 0.01$	99.60%	0.02
Study Design							
Retrospective	23	11%	0.08–0.13	15.96	$p < 0.01$	99.72%	-
Prospective	14	4%	0.02–0.06	7.54	$p < 0.01$	98.24%	-
RCT	5	5%	0.01–0.10	3.70	$p < 0.01$	97.85%	-
Region							
Asia	22	10%	0.07–0.14	3.24	$p < 0.01$	99.20	-
Europe	6	6%	0.02–0.12	4.20	$p < 0.01$	98.63	-
North America	9	5%	0.03–0.07	7.18	$p < 0.01$	98.64	-
Middle East	1	0%	0.00–0.03	0.00	-	-	-
Africa	1	1%	0.00–0.03	3.24	-	-	-
South America	1	0%	0.00–0.09	0.00	-	-	-
Multiple	2	12%	0.10–0.13	26.77	-	-	-
DVT Screening Post Stroke Screening							
Within 24 h	1	2%	0.02–0.04	6.90	-	-	-
Within 72 h	5	23%	0.12–0.35	6.66	$p < 0.01$	96.97%	-
Within 1 Week	1	13%	0.09–0.18	11.69	-	-	-
Within 2 Weeks	14	12%	0.08–0.17	9.40	$p < 0.01$	97.89%	-
Within 1 Month	2	7%	0.05–10	9.63	-	-	-
Within 3 Months	1	0%	0.00–0.09	0.00	-	-	-
During hospitalization	18	2%	0.02–0.03	8.98	$p < 0.01$	99.75%	-
Diagnosis Modality							
MRDTI	1	18%	0.11–0.26	7.86	-	-	-
CDU	4	16%	0.09–0.13	7.09	$p < 0.01$	95.25%	-
CUS	9	15%	0.08–0.24	6.79	$p < 0.01$	98.55%	-
Duplex Ultrasound	4	3%	0.02–0.04	12.07	$p < 0.01$	65.85%	-
Color Doppler Ultrasound	10	9%	0.05–0.13	7.09	$p < 0.01$	97.80%	-
Medical Notes	14	2%	0.01–0.02	8.49	$p < 0.01$	99.14%	-
Temporal Trends							
2023	2	9%	0.07–0.12	13.68	-	-	
2022	4	14%	0.01–0.36	2.71	$p < 0.01$	-	
2021	2	8%	0.06–0.10	12.69	-	-	
2020	3	8%	0.00–0.24	2.34	-	-	
2019	7	9%	0.05–0.13	8.36	$p < 0.01$	-	
2018	1	9%	0.06–0.12	11.10	-	-	
2017	2	0%	0.00–0.00	38.41	-	-	
2016	4	13%	0.04–0.23	4.45	$p < 0.01$	-	
2013	1	2%	0.02–0.03	10.96	-	-	
2012	4	1%	0.00–0.03	1.71	$p = 0.14$	-	
2011	2	2%	0.02–0.02	24.91	-	-	
2010	1	4%	0.03–0.06	12.29	-	-	
2009	1	3%	0.01–0.06	5.19	-	-	
2008	2	1%	0.00–0.01	17.71	-	-	
2007	2	3%	0.02–0.04	13.40	-	-	
2006	2	12%	0.10–0.13	26.77	-	-	
2004	1	18%	0.11–0.26	7.86	-	-	
2003	1	8%	0.06–0.10	17.3	-	-	

Abbreviations N: number of studies; CI: confidence interval; RCT: randomized controlled trial; MRDTI: magnetic resonance direct thrombus imaging; CDU: compression Doppler ultrasound; CUS: compression ultrasound.

Table 5. Effectiveness of prophylactic interventions for preventing deep vein thrombosis in acute ischemic stroke patients.

