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Etiological spectrum and imaging findings of non-traumatic intracranial hemorrhage in children

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ABSTRACT

Aims: Pediatric non-traumatic intracranial hemorrhage (pNTIH) is an important cause of acute neurological morbidity with an etiological spectrum distinct from that of adults. This study aimed to evaluate the imaging findings, etiological distribution, and age-related characteristics of pNTIH in children.

Methods: This retrospective, single-center study included 56 pediatric patients aged 0-18 years who were diagnosed with pNTIH between September 2022 and January 2026. Radiological evaluation included computed tomography (CT), magnetic resonance imaging (MRI), and advanced vascular imaging modalities, including CT angiography (CTA), MR angiography (MRA), and digital subtraction angiography (DSA). Etiologies were determined using a combination of clinical, laboratory, and imaging findings.

Results: Arteriovenous malformation (AVM) was the most common etiology (23.2%), followed by cerebral aneurysm (14.3%) and venous ischemia-related hemorrhage (12.5%). AVM, venous ischemia, and cavernoma-related hemorrhages were more common in older children, whereas treatment-related hemorrhage was more frequent in infants. Increasing age was associated with a higher likelihood of AVM-related hemorrhage (OR=1.14; 95% CI, 1.01-1.29; p=0.032). CTA was performed in 71.4% of patients, MRA in 53.6%, and DSA in 26.8%. Early mortality occurred in 3 patients (5.4%).

Conclusion: Non-traumatic intracranial hemorrhage in children demonstrates age-dependent etiological variation. Early multimodality imaging is essential for accurate etiological diagnosis and timely management.

Keywords: Pediatric intracranial hemorrhage, non-traumatic intracranial hemorrhage, computed tomography, magnetic resonance imaging

INTRODUCTION

Non-traumatic intracranial hemorrhage (pNTIH) in childhood refers to intraparenchymal, intraventricular, or extra-axial bleeding occurring between birth and 18 years of age in the absence of trauma.[1] Although ischemic stroke predominates in adults, hemorrhagic stroke accounts for nearly half of all pediatric stroke cases and remains a major cause of acute neurological morbidity and mortality in children.[2]

The terms hemorrhagic stroke and non-traumatic intracranial hemorrhage are frequently used interchangeably; however, they are not synonymous. According to the American Heart Association, a hemorrhagic stroke describes acute focal neurological dysfunction attributable to non-traumatic intracranial bleeding.[3] In contrast, the World Health Organization defines stroke as a rapidly developing clinical syndrome of vascular origin lasting more than 24 hours or resulting in death.[4] In this context, hemorrhagic stroke reflects the clinical syndrome, while pNTIH denotes the underlying radiological and pathological process.

The epidemiology and etiological spectrum of intracranial hemorrhage differ substantially between children and adults. In adults, hypertensive vasculopathy and cerebral amyloid angiopathy are leading causes of spontaneous intracerebral hemorrhage, whereas in children, vascular malformations, hematologic disorders, congenital heart disease, and systemic coagulopathies are more frequently implicated.[5-6] Although trauma remains the most common overall cause of intracerebral hemorrhage in childhood, distinguishing traumatic from non-traumatic hemorrhage may be challenging, particularly in infants and young children in whom minor head injuries are common and clinical history may be unreliable.[7]

Clinical presentation is primarily determined by hemorrhage volume, localization, and the degree of intracranial pressure elevation.[8] Headache is the most frequently reported

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symptom in pediatric hemorrhagic stroke, observed in up to 80% of cases except in neonates, and is typically related to increased intracranial pressure.[9] However, in younger children, especially infants, symptom recognition may be limited by developmental and communication constraints. Additional manifestations include focal or generalized seizures, focal neurological deficits, altered consciousness, nausea and vomiting, and location-specific findings such as visual disturbances or motor weakness.[10]

In clinical practice, the initial diagnostic impression in pediatric patients presenting with acute neurological symptoms frequently fails to predict the underlying etiology of non-traumatic intracranial hemorrhage.[11]

Multimodality imaging-including computed tomography (CT), magnetic resonance imaging (MRI), and vascular imaging techniques such as CT angiography (CTA) or MR angiography (MRA) plays a pivotal role not only in confirming the presence of hemorrhage but also in identifying age-specific etiologies such as vascular malformations, cerebral venous thrombosis, or hematologic disorders. Furthermore, the etiological profile of pNTIH varies across pediatric age groups, which may directly influence the selection and diagnostic yield of imaging modalities in the acute setting.[12] Despite this, standardized imaging pathways and age-stratified diagnostic performance of multimodal imaging approaches in pediatric pNTIH remain insufficiently characterized in the existing literature.

