

EXPERT-CONSENSUS REPORT

Pediatric Intracerebral Hemorrhage Management—Consensus Statement of the International Pediatric Stroke Organization—Part 1: Acute Phase and Workup

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ABSTRACT: Pediatric intracerebral hemorrhage (pICH) is a rare but serious neurologic emergency associated with significant morbidity. Although pICH accounts for nearly half of all pediatric strokes, it remains understudied, and dedicated evidence-based management guidelines are lacking. To address this gap, the International Pediatric Stroke Organization convened a multidisciplinary international working group in 2020 to develop a comprehensive, consensus-based framework for the acute evaluation and management of pICH in children aged 28 days to 18 years. The working group included child neurologists, neurointensivists, neurosurgeons, neuroradiologists, and neurointerventionalists. Subgroups conducted systematic literature reviews and formulated key clinical questions. A modified Delphi process was used to derive consensus statements across 6 domains: prehospital and emergency care, diagnostic imaging and workup, neurocritical care and medical management, neurosurgical and neurointerventional approaches, and identification of knowledge gaps. Through rounds of structured review and voting, 21 consensus statements were developed and approved. The process was endorsed by multiple professional societies. This represents the first international, multidisciplinary, multisociety consensus statement focused on the acute management of pICH in children. It provides structured, expert-driven guidance to inform clinical decision-making, reduce practice variability, and highlight areas for future research. These consensus statements aim to support clinicians worldwide in improving outcomes for children with pICH.

Key Words: nontraumatic cerebral hemorrhage ■ pediatric hemorrhagic stroke ■ pediatric intracerebral hemorrhage

See Editorial by Jordan and Kirton.

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CLINICAL PERSPECTIVE

What Is New?

- This consensus statement from the International Pediatric Stroke Organization provides the first comprehensive, multidisciplinary recommendations for the acute management of pediatric intracerebral hemorrhage, emphasizing standardized diagnostic and therapeutic pathways.

What Are the Clinical Implications?

- Early recognition of pediatric intracerebral hemorrhage symptoms, prompt imaging, and multidisciplinary acute management is critical for improving outcomes. This statement highlights key interventions including neuroprotective strategies, targeted neuroimaging, and tailored neurosurgical and neurocritical care approaches.
- Further multicenter studies are needed to validate these recommendations, refine imaging and treatment protocols, and identify optimal strategies for long-term recovery and prevention of recurrent hemorrhage in children.

Nonstandard Abbreviations and Acronyms

COL4A	collagen type IV α
CTA	computed tomography angiography
DSA	digital subtraction angiography
ICH	intracerebral hemorrhage
ICP	intracranial pressure
IPSO	International Pediatric Stroke Organization
pICH	pediatric ICH

Hemorrhagic stroke is a rare but serious emergency in children.^{1,2} The present consensus statements focuses on pediatric intracerebral hemorrhage (pICH), the most common form of hemorrhagic stroke in infants and children, defined as nontraumatic hemorrhage within the brain tissue or ventricles in children aged 28 days to 18 years.

Although pICH accounts for nearly half of all pediatric strokes, it remains an understudied condition with limited evidence to guide management. The incidence of pICH is estimated to range from 10 to 20 per million children per year,³ with underlying causes differing significantly from those in adults.^{4,5} Unlike adult intracerebral hemorrhage (ICH), which is often

associated with hypertension and cerebral amyloid angiopathy microangiopathies, pICH is primarily caused by vascular malformations, coagulopathies, brain tumors, and systemic diseases. These differences impact diagnostic workup, acute management, and long-term care strategies. Additionally, pICH carries a high risk of morbidity, with many survivors experiencing lasting neurological, cognitive, and functional impairments that affect independence and quality of life.⁶

Despite advances in neuroimaging and critical care, clinical decision-making in pICH remains challenging due to a lack of standardized guidelines and consensus-based recommendations. Care pathways vary depending on clinical severity, underlying risk factors, available resources, and societal factors. Recognizing this gap, the International Pediatric Stroke Organization (IPSO) was founded in 2019 to foster international and multidisciplinary collaborations aimed at improving the understanding, care, and outcomes of childhood cerebrovascular disease. Through an IPSO-led international effort, this document provides consensus-based guidance for the diagnosis and acute management of pICH, addressing key knowledge gaps and standardizing best practices for clinicians worldwide.

This consensus statement covers key areas essential to pICH management:

1. Prehospital and emergency evaluation: recognition of pICH symptoms, stroke protocols, and initial neuroimaging recommendations.
2. Initial workup: optimal neuroimaging, vascular studies, coagulopathy assessment, and genetic considerations.
3. Neurocritical care and medical management: neuroprotective strategies, seizure monitoring and prophylaxis, and intracranial pressure (ICP) management.
4. Neurosurgical interventions: indications for external ventricular drainage, hematoma evacuation, and surgical management of underlying vascular lesions.
5. Neurointerventional approaches: role of diagnostic and therapeutic angiography in pICH management.

By addressing these critical aspects, this document aims to provide a structured, evidence-informed framework for improving pICH care globally.

CONSENSUS METHODOLOGY

A dedicated working group was launched within IPSO in 2020 with the goal to develop a comprehensive set of recommendations for the management of children with pICH.

plCH Definition

plCH was defined as nontraumatic hemorrhage within the brain tissue or ventricles in children aged from 28 days to 18 years. This definition encompasses intraparenchymal hemorrhage, with or without accompanying intraventricular hemorrhage, and isolated intraventricular hemorrhage. plCH may also be associated with subarachnoid hemorrhage, but pure subarachnoid hemorrhage without ICH is not addressed in this article because the pathophysiology and natural history differ from those of ICH. ICH in neonates has separate causes and management than in older children. This work does not address neonatal ICH except when specified.

