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Original Articles

# A 10-Year Single-Center Experience in Pediatric Thromboembolism: Management Strategies and Clinical Outcomes

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**Competing interests:** The authors declare no competing interest.

**Abstract.** ***Background*: Pediatric thromboembolism is uncommon but clinically important, and its epidemiology varies according to age, underlying disease, catheter exposure, and referral patterns. This study evaluated the epidemiology, risk factors, management, and outcomes of pediatric thromboembolism over a 10-year period in a tertiary care center.**
***Methods*: We retrospectively reviewed children aged 0-18 years with symptomatic, radiologically confirmed thromboembolism between January 2010 and January 2020. Incidentally detected asymptomatic thromboses and cases without imaging confirmation were excluded. Venous and arterial events were analyzed separately.**
***Results*: Among 51,025 pediatric admissions, 70 patients had thromboembolism, corresponding to an admission-based rate of 13.7 per 10,000 admissions. Venous thromboembolism accounted for 51 cases (72.9%) and arterial thromboembolism for 19 cases (27.1%). Median age at diagnosis was 6 years (range, 1 day-18 years), and 49 patients (70.0%) were male. The central nervous system was the most frequent anatomical region involved. Central venous catheterization was the leading modifiable acquired risk factor. Active antithrombotic or interventional treatment was used in 68 patients (97.1%); low molecular weight heparin containing regimens were used in 46 patients (65.7%). Complete recovery was documented in 55 patients (78.6%), recurrence in 12 (17.1%), and mortality in 3 (4.3%). Recurrence was more frequent after venous than arterial events (23.5% vs 0%; p = .028).**
***Conclusion*: In this 10-year cohort, venous events predominated and catheter related thrombosis remained a major preventable contributor. Recurrence was confined to venous events, supporting careful long term follow up and individualized secondary prevention for high risk children. Standardized catheter care practices and selective, appropriately timed thrombophilia testing are practical priorities.**

**Keywords:** Pediatric thrombosis; Venous thromboembolism; Arterial thrombosis; Central venous catheter; Low molecular weight heparin.

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**Introduction.** Pediatric thromboembolism (TE), encompassing venous thromboembolism (VTE) and arterial thromboembolism (ATE), is rare compared with adult thrombotic disease but remains an important cause

of morbidity and mortality in children.[1-3] Increasing use of advanced imaging, improved survival of children with complex chronic disease, wider use of central venous catheters (CVCs), and heightened clinical awareness have increased recognition of pediatric TE in recent decades.[1,4]

Reported pediatric VTE rates vary substantially by study design, population, and denominator. Population-based rates are much lower than admission-based rates, whereas tertiary hospital cohorts are enriched for neonates, children with malignancy, congenital heart disease, infection, surgery, and intensive care exposure.[2,4-6] Neonates and adolescents are consistently recognized as higher-risk age groups, reflecting developmental hemostasis, catheter exposure, infection, dehydration, hormonal factors, and age-specific comorbidities.[4,6]

Risk factors for pediatric TE include CVCs, sepsis, immobilization, major surgery, nephrotic syndrome, malignancy, congenital heart disease, antiphospholipid antibodies, and inherited thrombophilia. In neonates, prematurity, infection, hypovolemia, perinatal asphyxia, and vascular catheterization are particularly relevant.[5-9] Interpretation of thrombophilia testing in children requires age-specific reference ranges and careful attention to acquired confounders such as vitamin K deficiency, liver dysfunction, nephrotic syndrome, acute thrombosis, and anticoagulant exposure.[8-11]

Evidence guiding pediatric antithrombotic treatment remains limited, and many recommendations are extrapolated from adult practice or based on pediatric observational cohorts. Unfractionated heparin, LMWH, and vitamin K antagonists have historically been the main treatment options, although pediatric practice continues to evolve.[12,13] International registries and collaborative cohorts have therefore been important for defining epidemiology, management, and outcomes.[14,15]

Most pediatric studies have focused primarily on VTE, whereas ATE is less frequently described and often has different mechanisms, risk factors, and clinical consequences. We therefore conducted a 10-year single-center retrospective analysis of both venous and arterial thrombotic events in children. The objectives were to describe admission-based frequency, age distribution, risk factors, clinical management, outcomes, and descriptive temporal patterns in a tertiary pediatric hematology center in Turkey.