Therefore, this study aimed to evaluate pediatric patients presenting with non-traumatic intracranial hemorrhage with respect to initial clinical suspicion, radiological diagnostic approach, age-based etiological distribution, and treatment course, and to contribute to the literature by providing a comprehensive clinicoradiological analysis from a tertiary referral center.

METHODS

Ethics

Ethical approval for this study was obtained from the Scientific Researches Evaluation and Ethics Committee No. 2 of Ankara Etlik City Hospital (Date: 03.03.2026, Decision No: AEŞH-BADEK2-2026-232). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

This was a single-center retrospective observational cohort study conducted at a tertiary pediatric referral hospital. Cranial imaging studies of pediatric patients who presented to the emergency department between September 2022 and January 2026 with non-traumatic complaints, including headache and suspected acute intracranial pressure elevation, were reviewed.

Eligible patients were identified through a search of the institutional picture archiving and communication system (PACS) and electronic medical records using imaging reports containing the terms "intracranial hemorrhage", "intraparenchymal hemorrhage", "intraventricular hemorrhage", or "extra-axial hemorrhage". Among these patients, 56 cases with radiologically confirmed intracranial hemorrhage were identified and included in the analysis.

Intracranial hemorrhage was defined as the presence of intraparenchymal, intraventricular, subarachnoid, or subdural hyperdensity on CT or corresponding signal characteristics on MRI sequences, including T1-weighted, T2-weighted, susceptibility-weighted imaging (SWI), or gradient-echo sequences.

The study population comprised children aged 0-18 years. Patients were stratified into four developmental age groups: 0-2 years (infancy), 2-6 years (early childhood), 6-12 years (middle childhood), and 12-18 years (adolescence).

Imaging Protocol and Radiological Evaluation

The initial imaging modality was non-contrast cranial CT in 50 patients and diffusion-weighted MRI in 6 patients. However, cranial CT performed within the first 24 hours of presentation was available for all patients, and the primary radiological evaluation was based on these CT examinations.

All imaging studies were retrospectively reviewed by two pediatric radiologists with 15 and 20 years of experience who were blinded to the final etiological diagnosis. In cases of disagreement, a consensus decision was reached.

When hemorrhage localization was analyzed, it was noted that a subset of patients exhibited bleeding involving more than one anatomical compartment, such as concomitant intraventricular and parenchymal hemorrhage. For systematic evaluation, hemorrhage sites were categorized into the following anatomical regions: intracerebral and cerebellar parenchyma, subarachnoid space, subdural space, subpial region, and the intraventricular system.

When available, follow-up imaging and vascular studies-including CTA, brain MRI, MRA, or digital subtraction angiography (DSA)-were evaluated to determine the underlying etiology. Arteriovenous malformations were graded using the Spetzler-Martin grading system,[13] and cavernous malformations were classified according to the Zabramski classification.[14] In patients with aneurysmal subarachnoid hemorrhage, hemorrhage severity was assessed using the Fisher classification.[15]

In patients presenting with hemorrhage, clinical preliminary diagnosis, presenting symptoms, demographic data, and bleeding etiologies were evaluated. The etiological factors were classified based on clinical findings, laboratory results, neuroimaging features, and review of medical records as follows: (1) intracranial tumor- or mass-related hemorrhage, (2) arteriovenous malformation (AVM), (3) arteriovenous fistula (AVF), (4) cavernoma, (5) cerebral aneurysm, (6) venous-origin hemorrhage (associated with cerebral venous ischemia and/or venous congestion), (7) infectious or inflammatory causes (including central nervous system infections, brain abscess, and acute hemorrhagic encephalomyelitis (AHEM), (8) hematological causes (including hematological malignancies, hemophagocytic lymphohistiocytosis (HLH), bleeding diathesis, and vitamin K deficiency), (9) treatment-related hemorrhage (occurring in the postoperative period or

following extracorporeal membrane oxygenation (ECMO)), and (10) unknown etiology.

Patients with a history of traumatic injury, prematurity-associated germinal matrix hemorrhage, or hemorrhage directly related to cranial surgical procedures or intracranial interventions were excluded from the study. Excluded postoperative cranial hemorrhages included subdural hematomas and hemorrhages localized to the neurosurgical operative site. In contrast, intracranial hemorrhages developing after extracranial cardiovascular surgery or during ECMO support were included, as these cases were considered systemic treatment-related non-traumatic hemorrhages rather than direct postoperative cranial surgical complications.