Methodology

IPSO convened the Hemorrhagic Stroke Working Group to identify knowledge gaps and present a practical framework for health care professionals. The initial working group was divided into 6 subgroups based on participants' field of expertise. Each subgroup identified a set of relevant practical questions for each topic, with a maximum of 10 questions. The groups then performed a systematic literature search for articles pertaining to the management of plCH in children, using the following search equation in PubMed. ("pediatrics"[MeSH Terms] OR "pediatrics"[All Fields] OR "pediatric"[All Fields]) AND ("intracerebral hemorrhage"[All Fields] OR "cerebral hemorrhage"[MeSH Terms] OR ("cerebral"[All Fields] AND "hemorrhage"[All Fields]) OR "cerebral hemorrhage"[All Fields] OR ("intracerebral"[All Fields] AND "hemorrhage"[All Fields] OR "intracerebral hemorrhage"[All Fields])), restricting the search to articles published after 1990. The last literature search was performed in March 2022. Each team then summarized the literature and drafted recommendations to each question based on the best evidence and shared expertise.

An interventional neuroradiologist (G.B.) and a child neurologist (L.L.L.) reviewed all sections for harmonization and derived a set of 21 statements. A modified Delphi approach was then implemented to reach consensus for each statement.⁷ All contributors were asked to vote (agree/disagree) on each statement and to provide feedback and comments for improvement in cases of disagreement. Statements that reached >80% agreement were approved and included in the present document. Conversely, statements that did not reach an 80% agreement were revised by G.B., L.L.L., and a second child neurologist (L.A.B.) following feedback provided by the study group and underwent a second round of consensus voting. The coordinating team drafted and circulated the final version of the article. Participants were asked to provide comments, disclosures, and other relevant information. All coauthors approved the submitted article on February 29, 2024.

Table Clinical Presentation of Pediatric Intracerebral Hemorrhage

Clinical presentation	Frequency
Headache	46%–80%
Nausea/vomiting	60%
Seizures	20%–40%
Focal neurological deficits	13%–50%
Altered level of consciousness	>50%

PREHOSPITAL PATHWAY AND PEDIATRIC STROKE PROTOCOL

In Children, What Symptoms Should Trigger a Code Stroke?

Typical symptoms that trigger a code stroke include most notably the occurrence of a focal neurological deficit (acute hemiparesis, aphasia); however, plCH can present with less specific symptoms such as vomiting, headache, or altered level of consciousness^{8–10} (see the [Table](#)). Younger children are more likely to experience seizures and nonspecific symptoms.^{1,11} Infants may present with lethargy and poor feeding. Vomiting and drowsiness in an infant can be mistaken for sepsis. Depending on the plCH cause, the onset can vary from rapidly progressing severe headache and neurological deficits to insidious symptoms or progression over days, which can cause a diagnostic delay.⁹

The most common causes of plCH are vascular malformations including arteriovenous malformations (AVMs), arteriovenous fistulas, cavernous malformations, and aneurysms. Patients with known vascular malformations warrant special attention when exhibiting the above-mentioned symptoms. Other conditions associated with plCH include arteriopathies (eg, moyamoya), disorders of coagulation (thrombocytopenia, congenital or acquired coagulopathy) including receiving anticoagulation and hemoglobinopathies (eg, sickle cell disease), and brain tumors.^{12–14}

Statement 1: Specific symptoms that should trigger a code stroke include sudden onset focal neurologic deficits including motor (gait disturbances, hemiparesis, or facial weakness) or nonmotor (visual impairment or sensory symptoms), new onset seizure followed by or preceded by persistent focal neurologic symptoms, or unexplained alteration of mental status in the presence of stroke risk factors.

What Are the Optimal Prehospital Pathway and Destination for a Child With Suspected Stroke?

In children with suspected plCH, the initial care pathway, prehospital management, and triage before hospital arrival is critically important to minimize delays

to treatment and optimize therapeutic opportunities. Recognizing and managing neurosurgical and neurological emergencies can be challenging in children. Institutional or regional code stroke pathways are designed to overcome this challenge. Locally and regionally tailored care protocols and specific care pathways have the potential to facilitate timely diagnosis and decrease time to hyperacute therapies including endovascular revascularization treatment in children with ischemic stroke.¹⁵

Timely assessment of any child with suspected stroke is best facilitated at centers with pediatric stroke care pathways that streamline assessment, determine appropriate neuroimaging, including potential need for anesthesia, and guided management by an interdisciplinary team including pediatric neurology, neurosurgery, and interventional neuroradiology,^{16,17} as well as access to pediatric neuro-intensive care. Prehospital providers should try to notify the destination emergency, neurology, and neuroimaging teams of the arrival of a child with a suspected stroke.

Statement 2: Pediatric stroke protocols should be in place to optimize prehospital and early in-hospital care. They should be adapted to geographic specificities and available resources, and maximize opportunities for acute multidisciplinary decision-making in specialized environments.

Statement 3: Emergency medical services transporting a child with suspected stroke should specify a destination hospital and should contact the hospital before arrival, when possible, similar to trauma transports.

INITIAL WORKUP

In Children With Suspected Stroke, What Is the Optimal Imaging Modality for Initial Diagnosis?

Immediate brain imaging is essential to confirm or exclude hemorrhagic and ischemic stroke diagnoses, assist in consideration of reperfusion therapies in children with arterial ischemic stroke, and facilitate timely implementation of emergent treatments tailored to the cause of symptoms.^{18,19} When feasible, without delaying care and in the absence of contraindications, magnetic resonance imaging (MRI) presents advantages over noncontrast computed tomography in suspected stroke. MRI is more sensitive to early ischemia^{20,21} and is better at differentiating stroke from stroke mimics.²² MRI is highly sensitive to the diagnosis of ICH.²³ Furthermore, MRI has a high yield in detection of underlying causes of hemorrhages, including vascular malformations, vasculitis, and cerebral venous thrombosis.^{24,25} MRI is the most sensitive and specific imaging modality for detecting

cerebral cavernous malformations, a frequent cause of ICH in children.^{10,14,26} MRI has high sensitivity for identifying arteriovenous malformations, the most frequent underlying cause of pICH.²⁵ MRI sequences for a suspected stroke in a child should include, at minimum, diffusion-weighted imaging, T2* gradient recalled echo or its 3-dimensional derivatives,²⁷ T2/fluid attenuated inversion recovery, and time of flight magnetic resonance angiography (MRA).²⁸ MRI protocols should be optimized to be fast and with a diagnostic-level signal-to-noise ratio.