## Methods

*Study population.* We retrospectively reviewed medical records of 51,025 patients aged 0-18 years admitted to the Department of Pediatrics, Atatürk University Hospital, between January 2010 and January 2020. We included 70 patients with clinically symptomatic and radiologically confirmed TE. Thrombosis was classified as community-acquired if present at admission and hospital-acquired if it developed during hospitalization.

Patients were stratified into five age groups reflecting pediatric hematology practice: neonates (0-28 days), infants (1-12 months), preschool children (1-6 years), school-aged children (6-12 years), and adolescents (12-18 years).

*Definitions and diagnostic confirmation.* TE was classified as VTE or ATE according to the affected vascular territory. The anatomical site was determined from imaging reports and clinical records. Morbidity was defined as persistent neurological deficit, seizures, hemiparesis, post-thrombotic syndrome, or other long-term sequelae documented at the final follow-up visit. Recurrence was defined as radiologically confirmed thrombosis at a new vascular site or re-thrombosis at a previously affected site after completion of initial therapy. Mortality was defined as death directly attributable to TE or TE-related complications.

Thrombosis was confirmed using location-specific imaging, including Doppler ultrasonography for extremity and abdominal vessels, echocardiography for intracardiac thrombosis, magnetic resonance angiography or venography for cerebral thrombosis, and computed tomography angiography when clinically indicated. Cases without imaging confirmation were excluded.

*Risk factors and thrombophilia assessment.* Risk factors were extracted from admission notes, discharge summaries, imaging reports, laboratory records, and consultation notes. Multiple risk factors were recorded when present. Acquired risk factors included CVCs, arterial catheterization, systemic infection or sepsis, malignancy, major surgery, trauma, immobilization, nephrotic syndrome, congenital heart disease, and prematurity.

Thrombophilia testing was selective rather than universal. It was considered for patients without an obvious acquired trigger and for selected patients with recurrent thrombosis. Established thrombophilia assays included factor V Leiden G1691A, prothrombin G20210A, protein C activity, protein S activity, antithrombin activity, and lupus anticoagulant when clinically indicated. A plasminogen activator inhibitor-1 (PAI-1) variant, historically included in the local testing panel, was reported separately and was not interpreted as an established thrombophilia marker. **Table 1** reports the number tested for each assay and uses the number tested as the denominator. Natural anticoagulant results were interpreted with age-specific reference ranges and in relation to acute thrombosis, illness, and anticoagulant exposure.

Available records documented lupus anticoagulant testing in 64 patients, factor V Leiden testing in 53, and

**Table 1.** Thrombophilic or prothrombotic conditions associated with pediatric thromboembolism.

| | | | |
|---|---|---|---|
| Lupus anticoagulant | 64/70 | 10 (15.6%) | Tested at diagnosis. Repeat confirmation data were unavailable in the retrospective records. |
| Factor V Leiden G1691A | 53/70 | 1 (1.9%), homozygous | Confirmed from the genetic test report. Family studies were unavailable because of the retrospective design. |
| Prothrombin G20210A | 53/70 | 2 (3.8%): heterozygous 1; homozygous 1 | Confirmed from the genetic test reports. Family studies were unavailable because of the retrospective design. |
| PAI-1 variant (descriptive only) | 64/70 | 11 (17.2%) | Historically included in the local panel; reported descriptively because its clinical relevance is limited and it is not considered an established thrombophilia marker. |
| Protein S activity | 64/70 | 2 low results (3.1%) | Obtained at diagnosis before anticoagulant treatment; both low results remained low when repeated after the acute phase. |
| Protein C activity | 64/70 | 1 low result (1.6%) | Obtained at diagnosis before anticoagulant treatment; the low result remained low when repeated after the acute phase. |
| Antithrombin activity | 64/70 | 1 low result (1.6%) | Obtained at diagnosis before anticoagulant treatment; the low result remained low when repeated after the acute phase. |