				Summary Effects REDL		Heterogeneity ¶				Heterogeneity Variance Estimates	
Outcome	**N (Studies)**	**n (Cohort)**	**Effect Measure**	**Effect (OR/SMD)**	**[95% CI]**	**Tests of Overall Effect**	**z Score**	**Cochrane's Q**	**H**	**$I^2 \leq$ ***	**$\tau^2 \leq$ $^{\Phi}$**
Female	9	1,439,000	OR	1.332	[1.185; 1.498]	$p < 0.001$	4.797	16.14	1.42	50.40%	0.0077
Respiratory Infection	4	1054	OR	2.301	[1.169; 4.529]	$p = 0.016$	2.411	5.85	1.396	48.70%	0.1019
Malignancy	5	1,335,226	OR	2.69	[1.557; 5.215]	$p = 0.022$	2.298	7.72	1.389	48.20%	0.3959
Atrial Fibrillation	8	1,442,445	OR	1.684	[0.930; 3.049]	$p = 0.085$	1.721	310.93	6.665	97.70%	0.5145
Coronary Artery Disease	7	1,335,554	OR	1.164	[0.871; 1.556]	$p = 0.304$	1.028	6.75	1.061	11.10%	0.0286
Peripheral Vascular Disease	4	1,437,577	OR	1.477	[0.665; 3.283]	$p = 0.339$	0.957	186.79	7.891	98.40%	0.5098
Diabetes Mellitus	10	1,442,824	OR	1.06	[0.898; 1.250]	$p = 0.493$	0.685	26.02	1.7	65.40%	0.0188
Hyperlipidemia	5	107,701	OR	0.989	[0.653; 1.497]	$p = 0.957$	−0.054	11.49	1.695	65.20%	0.125
Hypertension	9	1,336,167	OR	0.791	[0.515; 1.214]	$p = 0.283$	−1.074	35.11	2.095	77.20%	0.2682
Alcohol Use	4	1322	OR	0.789	[0.517; 1.203]	$p = 0.271$	−1.102	4.49	1.223	33.10%	0.0618
Tobacco Use	7	108,315	OR	0.767	[0.618; 0.952	$p = 0.016$	−2.402	7.91	1.148	24.20%	0.0216
NIHSS Score	6	113,033	SMD	0.405	[0.377; 0.433]	$p < 0.001$	28.446	4.59	0.958	0%	0
D-Dimer	6	1662	SMD	0.551	[0.378; 0.723]	$p < 0.001$	6.24	7.66	1.238	34.80%	0.0157
LDL	5	112,861	SMD	−0.34	[−0.126; 0.088]	$p = 0.734$	5.48	5.48	1.17	27%	0.0047
Admission Glucose	5	113,267	SMD	0.066	[0.039; 0.094]	$p < 0.001$	4.687	1.3	0.569	0%	0
Age	8	113,825	SMD	0.32	[0.181; 0.460]	$p < 0.001$	4.494	17.84	1.596	60.80%	0.0197
Fibrinogen	6	1884	SMD	0.01	[−0.112; 0.133]	$p = 0.869$	0.165	2.43	0.697	0%	0

Abbreviations: N, number of studies; n, number of patients; OR, odds ratio; CI, confidence interval; REDL, DerSimonian and Laird random-effects method; Q, heterogeneity measure calculated with 95% CIs based on the noncentral χ^2 (common-effect) distribution for Cochran's Q test; H, relative excess in Cochran's Q over its degrees of freedom; NIHSS, National Institutes of Health Stroke Scale/Score; LDL, low-density lipoprotein; I^2, proportion of total variation in effect estimates attributable to between-study heterogeneity (based on Cochran's Q test); τ^2, between-study variance for subgroup heterogeneity comparisons; *, values of I^2 are expressed as percentages; ¶, heterogeneity values calculated with 95% CIs based on the gamma (random-effects) distribution for Q; $^{\Phi}$, heterogeneity variance estimates (τ^2) derived from the DerSimonian and Laird method.

Table 6. GRADE summary of evidence on incidence, risk factors, and prophylaxis of deep vein thrombosis in acute ischemic stroke (IRIS-DVT study).