Noncontrast computed tomography (CT) and computed tomography angiography (CTA) have advantages in the emergency setting for pediatric patients with acute stroke symptoms including their wide availability, swiftness of imaging acquisition limiting the need for sedation, and high sensitivity for detecting pICH.⁴ Drawbacks are ionizing radiation and lower sensitivity for non-pICHs or mimics. Both non-contrast CT and MRI are able to quantify hematoma volume and determine signs that can lead to early neurological deterioration such as intraventricular extension of hemorrhage, hydrocephalus, and/or brain herniation.^{29,30}

Statement 4: In children with acute stroke symptoms, brain imaging should be performed as quickly as possible upon hospital arrival after patient stabilization, with MRI/MRA or CT/CTA based on hospital resources and patient factors. Vascular imaging should be included in imaging protocols.

Statement 5: In children with suspected acute ICH, urgent imaging with CT or MRI should be performed to rule out hemorrhage. This can be performed with CT/CTA or MRI/MRA, depending on the circumstances/clinical scenario and hospital resources.

After Imaging Diagnosis of an Acute Intracranial Hemorrhage in a Child, Is Immediate Vascular Imaging Needed?

After diagnostic confirmation of a pICH, vascular imaging should be performed as soon as possible, ideally within the same imaging session that identified the hemorrhage, to assess for a readily identifiable structural vascular cause, the most frequent cause.^{14,31} Early identification of the underlying cause is crucial for guiding management and preventing recurrent hemorrhage. Bleeding patterns can inform further investigations, and input from an experienced pediatric neuroradiologist, neurologist, neurosurgeon, and interventional neuroradiologist is essential. This is usually best achieved at a referral center.

CTA performed <96 hours from symptom onset has a high accuracy for detecting underlying vascular anomalies, with sensitivities ≥95% and specificities approaching 100%.⁴ Positive and negative predictive

values are reported to be >97% in younger adults.³² CT venogram is similarly accurate to detect cerebral venous thrombosis.

Due to the high prevalence of AVMs, advanced MRI techniques, such as dynamic contrast MRA and arterial spin labeling, have shown promise in the initial causative workup of pICH.²⁵ The sequences require validation in larger multicenter studies.

Statement 6: At the time of ICH diagnosis, urgent vascular imaging should be performed using CTA or MRA to assess for an underlying structural vascular cause.

Role of Digital Subtraction Angiography in the Causative Workup

In the context of pICH, MRA/magnetic resonance venogram or CTA/CT venogram are reliable tools for initial diagnosis of vascular malformations associated with pICH. Digital subtraction angiography (DSA) is nonetheless commonly required for the precise characterization of vascular anomalies seen on noninvasive imaging and/or the identification of occult lesions such as small arteriovenous shunts.¹⁶

Subarachnoid hemorrhage, atypical hematoma configuration, and edema out of proportion seen on CT at admission or the presence of abnormal vascular structures in the brain are all distinctive radiological features of identifiable secondary causes of pICH. In these cases, DSA is necessary to identify the underlying cause, including AVMs and less commonly other types of vascular malformations or arteriopathies.

DSA is also indicated in patients with no obvious cause of bleeding despite adequate noninvasive workup. The DSA yield is significantly higher in young adult patients without preexisting hypertension³³ and is justifiable in children with no other identified cause of pICH, because AVM is the most common cause of pICH in children aged to 2 to 18 years.¹⁴ Timing of DSA should take into consideration the clinical state of the patient, the neurosurgeon's judgment on the urgency of surgery, and the multidisciplinary assessment of the risk of early rebleeding based on noninvasive imaging.⁴ In patients with elevated ICP due to an acute pICH, supine positioning for DSA can exacerbate intracranial hypertension. A lie flat trial (head of the bed at 0° for up to 30 minutes) before transporting the patient to the angiography suite can help determine whether the child will tolerate the procedure. When safe from an ICP standpoint, diagnostic investigations with DSA in the first 1 to 2 days in younger patients with no known preexisting risk factor should be considered due to the high prevalence of readily identifiable structural vascular lesions as the cause.^{14,34} Delayed DSA can also show unexpected underlying structural lesions in patients without radiological suspicion of secondary

causes of ICH, and therefore, a follow-up DSA should be considered for all patients when their first DSA does not identify the cause of their pICH and there is not another specific cause found for their ICH (Figure 1).^{35,36}

Statement 7: Cerebral DSA should be performed on all children with no obvious cause on initial vascular workup or to better characterize vascular lesions identified on MRA or CTA. DSA should be performed, when available, by an experienced clinician.

Is There a Role for Systematic Imaging Follow-Up at the Acute Phase?

In children with ICH, the development of new neurologic symptoms should prompt immediate follow-up imaging by either MRI or CT. The decision to perform routine follow-up imaging in patients with pICH without a declining neurologic examination is not straightforward. The risk of rehemorrhage in the acute period is reported to be as high as 32% and depends on the underlying cause.^{37,38} Additionally, hematoma expansion from continued bleeding increases the risk of a poor clinical outcome in pICH. Hematoma expansion occurs in approximately one-third of patients, as assessed between 48 and 72 hours following baseline imaging.^{37,38} The risk of hematoma expansion is increased in children with platelet or coagulation abnormalities. This should be weighed with the concerns of radiation exposure and frequent need for sedation. Protocols for follow-up imaging of children at risk for clinical deterioration secondary to hematoma expansion do not currently exist.³⁹ Timing of repeat imaging should be individualized and depends on the type of hemorrhage, need to identify the cause, anticipated sequelae, and the patient's clinical and neurological status.^{40,41} Follow-up imaging with a low-dose CT protocol could help identify children with postadmission hemorrhage expansion. For patients with associated intraventricular hemorrhage, the role of follow-up imaging is also focused on assessing hydrocephalus, which may require cerebrospinal fluid diversion.

Statement 8: Children at high risk for hemorrhage expansion, for example, with platelet or coagulation abnormalities, should be considered for repeat brain imaging with CT or MRI within 48 hours, even if there are no new symptoms.

Statement 9: Children with stable neurological examinations with normal mental status who are not at high risk of hemorrhage expansion do not systematically require repeat brain imaging with MRI or CT at prespecified early time points.

Is Genetic Testing Recommended in Children With pICH?