Testing was selective, and percentages use the number tested as the denominator. Lupus anticoagulant results indicate positivity at diagnosis only and do not establish persistent antiphospholipid antibodies or antiphospholipid syndrome because repeat confirmation was unavailable. Natural anticoagulant assays were obtained before anticoagulant therapy; all initially low protein C, protein S, and antithrombin results remained low on repeat testing after the acute phase. Family studies were unavailable because of the retrospective design. The PAI-1 variant is reported descriptively and is not presented as equivalent to established thrombophilia markers.

prothrombin G20210A testing in 53. PAI-1 variant testing, protein C, protein S, and antithrombin were each performed in 64 patients. Lupus anticoagulant results represent positivity at diagnosis only because repeat confirmation was unavailable; we did not interpret them as persistent antiphospholipid antibodies or antiphospholipid syndrome. Natural anticoagulant assays were obtained at diagnosis before the initiation of anticoagulant therapy. All four abnormal natural anticoagulant results-two low protein S levels, one low protein C level, and one low antithrombin level-were reassessed after the acute phase and remained low on repeat testing.

*Treatment and outcomes.* Treatment regimens, treatment duration, complications, recurrence, morbidity, mortality, and follow-up duration were recorded. Follow-up duration was calculated from the index thromboembolic diagnosis to the last documented clinical assessment. Follow-up duration was available for 68 of 70 patients. The median follow-up duration was 12 months (IQR, 6–60 months; range, 25 days–144 months), and 37 of 68 patients (54.4%) had at least 12 months of follow-up. During the study period, direct oral anticoagulants were not routinely used for pediatric thrombosis management in our institution; therefore, no patient in this cohort received a DOAC.

*Ethics.* The study was approved by the Atatürk University Faculty of Medicine Clinical Research Ethics Committee (Decision No: B.30.2.ATA.0.01.00/470). Written informed consent was obtained from parents of all participants.

*Statistical analysis.* Data were summarized using IBM SPSS version 23. Exploratory exact comparisons were calculated in Python 3.12.13. Two-by-two tests used SciPy 1.17.0, and the 2 x 5 Fisher-Freeman-Halton test was calculated by exact fixed margin enumeration. Categorical variables were reported as frequencies and percentages, and continuous variables as medians and ranges. Sex and age-group distributions were compared between VTE and ATE. Neonates were also compared with older children, and recurrence was compared by thrombosis type. No adjustment was made for multiple comparisons, and multivariable modeling was not performed because of the small sample. A p value $< 0.05$ was considered statistically significant. The admission-based TE rate was calculated per 10,000 pediatric admissions. Annual variation was described using case counts because annual admission denominators were unavailable; the 2020 point represented January 2020 only, and no incidence trend test was performed. Multiple-response analysis was used when patients had more than one risk factor.

## Results

*Patient characteristics.* Among 51,025 pediatric admissions over the 10 years, 70 patients were diagnosed with symptomatic, radiologically confirmed TE, corresponding to an admission-based rate of 13.7 per 10,000 admissions. Thrombosis was community-acquired in 35 cases (50.0%) and hospital-acquired in 35 cases (50.0%). Median age at diagnosis was 6 years (range, 1 day- 18 years). Forty-nine patients were male (70.0%), and 21 were female (30.0%). Age group distribution was as follows: neonates, 18 (25.7%); infants, 5 (7.1%); preschool children, 12 (17.1%); school-aged children, 15 (21.4%); and adolescents, 20 (28.6%) (**Figure 1**, **Figure 2**).