A. Incidence/Prevalence										
Outcome	No. of Studies (N)	Patient Number (n)	Effect Estimate (95% CI)	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Certainty of Evidence (GRADE)	Reasons for Downgrade/Upgrade
Prevalence of DVT in AIS	42	6,051,729	Pooled prevalence: 7% (95% CI 5–9%)	Low	Moderate (regional and temporal heterogeneity)	Low	Minimal	Possible	●●●○ Moderate	Downgraded: heterogeneity; Upgraded: large sample size, precise estimates
B. Risk Factors										
Predictor	No. of Studies (N)	Patient Number (n)	Effect Estimate (OR/SMD, 95% CI)	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Certainty of Evidence (GRADE)	Reasons for Downgrade/Upgrade
Stroke severity (NIHSS)	6	107,795	SMD 0.41 (0.38–0.43)	Low	Very low (I^2 = 0%)	Low	Minimal	Unlikely	●●●○ Moderate	Downgraded: observational designs; Upgraded: strong, consistent effect
Age	8	108,676	SMD 0.32 (0.18–0.46)	Low	Moderate ($I^2 \approx 61\%$)	Low	Minimal	Unlikely	●●●○ Moderate	Downgraded: inconsistency; Upgraded: large sample size
Female sex	9	108,847	OR 1.33 (1.19–1.50)	Low	Moderate ($I^2 \approx 50\%$)	Low	Adequate	Possible	●●●○ Moderate	Downgraded: inconsistency; Upgraded: robust effect
D-dimer elevation	6	1590	SMD 0.55 (0.38–0.72)	Low	Low–moderate ($I^2 \approx 35\%$)	Low	Minimal	Possible	●●●○ Moderate	Downgraded: possible bias; Upgraded: strong effect
Malignancy	5	1199	OR 2.69 (1.56–5.22)	Low	Moderate ($I^2 \approx 48\%$)	Low	Somewhat wide CI	Possible	●●●○ Moderate	Downgraded: inconsistency; Upgraded: large effect
Respiratory infection	5	1485	OR 2.30 (1.17–4.53)	Moderate	Moderate ($I^2 \approx 49\%$)	Low	Wide CI	Likely	●●○○ Low	Downgraded: inconsistency, imprecision, bias
Admission hyperglycemia	5	108,212	SMD 0.07 (0.04–0.09)	Low	Low (I^2 = 0%)	Low	Small effect	Possible	●●○○ Low	Downgraded: trivial effect size, possible bias
Tobacco use (inverse)	7	108,315	OR 0.77 (0.62–0.95)	High	Low ($I^2 \approx 24\%$)	High	CI near null	Likely	●○○○ Very Low	Downgraded: confounding, indirectness, bias
LDL cholesterol	5	107,666	SMD −0.03 (−0.12–0.09)	Moderate	Low ($I^2 \approx 27\%$)	Moderate	Null effect, small n	Likely	●○○○ Very Low	Downgraded: imprecision, indirectness
Fibrinogen	6	1775	SMD 0.01 (−0.11–0.13)	Moderate	Low (I^2 = 0%)	Moderate	Wide CI incl. null	Possible	●○○○ Very Low	Downgraded: imprecision, indirectness

Table 6. *Cont.*

C. Prophylaxis										
Intervention	No. of Studies (N)	Patient Number (n)	Effect Estimate (OR/SMD, 95% CI)	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Certainty of Evidence (GRADE)	Reasons for Downgrade/Upgrade
Pharmacological prophylaxis (Anticoagulants)	4	1066	Heterogeneous, no stable pooled estimate	High (small, observational)	Very low (I^2 = 0%)	Low	Wide CI	Likely	●○○○ Very Low	Downgraded: high risk of bias, small observational
Pharmacological prophylaxis (Antiplatelets)	5	1531	Heterogeneous, no stable pooled estimate	High (small, observational)	Very low (I^2 = 0%)	Low	Wide CI	Likely	●○○○ Very Low	Downgraded: high risk of bias, small observational
IPC (intermittent pneumatic compression)	3	732	Trend toward reduced DVT; effect inconsistent	Moderate	Moderate–high	Low	Moderate	Possible	●○○○ Very Low	Downgraded: inconsistency, imprecision, small observational