Knowledge of genetic causes may aid in post-acute targeted treatment strategies and inform family planning decisions.^{42–44} Cerebral vascular malformations

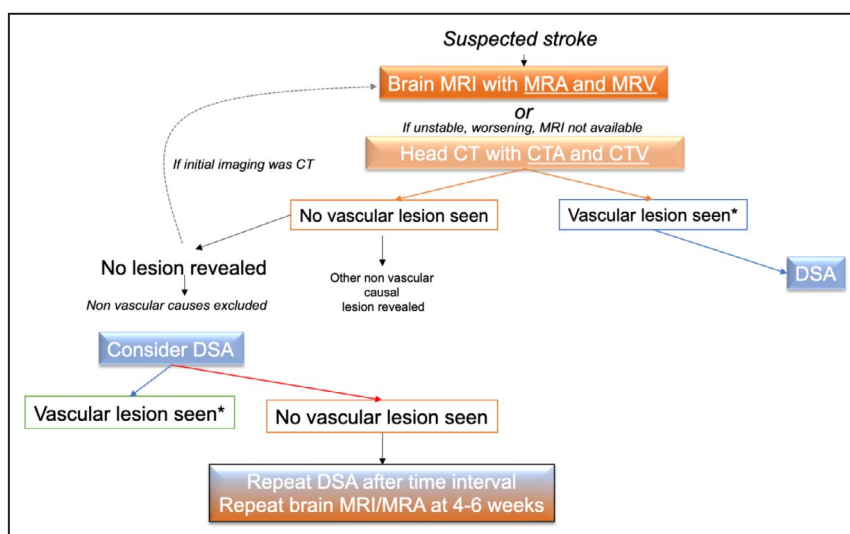


Figure 1. Proposed imaging protocols for children with suspected ICH.

Except if the vascular lesion is a cerebral cavernous malformation, then a DSA is not needed*. CT indicates computed tomography; CTA, computed tomography angiography; CTV, computed tomography venogram; DSA, digital subtraction angiography; ICH, intracerebral hemorrhage; MRA, magnetic resonance angiogram; MRI, magnetic resonance imaging; and MRV, magnetic resonance venogram.

are the most frequent causes for pICH, accounting for approximately half of the reported cases.^{14,45} Genetic conditions predisposing individuals to vascular malformations include familial cerebral cavernous malformations and hereditary hemorrhagic telangiectasia.^{46,47} A history of epistaxis or gastrointestinal bleeding in family members or a child with an AVM suggests the possibility of hereditary hemorrhagic telangiectasia.^{48–51} When hereditary hemorrhagic telangiectasia is suspected, physical examination includes a detailed mucocutaneous assessment for vascular malformations and telangiectasias. Other clinical clues that may suggest a Mendelian, monogenic cause include skin capillary malformations and or limb overgrowth in capillary malformation-AVM syndrome,⁵² neurocutaneous findings suggestive of neurofibromatosis type 1, skin rashes such as livedo reticularis (suggestive of deficiency of adenosine deaminase 2), as well as evidence of connective tissue abnormalities.

AVMs, cerebral cavernous malformations, and intracranial aneurysms are among the most frequent causes for pICH. Genetic mutations associated with AVM include *RASA-1*⁵² and *EPHB4*^{52,53}, *ENG*, *ACVRL1* (ALK-1⁵⁴), *SMAD4*, and presumably *GDF2* (BMP9).⁵⁵ Familial cerebral cavernous malformations are caused by *KRIT1* (CCM1), *CCM2*, and *PDCD10* (CCM3) gene mutations.⁵⁶

As the majority of sporadic brain AVMs harbor activating *KRAS* mutations in nidus endothelial cells,⁵⁷ novel mechanism-targeted medical treatments mainly related to mitogen-activated protein kinase (MEK) inhibition can

potentially halt vessel remodeling and prevent growth of AVMs. However, data on efficacy or safety in intracranial AVMs are limited.⁵⁸ Identification of somatic mutations within brain AVMs can be considered using various techniques, including endovascular biopsy of the endothelial wall of intracranial AVMs before a planned endovascular treatment session. This can be carefully considered in selected cases when clinical and radiologic features are not characteristic or when the AVM is not amenable for surgical or endovascular treatment.⁵⁹ Because obtaining tissue from fast-flow vascular malformation is not without risk, cell-free DNA for genetic diagnostic testing is increasingly being used, but the yield in intracranial AVMs is currently largely unknown.

When pICH from a ruptured intracranial aneurysm is confirmed on neuroimaging studies, a genetic underpinning could be further investigated. Monogenic entities, such as autosomal dominant polycystic kidney disease, Loeys-Dietz syndrome, Marfan syndrome, vascular Ehlers-Danlos syndrome type IV, and rarely Klippel-Trenaunay-Weber syndrome can manifest as single or multiple intracranial aneurysms. Each of these entities has unique genetic foundations, and genetic investigation should be performed according to the specific clinical stigmata.^{60,61}

Multiple studies have revealed *COL4A1* (type IV collagen $\alpha 1$)⁶² and *COL4A2* (type IV collagen $\alpha 2$)⁶³ mutations to be associated with pICH, which can occur during the neonatal or fetal period and occasionally later.⁶⁴ Both genes code for collagen proteins that ensure the integrity of the cerebral vasculature. Small

vessels are often abnormal due to the mutated protein and present as deep parenchymal ICH.⁶⁵ Large vessel aneurysmal disease was also reported in a subset of patients with *COL4A1*-related disorder, but this rarely causes hemorrhagic stroke, particularly in children.⁶⁶ Systemic manifestations suggestive of *COL4A* (collagen type IV α)-related disorders include ocular (cataracts, microcornea, Axenfeld-Rieger anomalies)⁶⁷ or renal involvement. For children without identified vascular abnormalities, coagulation pathway genes could be explored^{68,69} following a demonstration of a primary coagulopathy by clinical and laboratory evaluation. Disorders like hemophilia, factor X or XIII deficiency, or Von Willebrand disease should be ruled out and may be associated with severe pICH.⁷⁰

Statement 10: Personal and family history for children with ICH should be documented to identify children who should undergo specific genetic testing (eg, for hereditary hemorrhagic telangiectasia, familial cavernous malformation syndromes).

Statement 11: For children whose vascular malformations are resected and in whom a germline mutation is not suspected, somatic testing of the lesion should be considered.

Is Workup for Coagulopathy Needed for Children With ICH? Who Should Get It, Which Laboratories to Send?