Short-term clinical outcomes at 1-month follow-up and administered treatment modalities were recorded from the electronic medical records.

Statistical Analysis

The data were performed using SPSS software (Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation or median (range), and categorical variables as frequencies and percentages.

The distribution of age across etiological groups was assessed using the Kruskal-Wallis test. Associations between categorical variables were analyzed using the Chi-square test or Fisher's exact test as appropriate.

Univariable logistic regression analysis was performed to determine the relationship between bleeding etiology and age. 95% confidence intervals (CI) and odds ratios (OR) were calculated. A p-value<0.05 was considered statistically significant. Informed consent was waived due to the retrospective design of the study.

RESULTS

A total of 56 pediatric patients (aged 0-18 years) with non-traumatic intracranial hemorrhage were included in the analysis. The mean age was 8.9 years (range: 1 month-18 years). Nineteen patients were female, and 37 were male. Patients older than 6 years constituted the majority of the cohort (Table 1).

Table 1. Distribution of pediatric patients with non-traumatic intracranial hemorrhage according to developmental age groups

Age group (years)	n	%
0-2 years (infancy)	7	12.5%
2-6 years (early childhood)	8	14.3%
6-12 years (middle childhood)	29	51.8%
12-18 years (adolescence)	12	21.4%
Total	56	100%

Etiological analysis was performed in all 56 patients. AVM was the most common cause of hemorrhage (n=13, 23.2%), with a mean age of 12.2 years (Figure 1). Cerebral aneurysm was identified in 8 patients (14.3%), with a mean age of 7.6 years. Venous-origin hemorrhage associated with cerebral venous ischemia was observed in 7 cases (12.5%) (Figure 2).

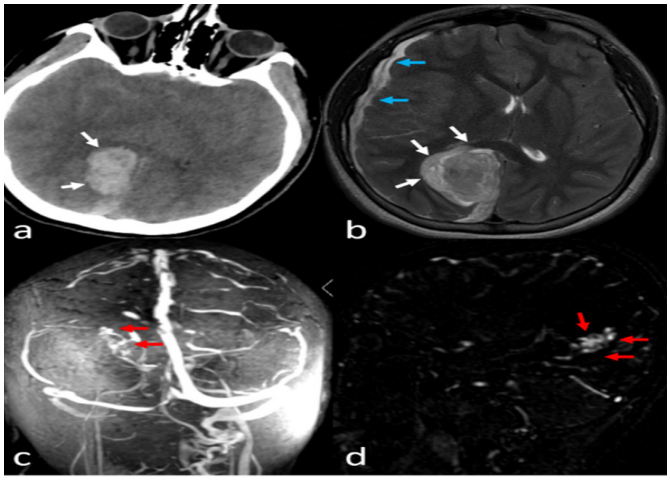


Figure 1. Occipital AVM

Axial CT (a) and T2-weighted MRI (b) images demonstrate intraparenchymal hemorrhage (white arrows) with extra-axial hemorrhage (blue arrows). MIP MRV (c) and MRA (d) images reveal an AVM nidus in the occipital region (red arrows), AVM: Arteriovenous malformation, CT: Computed tomography, MRI: Magnetic resonance imaging MIP: Maximum intensity projection, MRV: Magnetic resonance venography, MRA: Magnetic resonance angiography

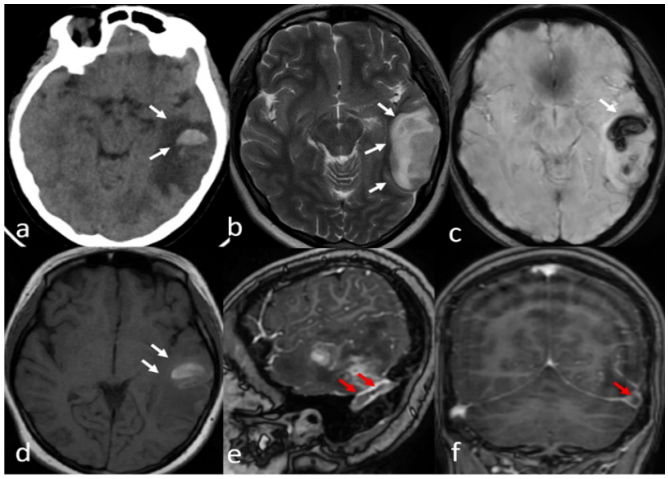


Figure 2. Hemorrhagic infarction associated with venous thrombosis

Axial CT (a), T2-weighted (b), SWI (c), and T1-weighted (d) images demonstrate hemorrhage (white arrows). Post-contrast sagittal (e) and coronal (f) T1-weighted images show a filling defect within the left transverse sinus (red arrows), CT: Computed tomography, SWI: Susceptibility-weighted imaging