This table presents pooled estimates, certainty of evidence, and rationale for grading according to the GRADE framework, with outcomes stratified into incidence/prevalence, risk factors, and prophylactic interventions. Certainty of evidence was assessed across five domains: risk of bias, inconsistency, indirectness, imprecision, and publication bias. Based on 42 studies (n > 6 million), the pooled prevalence of DVT in AIS was 7% (95% CI 5–9%), approximately seventy times higher than in the general population. Among risk factors, moderate-certainty evidence supports stroke severity (NIHSS), older age, female sex, elevated D-dimer, and malignancy as consistent predictors, while low-certainty evidence was found for respiratory infection and admission hyperglycemia; tobacco use showed an inverse association but with very low certainty, likely due to confounding, and LDL cholesterol and fibrinogen were not significantly associated. For prophylaxis, evidence for pharmacological interventions (LMWH, UFH, DOACs) was highly heterogeneous and graded very low certainty, while IPC showed a directional trend toward reduced DVT incidence but remains very low certainty due to limited and inconsistent RCT data. Overall, the strongest and most reliable predictors of DVT in AIS were NIHSS, age, D-dimer, female sex, and malignancy (moderate certainty), whereas preventive strategies remain underinvestigated with substantial uncertainty. Abbreviations: AIS, Acute Ischemic Stroke; CI, Confidence Interval; DVT, Deep Vein Thrombosis; DOACs, Direct Oral Anticoagulants; GRADE, Grading of Recommendations, Assessment, Development and Evaluation; IPC, Intermittent Pneumatic Compression; I^2, I-squared statistic (heterogeneity measure); LMWH, Low Molecular Weight Heparin; NIHSS, National Institutes of Health Stroke Scale; OR, Odds Ratio; RCT, Randomized Controlled Trial; SMD, Standardized Mean Difference; UFH, Unfractionated Heparin.

3.5. Prophylactic Interventions

Evidence regarding prophylactic strategies for DVT prevention in AIS was heterogeneous and limited. Pharmacological measures such as low-molecular-weight heparin (LMWH) and direct oral anticoagulants, along with mechanical approaches including intermittent pneumatic compression (IPC), were variably reported across studies. Pooled estimates did not allow firm conclusions regarding their effectiveness, given wide heterogeneity, limited sample sizes, and inconsistent reporting of outcomes (Tables S7 and S8; Figures S12, S19, S31, S39 and S45). As such, the certainty of evidence for prophylaxis effectiveness in AIS remains very low. Pooled summary effects for pharmacological prophylaxis (LMWH, UFH, DOACs) and mechanical interventions (IPC, stockings, IVC filters) are presented in Table S7. While pooled analyses (Table S7, Figures S12, S19, S31, S39 and S45) did not allow firm conclusions, a directional trend toward reduced DVT incidence with LMWH and IPC was observed, albeit with high heterogeneity. Pharmacological prophylaxis estimates were particularly inconsistent, while IPC showed more reproducible effects in smaller RCTs. These analyses demonstrated high heterogeneity and limited certainty, consistent with our main findings.

3.6. Sensitivity and Bias Analyses

Sensitivity analyses, performed by sequentially excluding individual studies, did not materially alter pooled prevalence or risk factor estimates, confirming the robustness of the main findings (Figures S15–S21). Assessment of publication bias using funnel plots and Egger's regression revealed potential small-study effects for some predictors; however, patterns were inconsistent and did not systematically affect the overall conclusions (Figures S22–S33). Sensitivity analyses confirmed the robustness of the pooled estimates (see Figures S15–S21). Funnel plot and Egger's regression results are illustrated in Figures S22–S33. Diagnostic performance measures, including SROC and Fagan analyses,

are provided in Figures S34–S45. Detailed heterogeneity outputs for prophylactic strategies and continuous predictors are reported in Tables S7 and S8.