For children without an identified cause of hemorrhage after a thorough workup aimed at identifying a structural causal lesion, an inherited or acquired hematologic cause of pICH should be excluded. The identification of directly correctable risk factors, such as thrombocytopenia or coagulopathy, will facilitate rapid treatment, thereby limiting the risk for hematoma expansion and secondary brain injury. Hemophilia A (factor VIII deficiency) or B (factor IX deficiency) and von Willebrand disease are the most common inherited causes of hematologic causes of pICH, accounting for nearly 90% of hematologic disorders causing ICH in children.⁷¹ Rarer forms (3%–5%) include factor VII deficiency, factor II, factor XIII deficiency, and vitamin K-dependent clotting factor deficiency.⁷¹ Among acquired hematologic disease, idiopathic thrombocytopenic purpura and anticoagulant therapy-associated hemorrhage are the most frequent causes of pICH.^{72,73} Anticoagulant therapy is particularly common in children with cardiac diseases, especially if requiring mechanical circulatory support including ventricular assist device and extracorporeal membrane oxygenation.⁷² If a bleeding disorder is suspected, a complete blood count with platelets, coagulation studies, prothrombin time, international normalized ratio, and activated partial thromboplastin time, as well as higher-level studies including factor VIII, IX, and XIII levels and von

Willebrand disease studies, should be performed. When a bleeding disorder is already known, a complete blood count with specific coagulation studies can be performed to help rapid correction.

NEUROCRITICAL CARE AND MEDICAL MANAGEMENT

The neurocritical care management of acute ICH requires a balance between ensuring adequate cerebral perfusion in the setting of increased ICP and mitigating the risk of rebleeding, all of which may be complicated by impaired autoregulation, disruptions in the blood–brain barrier, and systemic complications.⁷⁴

General Principles of Acute Management

Early and consistent involvement of an interdisciplinary team can facilitate best practices for patient care. Establishing a cause of pICH is critical to prevent bleeding recurrence, improve outcome, optimize management, and develop follow-up strategy.

What Measures Should Be Used for Neuroprotection?

Pediatric studies in neuroprotection are lacking. Current practices are either extrapolated from adult guidelines or based on small, mainly retrospective, pediatric studies and expert opinions.^{5,20,75} Neuroprotective measures should begin once a neurological/neurosurgical emergency is suspected and follow the principles of optimizing cerebral perfusion and decreasing oxygen consumption (eg, by managing pain and seizures) while obtaining a diagnosis and monitoring for specific complications.

The neuroprotective management of pICH follows similar principles as other types of brain injuries and focuses first on stabilizing the patient, assessing for the presence of elevated ICP, optimizing cerebral perfusion pressure, and identifying immediately correctable risk factors such as thrombocytopenia or coagulopathy (Figure 2).^{4,37,38} An interdisciplinary approach is important for optimal acute management.

ICP Management

Monitoring for signs of increased ICP is imperative. ICP may become elevated in pICH due to the mass effect from the hemorrhage or from obstructive or communicating hydrocephalus from intraventricular hemorrhage.^{76,77} In the unconscious, sedated, or intubated and ventilated children, signs of raised ICP are not always reliable; therefore, consideration of invasive ICP monitoring should be strongly considered and discussed with neurosurgery.⁷⁸ To avoid deterioration and secondary brain injury, the head

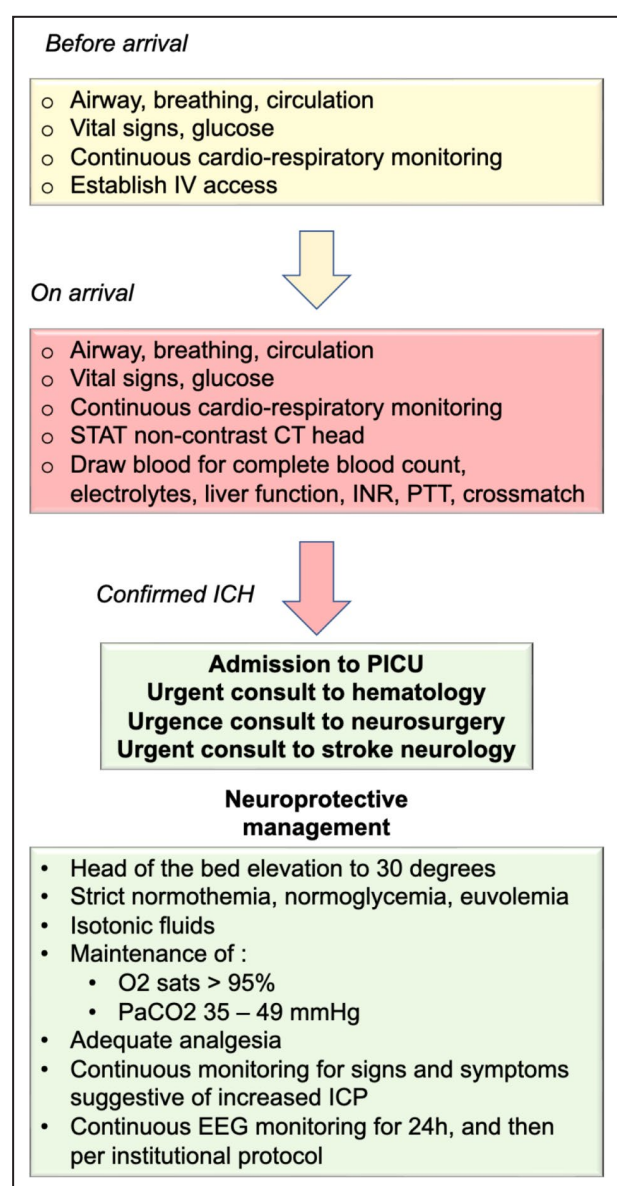


Figure 2. Neuroprotective measures in the context of suspected and confirmed pICH.