Cavernous malformations were detected in 5 patients (8.9%), with a mean age of 11.2 years (Figure 3). Infectious or inflammatory causes were identified in 6 patients (10.7%) (Figure 4). Hematological causes and bleeding diatheses were also present in 6 patients (10.7%), who were younger (mean age 4.1 years) (Figure 5). Intracranial tumor- or mass-related hemorrhage was identified in 3 cases (5.4%).

AVF was observed in 2 patients (3.6%). However, due to the very limited sample size, no subgroup-specific statistical analysis was performed for this category. Treatment-related hemorrhage occurred in 3 cases (5.4%), all of whom had undergone cardiac surgery and ECMO support in the postoperative period. The etiology remained unknown in 3 patients (5.4%) (Table 2).

Advanced vascular imaging modalities were frequently utilized during etiological evaluation. CTA was performed in 40 patients (71.4%), MRA in 30 patients (53.6%), and

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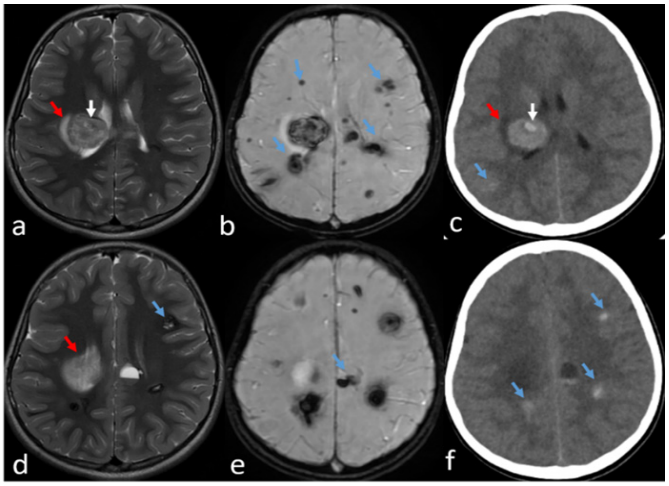


Figure 3. Multiple cavernomas

Axial T2-weighted (a, d), SWI (b, c), and CT (e, f) images demonstrate multiple cavernomas (blue arrows). The right thalamic cavernoma demonstrates acute hemorrhage (white arrows) with surrounding parenchymal edema (red arrows), SWI: Susceptibility-weighted imaging, CT: Computed tomography

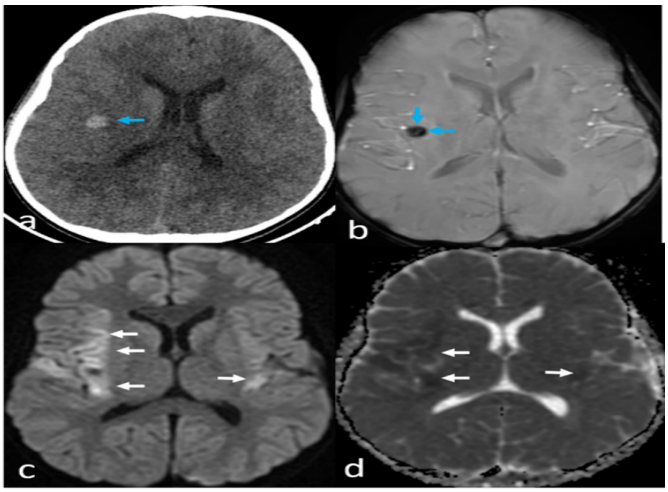


Figure 4. HSV encephalitis

Axial CT (a) and SWI (b) images demonstrate a small hemorrhagic focus in the right temporal lobe (blue arrows). Diffusion-weighted images (c) and ADC maps (d) show diffusion restriction in the bilateral temporoinsular regions (white arrows), HSV: Herpes simplex virus, CT: Computed tomography, SWI: Susceptibility-weighted imaging, ADC: Apparent diffusion coefficient