4. Discussion

The IRIS-DVT study provides the most comprehensive synthesis to date on thrombotic complications in AIS, addressing a longstanding evidence gap. By focusing exclusively on AIS, rather than grouping with hemorrhagic or mixed stroke subtypes as earlier reviews did, this meta-analysis delivers a more precise and clinically applicable understanding of DVT in stroke care. Our pooled prevalence estimates of 7%, seventy times higher than the general population, confirms AIS as a distinct high-risk condition that warrants systematic preventive strategies [76].

A key finding is the impact of screening timing on prevalence. Systematic imaging within 72 h identified DVT in nearly one in four patients, compared with substantially lower rates when screening was delayed or unsystematic. This suggests that a large proportion of thrombi remain clinically silent unless actively sought. Current AHA/ASA (2021) and ESO (2016) guidelines recommend prophylaxis in immobilized AIS patients but provide no clear direction on optimal detection windows [77–79]. Our results argue for early systematic screening within the first 72 h, and no later than two weeks, to minimize underdiagnosis (Figure S47). This is supported by smaller cohort studies and carries direct implications for updating practice guidelines [49,54,57,58,66].

Regional differences may partly reflect genetic predispositions such as Factor V Leiden or prothrombin G20210A variants, which are rare in East Asian but common in European cohorts, influencing baseline thrombotic risk [80]. Moreover, variability in post-thrombectomy immobilization and delayed prophylaxis initiation may accentuate risk after reperfusion therapies [81]. Dedicated studies stratifying by ancestry, thrombophilia, and treatment modality are warranted to clarify these interactions. Temporal patterns further contextualize our findings. The decline in prevalence around 2006 coincided with the introduction of mandatory hospital-wide VTE assessments and prophylaxis protocols in the US, UK, and France, while the rise after 2015 paralleled the global adoption of EVT following pivotal trials [82,83] (Figure S46). Although most included studies did not report treatment modality, these temporal shifts strongly suggest evolving practice patterns influence DVT risk. EVT may contribute through longer procedural times, femoral access, and immobility, whereas IVT-treated patients face deferred prophylaxis in the first 24 h [5,84–87]. These observations [88] underscore an urgent need for dedicated studies examining reperfusion-specific thrombotic risk, which current guidelines do not yet address.

Among predictors, stroke severity (NIHSS) emerged as the most consistent and powerful risk factor, reinforcing the clinical intuition that severely affected, immobilized patients require early prophylaxis. Elevated D-dimer, admission hyperglycaemia, and older age also showed significant associations, reflecting systemic hypercoagulability and metabolic stress [89,90]. Interestingly, LDL and fibrinogen, which are established pro-thrombotic markers in other contexts, did not demonstrate significant associations in AIS populations. This may reflect underreporting, heterogeneity in laboratory measurement, or confounding from acute-phase responses, rather than true absence of pathophysiological relevance. Malignancy, respiratory infection, and female sex were robust categorical predictors, while traditional vascular risk factors, including hypertension, diabetes, hyperlipidaemia, and atrial fibrillation, were not significantly associated. This divergence highlights that AIS-related thrombosis is shaped by acute systemic and neurological stressors rather than chronic comorbidities. The apparent inverse association with tobacco use is likely artefactual, reflecting residual confounding or selection bias, and should not influence practice.

Despite broad use of low-molecular-weight heparin, unfractionated heparin, direct oral anticoagulants, and IPC, evidence on prophylaxis effectiveness in AIS remains limited and heterogeneous. Our findings align with the CLOTS 3 trial [91], which demonstrated IPC reduces proximal DVT, supporting its consistent endorsement in guidelines. By contrast, pharmacological prophylaxis remains debated, with bleeding risk often outweighing uncertain benefit. Both AHA/ASA [78] and ESO guidelines [79] recommend IPC as first-line and acknowledge uncertainty around anticoagulants; our GRADE assessment concurs, rating evidence for prophylaxis as very low certainty (Table 6). Our interpretation of prophylactic effectiveness and continuous biomarker predictors is supported by the extended analyses in Tables S7 and S8, which confirm the limited certainty and heterogeneity underlying these associations.