CT indicates computed tomography; EEG indicates electroencephalogram; ICP, intracranial pressure; INR, international normalized ratio; pICH, pediatric intracerebral hemorrhage; PICU, pediatric intensive care unit; and PTT, partial thromboplastin time.

should be elevated to 30°⁷⁹ and maintained in a midline position to facilitate venous drainage, followed by strict control of arterial carbon dioxide to prevent excessive vasoconstriction/vasodilation.⁸⁰ Because the patient's head of bed is typically placed at 0° during DSA, a patient's current ICP control should be considered when deciding on the timing of DSA. As noted above, a lie flat trial in the intensive care unit can help determine whether a child will tolerate the supine position without elevated ICP. Neurosurgical interventions, including external ventricular drainage, hemorrhage evacuation,

and craniectomy are discussed in the dedicated neurosurgical management section.

Nonsurgical interventions for increased ICP include administration of hyperosmolar therapy such as intravenous mannitol or hypertonic saline.⁷⁸ Plasma osmolality and electrolytes need to be closely monitored during hyperosmolar therapy to avoid hypotension and hypovolemia. To control ICP, pain should be prevented by adequate analgesia, and in intubated patients, shivering should be controlled with muscle relaxants. Hyperventilation can be used as a temporizing intervention but must be used with caution, because hyperventilation may cause worsening cerebral ischemia.⁸⁰

Blood Pressure Management

Evidence is lacking about blood pressure management goals after pICH. The role of blood pressure reduction to limit hemorrhage expansion and avoid rebleeding in pICH is unknown. Excessive hypertension should be avoided, although exact blood pressure measurements associated with increased bleeding risk in children are unknown. Careful lowering of systolic blood pressure to a target of the 95th percentile for age, sex, height, and weight is reasonable while avoiding hypotension, which could inadvertently reduce cerebral perfusion leading to secondary brain injury.²⁰

Other Neuroprotective Measures

Fever during the first 72 hours has been independently associated with poor outcomes in adult patients with ICH.⁸¹ Therefore, temperatures >37°C should be aggressively treated with antipyretics and cooling blankets. Euvolemia and normoglycemia should be maintained and treatment with glucocorticoids avoided, because randomized controlled trials in adults with ICH showed no neuroprotective benefits^{82,83} and a potential for subsequent hyperglycemia, which can worsen brain injury.^{84,85}

Treating comorbidities such as coagulopathy, anemia, endocrine and electrolyte disturbances, as well as preventing in-hospital complications such as infections or deep vein thrombosis is important, because these contribute to poorer outcomes.⁸⁶ Because patients with pICH have high rates of invasive procedures and often require a prolonged pediatric intensive care unit admission,⁸⁷ treatment of other acute brain injury-related complications include pain management, mood disturbances, delirium, and sleep–wake cycle abnormalities.^{88,89} Post-intensive care syndrome⁹⁰ can have a profound effect on a patient's health-related quality of life.⁹¹

Statement 12: An interdisciplinary approach is important for optimal acute management. The team should include, when possible, a pediatric intensivist,

neurosurgeon, pediatric neurologist preferentially a pediatric neurologist with stroke expertise, neurointerventionalist, neuroradiologist, and hematologist when no underlying vascular lesion is identified or when a platelet or coagulation disorder is present.

Statement 13: Signs of raised ICP are not reliable in unconscious, sedated, or intubated and ventilated children. Therefore, invasive ICP monitoring should be strongly considered and discussed with neurosurgery for children in whom increased ICP may occur but may not exhibit clear signs or symptoms.

What Is the Role of Electroencephalogram Monitoring in a Child With ICH? When? How Long?

Children with acute ICH are at risk of seizures,^{92–94} which may, in turn, increase cerebral metabolic demand,⁹⁵ contribute to ongoing neuroinflammation,⁹⁶ and disrupt normal physiologic regulatory mechanisms, thereby causing secondary ischemia and exacerbating the underlying brain injury. In a mixed cohort of critically ill children, a maximum hourly seizure burden >12 minutes was associated with significant neurologic decline,⁹⁷ and evidence of seizure burden impacting outcome in adults with subarachnoid hemorrhage is accumulating.⁹⁸

Although the impact of continuous electroencephalogram (EEG) monitoring after pICH on the detection and management of electrographic-only and subtle clinical seizures has not been well-studied, guidelines and consensus summary statements recommend that clinicians consider continuous EEG monitoring in children with acute supratentorial brain injury, including ICH, and altered mental status^{99,100} as well as in those with abnormal movements and/or vital sign derangements concerning for seizure.^{20,99,101,102} The ideal duration of monitoring for seizures in a critically ill child depends on the clinical scenario and EEG risk factors on the initial 1-hour recording. Of patients with nonconvulsive seizures, 80% to 95% can be identified within 24 to 48 hours,^{92,103–106} and recording for at least 24 hours is recommended by the American Clinical Neurophysiology Society.¹⁰⁰ In patients who are younger, comatose, pharmacologically sedated, have periodic discharges, or an otherwise abnormal EEG background, nonconvulsive seizures may occur later, and monitoring for 48 hours or more may be necessary.^{103,107,108}

In addition to detecting seizures, an EEG may identify new or ongoing cerebral ischemia, thereby facilitating neuroprotective strategies to optimize cerebral perfusion. This may be particularly relevant in the management of pICH when trying to balance cerebral perfusion and the risk of recurrent hemorrhage. The EEG background undergoes a predictable series of

changes in the setting of decreasing blood flow,¹⁰⁹ and this precedes clinical symptoms indicative of the onset of delayed cerebral ischemia in adults with aneurysmal subarachnoid hemorrhage, thereby allowing for timely intervention.^{110–112}

Statement 14: Continuous EEG monitoring should be considered for the first 24 to 48 hours after ICH in children who are not awake and alert or in whom mental status or neurological examination are fluctuating.

Statement 15: Clinicians should have a low threshold for continuous EEG monitoring, particularly in young infants with altered mental status, unexplained tachycardia, or unusual movements.

What Is the Role of Seizure Prophylaxis in a Child With ICH? For How Long?