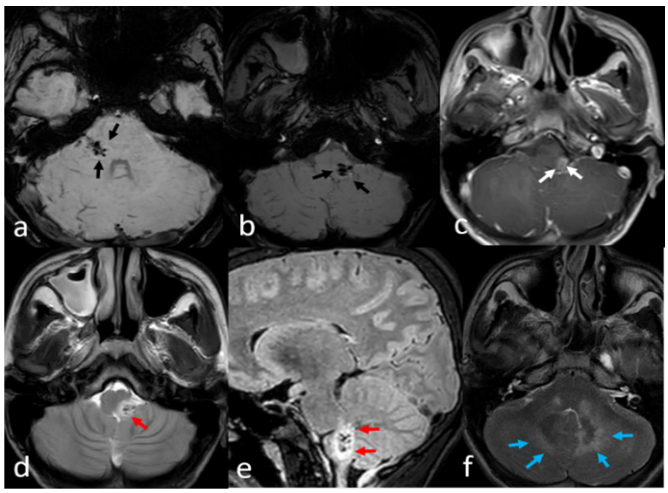


Figure 5. Primary central nervous system hemophagocytic lymphohistiocytosis (CNS-HLH)

Axial SWI images (a, b) demonstrate hemosiderin deposition (black arrows). Contrast-enhanced T1-weighted image (c) shows mild enhancement (white arrow). T2-weighted (d) and FLAIR (e) images demonstrate surrounding vasogenic edema (red arrows). Follow-up MRI obtained two months later shows symmetrical bilateral cerebellar white matter edema (blue arrows), consistent with CNS-HLH, CNS: Central nervous system, HLH: Hemophagocytic lymphohistiocytosis, SWI: Susceptibility-weighted imaging, FLAIR: Fluid-attenuated inversion recovery, MRI: Magnetic resonance imaging

Table 2. Etiological distribution and age characteristics of pediatric patients with non-traumatic intracranial hemorrhage

Etiological category	n	%	Age, mean±SD (months)
Arteriovenous malformation (AVM)	13	23.2	147±45
Cerebral aneurysm	8	14.3	91.8±60
Venous ischemia	7	12.5	125.3±74
Cavernoma	5	8.9	134.4±63
Infectious/inflammatory causes	6	10.7	112±57
Hematological causes	6	10.7	78.7±90
Intracranial tumor/mass-related hemorrhage	3	5.4	66±98
Unknown etiology	3	5.4	2±1
Arteriovenous fistula (AVF)	2	3.6	204*
Treatment-related hemorrhage	3	5.4	20.3±24

*Both patients with AVF were 17 years old, SD: Standard deviation, AVM: Arteriovenous malformation, AVF: Arteriovenous fistula

DSA in 15 patients (26.8%). These modalities contributed substantially to the detection and characterization of vascular lesions, particularly AVMs and aneurysms.

Age distributions showed variation among etiological groups (p=0.013). AVM-, venous ischemia-, and cavernoma-related hemorrhages were more frequently observed in older children and adolescents, whereas treatment-related hemorrhage was more commonly observed during infancy. The widest age distribution was observed in the hematologic causes group. Detailed age distributions across diagnostic categories are presented in **Figure 6**.

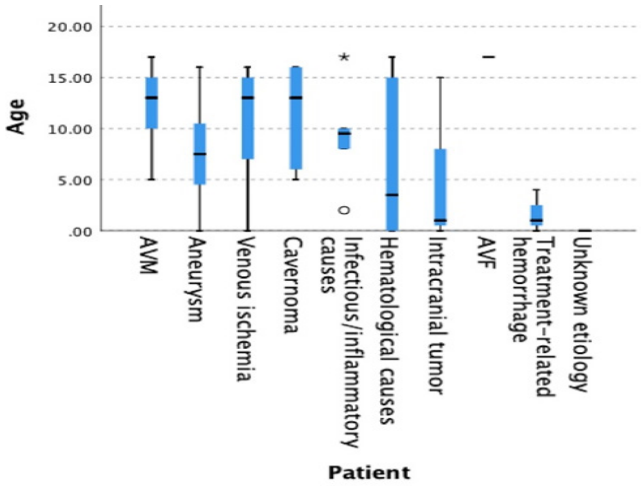


Figure 6. Age distribution across etiological categories of pediatric non-traumatic intracranial hemorrhage

AVM: Arteriovenous malformation, AVF: Arteriovenous fistula

Increasing age was associated with a higher likelihood of AVM-related hemorrhage (OR=1.14, 95% CI: 1.01-1.29, p=0.032). Treatment-related hemorrhage was more frequently observed in infancy; however, this finding should be interpreted with caution due to the limited number of cases.

Univariable logistic regression analysis demonstrated an association between increasing age and AVM-related bleeding. Increasing age was associated with a higher likelihood of AVM etiology (OR=1.14, 95% CI: 1.01-1.29, p=0.032). No statistically

significant association was identified between age and the remaining etiological categories (**Table 3**).