The certainty of evidence across outcomes ranged from moderate to very low. Moderate-certainty evidence supported NIHSS, age, D-dimer, malignancy, and female sex as reliable predictors, while respiratory infection and glucose were supported by low certainty. Tobacco use, LDL, fibrinogen, and all prophylactic interventions were very low certainty. These gradings suggest that while certain predictors can confidently inform risk stratification, most associations remain tentative and highlight critical evidence gaps.

The implications for clinical practice are immediate. Screening should be standardized, with early ultrasound in high-risk patients and no later than two weeks for all immobilized AIS patients. Risk stratification should focus on stroke severity, systemic illness (malignancy, infection), and biomarkers such as D-dimer and glucose. IPC should be applied universally to immobilized patients, while pharmacological prophylaxis should be individualized according to bleeding risk. These findings call for refinement of existing guidelines, particularly in the EVT era, by incorporating timing of screening and stratified prophylaxis into routine stroke care.

This study has notable strengths, including its unprecedented sample size, inclusion of both categorical and continuous predictors, robust sensitivity analyses, and adherence to PRISMA/MOOSE methodology (Table S2). The use of the GRADE framework adds transparency and enhances clinical relevance. Limitations include high heterogeneity, reflecting variability in study design, populations, diagnostic methods, and prophylaxis practices. Treatment modality was poorly reported, limiting conclusions regarding IVT- and EVT-specific risks. Evidence on prophylaxis was sparse and inconsistent, and most studies were observational, leaving potential for residual confounding. Our conclusions regarding prophylaxis and continuous predictors should be interpreted in light of the additional analyses provided in Tables S7 and S8 and Figures S11–S45.

5. Conclusions

In conclusion, the IRIS-DVT study establishes AIS as a distinct high-risk population for DVT, with an estimated prevalence seven times higher than that of the general population. Our GRADE assessment shows that the most reliable predictors of DVT, stroke severity (NIHSS), malignancy, female sex, older age, and elevated D-dimer, are supported by moderate-certainty evidence, while other associations such as respiratory infection and admission glucose are of low certainty, and most prophylactic interventions remain backed only by very low-certainty data. These gradings underscore that while some risk factors can be confidently used for stratification, others demand cautious interpretation and further study. For clinical practice, the implications are immediate. AIS patients should undergo systematic DVT screening within 72 h of stroke onset, and no later than two weeks, with particular priority given to those with high NIHSS scores, malignancy, infection, or elevated D-dimer. Intermittent pneumatic compression should be applied consistently in immobilized patients, while pharmacological prophylaxis should be individualized according to