The optimal duration of antiseizure treatment in pICH is unknown and depends on the clinical scenario, the electroencephalographic features, and the development of intracerebral posthemorrhagic complications. However, their use may be warranted if EEG monitoring is not readily available and the child does not have a reliable examination, because seizures are frequently reported in children with pICH.¹¹³

Studies that have examined prophylactic antiseizure medications (ASMs) in adults found that those treated had an increased risk of death and disability, hence prophylactic ASM is not recommended in this age group.^{4,114–119} Furthermore, a small trial that randomized patients to 1 month of valproic acid did not demonstrate a reduction in seizures at 1 year.¹²⁰ A meta-analysis of 8 studies, which were mostly observational, found that prophylaxis did not prevent seizures <14 days from pICH or at longest follow-up.¹²¹

Few retrospective or prospective studies address epilepsy after pICH, and there are no clinical trials that examine the efficacy or safety of ASM prophylaxis. In a prospective cohort of 73 pediatric patients with spontaneous ICH, 19 of 53 children (36%) had seizures at presentation.¹¹³ Of 7 children who had acute symptomatic seizures after presentation but within 7 days of hemorrhage, 4 had seizures despite treatment with an ASM. Four children did not have an acute symptomatic seizure yet were discharged on a prophylactic ASM, all of whom had vascular malformations. Nine of 67 survivors developed epilepsy (8 children and 1 neonate), with a 2-year epilepsy-free survival rate of 87%. The only risk factor for epilepsy was elevated ICP requiring surgical management. Of the 9 who developed epilepsy (8 children and 1 neonate), 5 had continued taking antiseizure medications after hospital discharge, a finding that indicated that continuation of ASMs does not prevent future seizures in all patients. Elevated ICP requiring intervention at the acute phase may increase the risk of developing remote symptomatic epilepsy.¹¹³

Out of the 53 children in the study, 27 were loaded with an ASM. In the 27 children loaded with a ASM, 5 children were loaded with phenobarbital (all aged <2 years), 11 children with phenytoin, and 11 children with levetiracetam. A majority of children who were started on a maintenance ASM in the hospital were started on levetiracetam (25 out of 30), and a majority of children who were discharged on an ASM were discharged on levetiracetam (21 out of 26).¹¹³ This is not surprising given that levetiracetam has a better safety profile and similar efficacy in children presenting with benzodiazepine refractory status epilepticus compared with phenytoin.¹²² Lacosamide is another ASM that has been used in critically ill children with minimal side effects.¹²³

Statement 16: Although there are no data to support prophylaxis with ASMs for children with ICH without acute symptomatic seizures, it may be reasonable to use a short course of an ASM with a relatively benign side effect profile like levetiracetam for 1 to 2 weeks in children who are high risk for seizures or in whom a seizure might be particularly deleterious. Children at high risk for seizures may include those with altered mental status, those with elevated ICP, or those with large, untreated vascular malformations.

What Is the Role of Transcranial Doppler Ultrasound in the Management of an Acute pICH?

Children with pICH are prone to increased ICP and its potential deleterious effects. Invasive ICP monitoring is the most common method for direct ICP assessment. Transcranial Doppler ultrasound is a noninvasive technique using acoustic windows of the skull to interrogate intracranial vessels and has been applied in attempts to noninvasively estimate ICP and cerebral perfusion pressure. In a survey of multiple pediatric intensive care units, the 2 most common (more than one-third of respondents) management changes after performing transcranial Doppler ultrasound were making decisions about performing additional neuroimaging and manipulation of cerebral perfusion pressure with fluids and vasopressors.¹²⁴

The use of noninvasive detection methods, such as transcranial Doppler ultrasound as well as continuous EEG monitoring, may aid in distinguishing between compensated hydrocephalus with normal ICP and slowly progressive hydrocephalus with increased ICP¹²⁵ and may identify vasospasm if there is an associated subarachnoid hemorrhage.¹²⁶ Limitations in the routine use of transcranial Doppler ultrasound for vasospasm detection in children include lack of established criteria for vasospasm (age-dependent cerebral blood flow velocities, Lindegaard ratio), rarity of abundant subarachnoid hemorrhage outside the

context of pure subarachnoid hemorrhage due to aneurysm rupture, poor characterization of the duration of risk for vasospasm,¹²⁴ and lack of interoperator reproducibility.

Statement 17: Transcranial Doppler ultrasound has not been studied adequately in intracerebral hemorrhage; therefore, there are insufficient data to provide firm recommendations for its use.

Timing and Principles of Anticoagulation Resumption After pICH

When to resume anticoagulation presumably depends on the cause of ICH, associated conditions (prosthetic cardiac valve), and remains controversial, even in the adult population. Careful assessment of the risk-benefit ratio is required in children on mechanical cardiac support, because clinicians may not feel it is possible to stop anticoagulation in these children. Intensivists might adjust left ventricular assist device parameters to ensure full ejection, with the goal of minimizing thrombus formation during reduced or no anticoagulation. Hematologic consultation is recommended to optimize and monitor coagulation parameters following identification of pICH, including monitoring daily lactate dehydrogenase and plasma hemoglobin levels to identify intrapump thrombus formation and hemolysis.

NEUROSURGICAL MANAGEMENT

In Children With Acute Intracerebral Hemorrhage, What Are the Clinical and Imaging Criteria for External Ventricular Drainage Placement?

Hydrocephalus is a strong determinant of poor outcome following pICH,^{29,30,127} occurring in almost half of children during the initial hospitalization.^{128,129} Acute hydrocephalus is a major driver of raised ICP and is a therapeutic target in the context of pICH. There is no high-level evidence for the selection of children with ICH for external ventricular drainage, but retrospective data indicate lower Glasgow Coma Scale score on admission (typically <8), visibility of temporal horns of ventricles, and higher modified Graeb scores (a semiquantitative scale for intraventricular hemorrhage volume measurement)¹³⁰ are strong predictors for the need of external ventricular drainage placement.

Given the risks of rapid deterioration due to increasing ICP in the context of hydrocephalus, external ventricular drainage placement should be considered before transfer whenever feasible and clinically indicated.

Statement 18: In children with acute ICH, the presence of altered consciousness, neurological

deterioration, hydrocephalus, or intraventricular blood on initial or subsequent neuroimaging should prompt consideration of external ventricular drainage.

In Children With Acute Intracerebral Hemorrhage, What Are the Clinical Imaging Criteria for Hemorrhage Evacuation/Hemicraniectomy?