Table 3. Univariate logistic regression analysis to identify the determinants of AVM

Variable	OR (95% for AVM presence)	p-value
Age (years)	1.14 (1.01-1.29)	0.032

AVM: Arteriovenous malformation, OR: Odds ratio

AVMs were predominantly located in the supratentorial region (n=11), with cerebellar involvement in 2 patients. According to the Spetzler-Martin grading system, AVMs were classified as grade 2 in 5 patients, grade 3 in 6 patients, grade 4 in 1 patient, and grade 5 in 1 patient.

Among patients with aneurysmal hemorrhage (n=8), Fisher grade was 3 in 5 and 4 in 2; grading was not applicable in 1 patient.

According to the Zabramski classification, cavernous malformations (n=5) were classified as type II in four patients; two of these patients had multiple lesions.

During the 1-month follow-up period, three patients died in the early period, including two patients with AVM-related hemorrhage and one patient with an intraventricular hemorrhagic mass of undetermined etiology.

Treatment strategies varied according to the underlying etiology. Patients with vascular lesions underwent neurosurgical and/or endovascular treatment when indicated, whereas patients with hematological, infectious, and treatment-related hemorrhages were managed according to the underlying disease and supportive care protocols.

DISCUSSION

Understanding the etiology of non-traumatic intracranial hemorrhage in pediatric patients is essential not only for prompt diagnosis and early therapeutic intervention in the acute setting but also for guiding long-term management, predicting potential complications, and assessing recurrence risk.[9]

Clinical manifestations of intracranial hemorrhage may be subtle and nonspecific in pediatric patients, particularly in neonates and infants.[12] Open cranial sutures, fontanelles, and limited verbal expression may mask classical neurological findings and delay diagnosis.[16] Therefore, maintaining a high index of suspicion and performing early neuroimaging are critical when cerebrovascular pathology is suspected. Additionally, distinguishing spontaneous hemorrhage from traumatic bleeding can be challenging in young children due to the relatively high frequency of minor or unwitnessed trauma in this age group.[17]

Current pediatric stroke guidelines recommend brain MRI, including diffusion-weighted imaging, as the preferred first-line modality in children presenting with acute focal neurological deficits because of its superior sensitivity for early ischemic injury and lack of ionizing radiation exposure.[18] However, limited availability, longer acquisition time, and the potential need for sedation may restrict its immediate use in emergency settings. In contrast, CT remains a rapid and widely available diagnostic modality for the evaluation of suspected acute intracranial hemorrhage, particularly in unstable patients.[19] In our cohort, CT served as the initial imaging modality in the majority of patients, while advanced imaging techniques, including CTA, MRA, and digital subtraction angiography, enabled etiological clarification in selected cases.

More than 70% of patients in our study were older than 6 years, which is consistent with previous epidemiological studies demonstrating higher incidence rates of intracranial hemorrhage in school-age children and adolescents.[1-17] Notably, vascular etiologies were the most common cause of non-traumatic intracranial hemorrhage in our cohort, with arteriovenous malformations representing the leading etiology.[20,21] Although AVMs are congenital vascular anomalies, clinical presentation often occurs later in childhood, possibly due to progressive hemodynamic stress and delayed rupture risk.[22]

Univariable logistic regression analysis demonstrated an association between increasing age and a higher likelihood of AVM-related hemorrhage. Specifically, increasing age was associated with a higher likelihood of AVM etiology; however, this finding should be interpreted cautiously, given the limited sample size and uneven subgroup distribution. These findings suggest that the etiological profile of pediatric intracranial hemorrhage may vary across age groups and support consideration of vascular malformations in older children presenting with spontaneous hemorrhage. Treatment-related hemorrhage was more frequently observed in younger patients, particularly infants, although this observation should be interpreted cautiously because of the limited number of cases. This finding may reflect the increased vulnerability of critically ill pediatric patients requiring intensive care support or extracorporeal membrane oxygenation; however, larger studies are needed to further clarify this relationship.

From a practical imaging perspective, these findings may support the early incorporation of vascular imaging techniques such as CTA, MRA, or DSA in older children and adolescents presenting with non-traumatic intracranial hemorrhage, particularly when lobar or supratentorial hemorrhage patterns are identified on initial CT or MRI. Conversely, in infants and younger children, treatment-related, hematologic, and infectious etiologies should also be carefully considered during the diagnostic evaluation.