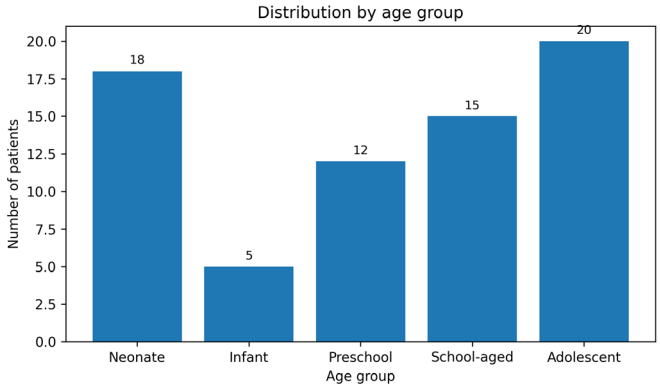



**Figure 1.** Distribution of patients by age group.

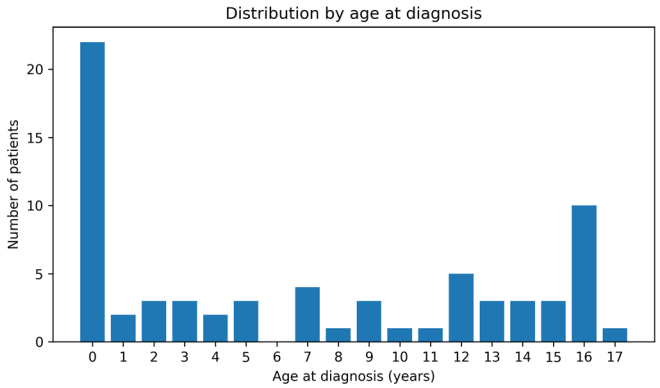



**Figure 2.** Distribution of patients by age at diagnosis.

*Thrombosis type and location.* VTE accounted for 51 cases (72.9%), whereas ATE accounted for 19 cases (27.1%). Because venous and arterial thromboses differ in pathophysiology, triggers, and management, they were analyzed separately when appropriate. Intracranial thrombosis was the most frequent anatomical category overall, followed by intracardiac, gastrointestinal or portal venous system, and lower extremity thrombosis (**Table 2**). In arterial events, the middle cerebral artery was the most frequently involved vessel. Intracardiac thrombosis was observed predominantly in neonates.

Exploratory comparisons are presented in **Table 3**. Sex distribution did not differ between ATE and VTE (male sex, 63.2% vs 72.5%; p = 0.559). The overall age-group distribution also did not differ significantly (p = 0.086). Neonates represented 42.1% of ATE and 19.6% of VTE; when comparing neonates with older children, p = 0.070. Recurrence occurred in 12 of 51 patients with VTE (23.5%) and none of the 19 patients with ATE (p = 0.028).

The prominence of central nervous system thrombosis in this cohort differs from many catheter-focused registries, in which extremity or superior vena cava thrombosis predominates. This distribution likely reflects the institution's referral profile, availability of neuroimaging, and regional clinical factors that may increase detection of cerebral thrombosis.

*Risk factors.* Most patients had at least one acquired or inherited risk factor. The most frequently recorded acquired factors were central venous catheterization, major surgery, prematurity, severe infection or sepsis, and malignancy. ATE was more common among neonates and was frequently associated with arterial catheterization or congenital heart disease. VTE predominated in older children and was associated with CVC exposure, chronic disease, malignancy, and selected thrombophilia markers.