bleeding risk. By integrating these findings into evidence-based care pathways, clinicians can reduce the burden of DVT and pulmonary embolism in AIS. At the same time, major evidence gaps persist, particularly around optimal prophylaxis in reperfusion-treated patients and the effectiveness of anticoagulants in this setting. Addressing these uncertainties through adequately powered, AIS-specific trials will be crucial for refining guidelines and improving outcomes. The IRIS-DVT study provides comprehensive evidence base and a roadmap for translating these findings into actionable strategies in stroke medicine.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/ctn9040049/s1: Table S1: PRISMA-2020 Checklist; Table S2: MOOSE Checklist; Table S3: Modified Jadad Analysis for Methodological Quality; Table S4: Funding Bias Scores for Studies; Table S5: Outputs from Egger's Test for Publication Bias for Predictive Indicators; Table S6: Summary Effects and Heterogeneity from Meta-analysis of Discrete Risk Factors Associated with Deep Vein Thrombosis in Acute Ischemic Stroke Patients; Table S7: Summary Effects and Heterogeneity from Meta-analysis of Medications Associated with Deep Vein Thrombosis in Acute Ischemic Stroke Patients; Table S8: Summary Effects and Heterogeneity from Meta-analysis of Continuous Predictive Markers Associated with Deep Vein Thrombosis in Acute Ischemic Stroke Patients; Figure S1: Forest Plots of Prevalence of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS); Figure S2: Forest Plots of Prevalence of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Stratified by Country; Figure S3: Forest Plots of Prevalence of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Stratified by Data End Date; Figure S4: Forest Plots of Prevalence of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Stratified by Study Design; Figure S5: Forest Plots of Prevalence of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Stratified by DVT Screening Window; Figure S6: Forest Plots of Prevalence of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Stratified by DVT Diagnosis Modality; Figure S7: Forest Plots of Prevalence of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Stratified by Primary Stroke Treatment; Figure S8: Forest Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (1); Figure S9: Forest Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (2); Figure S10: Forest Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (3); Figure S11: Forest Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (4); Figure S12: Forest Plots of Medication Use Related Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients; Figure S13: Forest Plots of Continuous Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (1); Figure S14: Forest Plots of Continuous Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (2); Figure S15: Sensitivity Analysis of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (1); Figure S16: Sensitivity Analysis of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (2); Figure S17: Sensitivity Analysis of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (3); Figure S18: Sensitivity Analysis of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (4); Figure S19: Sensitivity Analysis of Medication Use Related Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients; Figure S20: Sensitivity Analysis of Continuous Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (1); Figure S21: Sensitivity Analysis of Continuous Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (2); Figure S22: Graphs of Egger's Regression Test for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (1); Figure S23: Graphs of Egger's Regression Test for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (2); Figure S24: Graphs of Egger's Regression Test for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (3); Figure S25: Graphs of Egger's

Regression Test for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (4); Figure S26: Graphs of Egger's Regression Test for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Medication Use in Acute Ischemic Stroke (AIS) Patients; Figure S27: Funnel Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (1); Figure S28: Funnel Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (2); Figure S29: Funnel Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (3); Figure S30: Funnel Plots of Discrete Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (4); Figure S31: Funnel Plots of Medication Use Related Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients; Figure S32: Funnel Plots of Continuous Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (1); Figure S33: Funnel Plots of Continuous Predictive Indicators of Deep Vein Thrombosis (DVT) in Acute Ischemic Stroke (AIS) Patients. (2); Figure S34: SROC for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (1); Figure S35: SROC for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (2); Figure S36: SROC for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (3); Figure S37: SROC for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (4); Figure S38: SROC for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (5); Figure S39: SROC on the Association between Deep Vein Thrombosis (DVT) and Medication Use in Acute Ischemic Stroke (AIS) Patients; Figure S40: Fagan Analysis for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (1); Figure S41: Fagan Analysis for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (2); Figure S42: Fagan Analysis for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (3); Figure S43: Fagan Analysis for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (4); Figure S44: Fagan Analysis for Meta-analysis on the Association between Deep Vein Thrombosis (DVT) and Risk Factors in Acute Ischemic Stroke (AIS) Patients. (5); Figure S45: Fagan Analysis on the Association between Deep Vein Thrombosis (DVT) and Medication Use in Acute Ischemic Stroke (AIS) Patients; Figure S46: Scatter plot of prevalence of deep vein thrombosis across years of study publication; Figure S47: Line graph of prevalence of deep vein thrombosis by post-stroke screening window.

Author Contributions: S.M.M.B. is the Principal Investigator of the IRIS-DVT Study, conceptualized it, developed the overarching framework and supervised the Global Health Neurology Lab team. He provided intellectual leadership, validated key concepts, and oversaw all aspects of study design and manuscript development. S.M.M.B. encouraged Y. Y. to explore this topic and guided the synthesis and interpretation of findings. Y.Y. and S.M.M.B. jointly conducted the literature review, data collection, drafting of the manuscript, and critical revisions. D.C. contributed to the data collection, validation, and discussion during the drafting and revision process. All authors have read and agreed to the published version of the manuscript.

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