Our findings also highlight the important role of multimodality neurovascular imaging in the etiological evaluation of pediatric intracranial hemorrhage. Advanced vascular imaging techniques were valuable for detecting underlying vascular abnormalities and distinguishing vascular from nonvascular causes of hemorrhage, which may aid therapeutic planning. AVMs and aneurysms were predominantly located within the supratentorial compartment, whereas venous-origin hemorrhages were associated with extensive cerebral venous sinus thrombosis on follow-up vascular imaging. Cavernous malformations with active hemorrhage were most commonly classified as Zabramski type II lesions.

The absence of a statistically significant association between age and hematologic etiologies may indicate that coagulopathy-related hemorrhage can occur throughout the pediatric age spectrum, independent of developmental stage. In these patients, bleeding risk is likely influenced more by disease severity and treatment-related factors than by chronological age alone.

The overall early mortality rate in our cohort was relatively low (5.4%). However, interpretation of mortality-related findings is limited by the relatively small sample size and retrospective design. Timely diagnosis, rapid therapeutic intervention, and multidisciplinary management at a tertiary pediatric referral center may have contributed to favorable early outcomes. Overall, our findings emphasize the importance of age-oriented etiological evaluation and multimodal vascular imaging in children presenting with non-traumatic intracranial hemorrhage.

Limitations

This study has several limitations. First, its retrospective single-center design may limit the generalizability of the findings and introduce inherent selection bias. In addition, referral patterns at a tertiary pediatric referral center may have led to an overrepresentation of complex vascular and critically ill patients. Second, although this study represents one of the larger single-center pediatric non-traumatic intracranial hemorrhage (pNTIH) cohorts, the overall sample size and the relatively small number of patients in certain etiological subgroups limited the statistical power of subgroup analyses and precluded more detailed comparative analyses and multivariable regression modeling adjusted for potential confounding factors. Finally, long-term neurological, functional, and neurodevelopmental outcomes could not be systematically assessed because of incomplete follow-up data. Future multicenter prospective studies with larger patient populations and standardized longitudinal follow-up are needed to further validate these findings.

CONCLUSION

Non-traumatic intracranial hemorrhage in pediatric patients represents a heterogeneous clinical entity with a broad etiological spectrum that varies across developmental age groups. In our cohort, vascular abnormalities -particularly arteriovenous malformations- were identified as the most common underlying cause of hemorrhage, with a significantly higher prevalence observed in older children and adolescents. Multimodality imaging played a critical role in both confirming the presence of hemorrhage and identifying the underlying etiology. While CT served as the primary imaging modality in the acute setting, advanced vascular imaging techniques enabled etiological differentiation and informed subsequent management strategies.

These findings emphasize the importance of an age-oriented diagnostic approach supported by early neuroimaging in pediatric patients presenting with spontaneous intracranial hemorrhage. Recognition of age-dependent etiological patterns may facilitate timely diagnosis, guide imaging selection, and improve risk stratification in this vulnerable population.

ETHICAL DECLARATIONS

Ethics Committee Approval

Ethical approval for this study was obtained from the Scientific Researches Evaluation and Ethics Committee No. 2 of Ankara Etlik City Hospital (Date: 03.03.2026, Decision No: AEŞH-BADEK2-2026-232).

Informed Consent

As this was a retrospective study, formal written informed consent was not required and was therefore not obtained. This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: ŞY, BU; Design: ŞY; Control: ŞY, BU; Data Collection and/or Processing: ŞY, BU; Analysis and/or Interpretation: ŞY, BU, HU; Literature Review: ŞY, BU; Article Writing: ŞY, BU, HU; Critical Review: All Authors.