Thrombophilia testing was selective rather than universal. Lupus anticoagulant was positive at diagnosis in 10 of 64 tested patients (15.6%); repeat confirmation was unavailable, so this finding does not establish persistent antiphospholipid antibodies or antiphospholipid syndrome. Factor V Leiden G1691A was tested in 53 patients and was detected in one patient (1.9%) in the homozygous state. Prothrombin G20210A was tested in 53 patients and was detected in two patients (3.8%), one heterozygous and one homozygous. The PAI-1 variant was identified in 11 of 64 tested patients (17.2%) but was treated as a descriptive, non-established marker rather than a principal thrombophilia finding. Protein C, protein S, and antithrombin were each assessed in 64 patients. Genetic tests and natural anticoagulant assays were obtained at diagnosis before the initiation of anticoagulant therapy. All abnormal natural anticoagulant results-two low protein S levels, one low protein C level, and one low antithrombin level-were reassessed after the acute phase and remained low on repeat testing.

*Treatment and outcomes.* Active antithrombotic or interventional treatment was used in 68 patients (97.1%), whereas two patients received supportive care because of contraindications. LMWH-containing regimens were used in 46 patients (65.7%), including LMWH monotherapy in 38 (54.3%). Oral anticoagulant monotherapy was used in 19 patients (27.1%). Recombinant tissue plasminogen activator was used in 6 patients (8.6%), either alone or with LMWH. One patient underwent thrombectomy in addition to anticoagulation (**Table 4**).

At the final recorded assessment, patients were assigned to mutually exclusive outcome categories: complete recovery in 55 of 70 patients (78.6%), recurrent thrombosis in 12 (17.1%), and TE-related mortality in 3 (4.3%). Patients with any documented recurrent thrombosis were classified in the recurrence category regardless of subsequent recovery; patients who died of TE or a TE-related complication were classified in the mortality category; and the remaining patients with complete clinical and radiological resolution and no recurrence were classified as complete recovery. Follow-up duration was available for 68 of 70 patients. The median follow-up duration was 12 months (IQR, 6–60

**Table 2.** Distribution of patients by anatomical location of thrombosis.

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Neonate | 3 | 8 | 5 | 1 | 0 | 1 | 18 |
| Infant | 4 | 0 | 1 | 0 | 0 | 0 | 5 |
| Preschool | 6 | 2 | 2 | 0 | 2 | 0 | 12 |
| School-aged | 6 | 2 | 0 | 0 | 6 | 1 | 15 |
| Adolescent | 9 | 1 | 2 | 2 | 5 | 1 | 20 |
| Total | 28 | 13 | 10 | 3 | 13 | 3 | 70 |

**Table 3.** Characteristics of patients with arterial and venous thrombosis.

| | | | | |
|---|---|---|---|---|
| Sex | Male | 12 (63.2) | 37 (72.5) | 0.559 |
| | Female | 7 (36.8) | 14 (27.5) | |
| Age group | Neonate | 8 (42.1) | 10 (19.6) | 0.086 |
| | Infant | 1 (5.3) | 4 (7.8) | |
| | Preschool | 5 (26.3) | 7 (13.7) | |
| | School-aged | 1 (5.3) | 14 (27.5) | |
| | Adolescent | 4 (21.1) | 16 (31.4) | |
| Recurrence | Yes | 0 (0.0) | 12 (23.5) | 0.028 |
| | No | 19 (100.0) | 39 (76.5) | |

P values are two-sided exact tests. The age group comparison used the Fisher-Freeman-Halton exact test. Analyses were exploratory and unadjusted. ATE, arterial thromboembolism; VTE, venous thromboembolism.