Hemorrhage evacuation can play an important role in reducing ICP and secondary brain injury due to mass effect in patients with ICH in the posterior fossa and large or symptomatic subcortical lobar hemorrhages.^{129,131} No high-level evidence exists in the pediatric population, but retrospective data have shown that despite more severe clinical presentations, children who were treated with hemorrhage evacuation and/or hemicraniectomy experienced similar long-term functional outcomes and mortality rates as those who did not receive these interventions.^{129,132} In turn, it is important to consider hemorrhage evacuation in patients with significant midline shift, altered mental status, acutely worsening symptoms, or threatening cerebral herniation (temporal, tonsillar) due to mass effect. Hemicraniectomy may be considered in children with refractory increased ICP despite medical treatment optimization, hemorrhage evacuation, and adequate management of hydrocephalus.^{129,132,133}

Statement 19: In children with acute ICH requiring decompression due to raised ICP and/or brain herniation, hemorrhage evacuation and hemicraniectomy should be considered.

Acute Surgical Management of the Underlying Cause?

In children with acutely ruptured AVMs, acute nidal resection is often not justified because of the low risk of early rebleeding proportional to the operative risks in an acutely hemorrhagic context.^{129,134} Exceptions may include superficially located nidus, especially in low-grade lesions for which hemorrhage evacuation is performed¹³⁵ and when complete nidal resection is deemed feasible by an experienced team.¹³⁴ In patients with acutely hemorrhagic cavernomas identified on initial imaging, microsurgical excision is often performed within the first 2 weeks.¹³⁶

Statement 20: In children with acute ICH caused by an AVM, acute surgical excision of the AVM is typically avoided unless there are high-risk features for early rerupture. When the causal lesion is a cavernous malformation, surgical excision is commonly performed in the first weeks following the hemorrhage, whenever a favorable risk–benefit ratio exists.

INTERVENTIONAL NEURORADIOLOGY

Considerations for Facilities Without Pediatric Neurointerventional Capability

Children who arrive at a facility without the capability for safe performance and/or interpretation of DSA should be transferred to a pediatric neurointerventional radiology (NIR) capable facility/center after stabilization within 24 hours.¹⁶ Although it is well understood that dedicated pediatric NIR is rare, the capacity to treat children is important. If a facility is unable to provide the complete spectrum of cerebrovascular care to a child (eg, external ventricular drainage, DSA, embolization, clipping, AVM resection, vasospasm treatment, intensive care unit, and neurosurgical care), patients should be promptly transferred to a center that has these capabilities. For pediatric patients who are unable to undergo transfer due to ongoing instability and elevated ICP, it is advisable that repeat and/or advanced noninvasive imaging be performed and the patient transferred when stable.

General Principles for Therapeutic Angiography

Children who present with a pICH with a vascular cause and were diagnosed with noninvasive imaging should undergo DSA with a plan for intervention, unless the clinical condition requires urgent evacuation of the hematoma and decompressive craniectomy.¹²⁹ The proposed intervention depends upon the cause and the findings of the initial DSA runs.

For pICH secondary to high-flow vascular malformations such as AVM and arteriovenous fistulas, DSA is performed to define angioarchitecture of the vascular malformation for surgical planning but more importantly to identify and treat characteristics that suggest imminent rerupture/rebleed (eg, flow-related parent artery aneurysms and/or intranidal aneurysms).¹⁶

For pICH secondary to moyamoya, the goal of therapeutic DSA is to identify ruptured aneurysms in need of treatment, but it also can be used as a diagnostic tool to assess the severity of disease, external carotid artery collaterals, and related hemodynamics.¹³⁷

In cases of pICH resulting from venous infarction due to cerebral venous thrombosis, and in the presence of clinical deterioration despite optimal medical therapy, such as worsening neurological status, refractory intracranial hypertension, or progressive infarction on imaging, one may contemplate the use of conventional DSA with potential interventions, including mechanical thrombectomy and/or thrombolysis that is in concordance with the American Heart Association's cerebral venous thrombosis consensus statement.¹³⁸

We propose the standard of care for a readily identified vascular lesion amenable to endovascular therapy is management within 24 hours of diagnosis or following respiratory, hemodynamic, and ICP stabilization.

plCH With Concurrent Causes Where Urgent Conventional/Therapeutic Angiography Is Not Indicated

In the case of plCH secondary to cavernous malformation, brain tumor, or other readily identified nonmacrovascular cause, urgent DSA may not be necessary. Diagnostic and therapeutic cerebral angiography may be performed for preoperative evaluation and/or embolization of certain brain tumors (eg, hemangiopericytomas, intraventricular meningiomas), as per local expertise and neurosurgical anticipation of the perioperative bleeding risk. Last, DSA for diagnostic purposes only is not recommended for children aged <3 months. If DSA must be pursued in these patients, the purpose should be to perform an intervention.¹⁶

Timing of Angiography in Unstable Children or With Raised ICP

Priority should be given to respiratory and hemodynamic stabilization. Noninvasive imaging should be performed if it does not jeopardize stabilization efforts. If the patient has clinical and/or radiographic signs of increased ICP, strategies to reduce ICP should be attempted before DSA. DSA should not be performed in a hemodynamically unstable patient or with steadily increasing ICP, except in the rare case where it is required before surgical decompression.

Statement 21: Children with plCH who have a definite cerebral cavernous malformation identified as the cause on noninvasive imaging do not require a cerebral angiogram in the majority of cases.

Neurointerventional Access in Low-Resource Settings

Pediatric patients who present with ICH to hospitals without access to DSA should be transferred to capable centers, as mentioned previously, when stabilized. The increasing rise of centers performing thrombectomy for stroke management in low-income countries offers increasing opportunities for children with plCH to be transferred to centers capable of neurointervention. Establishing pathways and infrastructure in advance to help facilitate access to these resources is recommended. If transfer is not an option, clinicians should get in contact with an experienced team and consider repeat noninvasive imaging as often as needed to best determine the patient's medical care and avoid poor outcomes.

CONCLUSIONS

We have provided comprehensive updates on the acute diagnosis and management of plCH, with a specific focus on integrating current literature and expert consensus into clinical practice. Our aim is to equip health care professionals with the most current and relevant information, addressing existing controversies and identifying gaps in knowledge. This scientific statement is intended to serve as a resource for practitioners involved in the acute care of children presenting with ICH, ensuring informed and effective clinical decision-making. Further prospective, multicenter, international studies are needed to facilitate evidence-based practice.

ARTICLE INFORMATION

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