REFERENCES

1. Ciochon UM, Bindslev JBB, Hoei-Hansen CE, et al. Causes and risk factors of pediatric spontaneous intracranial hemorrhage-a systematic review. *Diagnostics (Basel).* 2022;12(6):1459. doi:10.3390/diagnostics12061459
2. AlZamer D, Al-Zamer YS, Al-Khafaji M, et al. Understanding pediatric stroke: etiology, diagnosis, and management in children under 15 years. *Cureus.* 2026;18(2):e103412. doi:10.7759/cureus.103412
3. Greenberg SM, Ziai WC, Cordonnier C, et al. 2022 guideline for the management of patients with spontaneous intracerebral hemorrhage: a guideline from the American Heart Association/American Stroke Association. *Stroke.* 2022;53(7):e282-e361. doi:10.1161/STR.0000000000000407
4. Feigin VL, Brainin M, Norrving B, et al. World Stroke Organization: global stroke fact sheet 2025. *Int J Stroke.* 2025;20(2):132-144. doi:10.1177/17474930241308142
5. Boulouis G, Stricker S, Benichi S, et al. Etiology of intracerebral hemorrhage in children: cohort study, systematic review, and meta-analysis. *J Neurosurg Pediatr.* 2021;27(3):357-363. doi:10.3171/2020.7.PEDS20447
6. Tartarin H, Morotti A, Van Etten ES, et al. Uncommon causes of nontraumatic intracerebral hemorrhage. *Stroke.* 2024;55(5):1416-1427. doi:10.1161/STROKEAHA.123.043917
7. Binder H, Majdan M, Leitgeb J, et al. Management and outcome of traumatic intracerebral hemorrhage in 79 infants and children from a single level 1 trauma center. *Children (Basel).* 2021;8(10):854. doi:10.3390/children8100854
8. Auer RN, Sutherland GR. Primary intracerebral hemorrhage: pathophysiology. *Can J Neurol Sci.* 2005;32 Suppl 2:S3-S12.

9. Boulouis G, Fox CK, Waak M, et al. Pediatric intracerebral hemorrhage management-consensus statement of the International Pediatric Stroke Organization-Part 1: acute phase and workup. *J Am Heart Assoc*. 2026; 15(1):e039594. doi:10.1161/JAHA.124.039594
10. Mrakotsky C, Mailo JA, Chevignard M, et al. Pediatric intracerebral hemorrhage management-consensus statement of the International Pediatric Stroke Organization-Part 2: outcomes, rehabilitation, and transition to adulthood. *J Am Heart Assoc*. 2026;15(1):e039595. doi:10.1161/JAHA.124.039595
11. Hou W, Wang S, Fang J. Etiology and early clinical predictors of neurological outcomes in pediatric spontaneous intracranial hemorrhage: a retrospective exploratory study. *Front Neurol*. 2026;16:1750475. doi:10.3389/fneur.2025.1750475
12. Sporns PB, Psychogios MN, Fullerton HJ, Lee S, Naggara O, Boulouis G. Neuroimaging of pediatric intracerebral hemorrhage. *J Clin Med*. 2020; 9(5):1518. doi:10.3390/jcm9051518
13. Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations. *J Neurosurg*. 1986;65(4):476-483. doi:10.3171/jns.1986.65.4.0476
14. Zabramski JM, Wascher TM, Spetzler RF, et al. The natural history of familial cavernous malformations: results of an ongoing study. *J Neurosurg*. 1994;80(3):422-432. doi:10.3171/jns.1994.80.3.0422
15. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. *Neurosurgery*. 1980;6(1):1-9. doi:10.1227/00006123-198001000-00001
16. Medley TL, Miteff C, Andrews I, et al. Australian clinical consensus guideline: the diagnosis and acute management of childhood stroke. *Int J Stroke*. 2019;14(1):94-106. doi:10.1177/1747493018799958
17. Ciochon UM, Truelsen TC, Hoei-Hansen CE, et al. Causes of acute intracranial hemorrhages in pediatric population from a single tertiary center in Denmark: a retrospective study. *Pediatr Neurol*. 2025;168:6-12. doi:10.1016/j.pediatrneurol.2025.04.002
18. Tierradentro-García LO, Zandifar A, Ullman NL, et al. Imaging of suspected stroke in children, from the AJR Special Series on Emergency Radiology. *AJR Am J Roentgenol*. 2023;220(3):330-342. doi:10.2214/AJR.22.27816
19. Al-Rubaie A. Emerging imaging techniques and clinical insights in traumatic subarachnoid hemorrhage and brain injury. *Neuroimaging*. 2026;1(1):3. doi:10.3390/neuroimaging1010003
20. Askarova AE, Zhurkabayeva BD. Hemorrhagic stroke in children. *J Cent NervSystDis*.2024;16:11795735241289913.doi:10.1177/11795735241289913
21. Lam JFC, Cheung EYH, Chan EKY, et al. Pediatric hemorrhagic stroke: a retrospective case series in Hong Kong. *Brain Hemorrhages*. 2023;4(1): 6-12. doi:10.1016/j.hest.2022.05.006
22. Bouwman FCM, Verhoeven BH, Klein WM, Schultze Kool LJ, de Blaauw I. Congenital vascular malformations in children: from historical perspective to a multidisciplinary approach in the modern era-a comprehensive review. *Children (Basel)*. 2024;11(5):567. doi:10.3390/children11050567