**Table 4.** Treatment of pediatric thromboembolism.

| | | |
|---|---|---|
| Oral anticoagulant (warfarin) | 19 | 27.1 |
| LMWH | 38 | 54.3 |
| r-TPA and LMWH | 4 | 5.7 |
| r-TPA | 2 | 2.9 |
| ASA and LMWH | 2 | 2.9 |
| ASA | 1 | 1.4 |
| Supportive care owing to contraindication | 2 | 2.9 |
| Surgery, LMWH, and oral anticoagulant | 1 | 1.4 |
| ASA, LMWH, and oral anticoagulant | 1 | 1.4 |
| Total | 70 | 100.0 |

Active antithrombotic or interventional treatment was used in 68 patients (97.1%). LMWH-containing regimens were used in 46 patients (65.7%). ASA, acetylsalicylic acid; LMWH, low molecular weight heparin; r-TPA, recombinant tissue plasminogen activator.

months; range, 25 days–12 years), and 37 of 68 patients (54.4%) with available follow-up were followed for at least 12 months. All 12 recurrent events were venous and radiologically confirmed. Nine of the 12 patients with recurrence had at least one identifiable risk factor. No acquired trigger was identified in three recurrent cases, suggesting an unrecognized hereditary predisposition or incompletely captured acquired risk factors.

*Temporal distribution.* The number of TE cases varied across the study period, with the lowest count in 2015 (n = 1) and the highest count in 2020 (n = 13). Because annual admission denominators were unavailable and the total number of events was small, these data should be interpreted as descriptive case-count variation rather than conclusive evidence of an increasing incidence trend.

**Discussion.** This 10-year single-center study describes venous and arterial thromboembolism in 70 children and provides admission-based data from a tertiary pediatric hematology center in Turkey. Venous events predominated, catheter-related and other acquired triggers were important, and central nervous system involvement was frequent. In exploratory analysis, recurrence was more common after VTE than ATE

(23.5% vs 0%; p = 0.028), whereas sex and overall age-group distributions did not differ significantly.

The equal distribution of community-acquired and hospital-acquired cases is clinically relevant. Community-acquired thrombosis was often associated with chronic underlying disease or thrombophilia evaluation. In contrast, hospital-acquired thrombosis was more frequently related to catheter exposure, intensive care, surgery, infection, or other inpatient factors. This distinction matters because prevention strategies differ; community-acquired events require recognition of patient-specific risk factors, whereas hospital-acquired events require structured catheter and inpatient thrombosis prevention practices.[14,15]

Neonates and adolescents represented prominent age groups in this cohort, consistent with previous pediatric studies.[4,6,16] Neonates are vulnerable because of developmental hemostasis, prematurity, infection, hypoxia, dehydration, and vascular catheterization. Adolescents may be exposed to additional acquired risk factors, including immobility, obesity, hormonal influences, chronic inflammatory disease, and malignancy related therapy. These age-specific mechanisms support the need for risk assessment tools tailored to developmental stage rather than extrapolated directly from adult models.

A notable finding was the high frequency of intracranial thrombosis. In many large pediatric VTE series, extremity- or catheter-related thrombosis is most common; however, institutional referral patterns can substantially influence the distribution of thrombotic sites.[14,15,17] Our hospital functions as a regional referral center with access to advanced neuroimaging, which may have increased detection of cerebral venous sinus thrombosis and arterial stroke. Regional dehydration, infection, and delayed presentation may also contribute, although these hypotheses cannot be confirmed from the retrospective dataset.

CVCs remained the leading modifiable risk factor, in agreement with international pediatric data.[14,15,18] The relative frequency of catheter-related thrombosis in our cohort appeared lower than in some North American and Canadian series. Direct comparison is limited because cohorts differ in case ascertainment, definitions, patient mix, catheter type, and denominators. Prevention should emphasize appropriate catheter size and site, aseptic insertion and maintenance, daily review of line necessity, timely removal, and surveillance of high risk children.

Inherited thrombophilia was identified in a subset of tested patients, but testing was not universal. Factor V Leiden and natural anticoagulant deficiencies are among the inherited factors reported in pediatric thrombosis.[8,11,19,20-23] Because protein C, protein S, and antithrombin levels may be affected by acute thrombosis and anticoagulant treatment, abnormal results require confirmation after the acute phase. In our cohort, initially low protein S in two patients, protein C in one patient, and antithrombin in one patient remained low on repeat testing after the acute phase, supporting persistent deficiencies. Genetic testing confirmed homozygous factor V Leiden in one patient and prothrombin G20210A variants in two patients, one heterozygous and one homozygous. However, family studies were not available because of the retrospective design; therefore, these uncommon homozygous findings should be interpreted with particular caution and should not be used to infer prevalence. Lupus anticoagulant positivity was recorded only at diagnosis and, without repeat confirmation, does not establish persistent antiphospholipid antibodies or antiphospholipid syndrome. The PAI-1 variant was retained only as a descriptive historical laboratory finding because its clinical relevance is limited and it is not considered equivalent to established thrombophilia markers.

Treatment patterns reflected pediatric practice during the study period. LMWH was the most frequently used agent, consistent with its predictable pharmacokinetics, weight-based dosing, and lower monitoring burden compared with vitamin K antagonists.[12,13] Thrombolysis was reserved for selected severe cases, and thrombectomy was rare. No patient received a DOAC, which reflects institutional practice during the January 2010-January 2020 study period rather than a conclusion about contemporary pediatric anticoagulation options.[24-28]

Clinical outcomes were favorable overall, with complete recovery in 79% and mortality in 4%. Recurrence occurred in 17% and was confined to venous events. This recurrence rate is clinically important and aligns with reports emphasizing the morbidity of recurrent pediatric VTE.[20,29,30] Children with persistent risk factors, recurrent catheter exposure, chronic inflammatory disease, malignancy, nephrotic syndrome, or unprovoked thrombosis may require individualized follow-up and consideration of secondary prophylaxis.

A subset of patients had malignancy. Thrombosis in pediatric cancer is multifactorial and may be related to CVCs, disease biology, infection, corticosteroids, and asparaginase exposure.[12,28] In our cohort, thrombosis among children with acute lymphoblastic leukemia occurred close to L-asparaginase administration, supporting the need for close clinical vigilance during high risk treatment windows. The small number of malignancy-associated events, however, precludes firm conclusions regarding cancer-specific risk.

*Limitations.* This study has several limitations. Its retrospective design may have led to incomplete capture of risk factors, laboratory timing, treatment duration, follow-up, and long-term outcomes. Thrombophilia testing was selective, family studies were unavailable, and lupus anticoagulant positivity at diagnosis could not

be confirmed on repeat testing; therefore, the data cannot establish persistent antiphospholipid antibodies or antiphospholipid syndrome and should not be used to estimate thrombophilia prevalence. The single tertiary-center setting may have enriched the cohort for complex or neurologic cases, limiting generalizability. The denominator was hospital admissions rather than the general pediatric population; the reported value is therefore an admission-based frequency, not a population incidence. Finally, the exploratory comparisons were unadjusted and underpowered, and the study was not designed to compare treatment regimens or test temporal incidence trends.

**Conclusions.** In this 10-year single-center cohort, pediatric thromboembolism occurred at an admission-based rate of 13.7 per 10,000 admissions. Venous events predominated, while arterial events were most frequent in neonates and were often associated with catheterization or congenital heart disease. The central nervous system was the most frequently involved anatomical region, likely reflecting the institution's referral and imaging profile.

The findings support three practical priorities. First, pediatric services should standardize CVC insertion and maintenance, review catheter necessity daily, and remove lines promptly when no longer required. Second, thrombophilia testing should be selective, particularly for unprovoked or recurrent events or a strong family history; natural anticoagulant abnormalities should be interpreted with age-specific ranges and confirmed after acute confounders resolve. Third, prolonged follow-up should be considered for children with recurrent or unprovoked VTE, persistent risk factors, repeated catheter exposure, or thrombotic sequelae. Prospective multicenter studies are needed to refine pediatric-specific risk stratification, prophylaxis, and treatment strategies.

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**Data availability statement.** The data that support the findings of this study are available from the corresponding author upon reasonable request and subject to institutional ethics requirements.

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