

Beyond the Scan: Laboratory Investigations in Infant Head Trauma

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Head trauma in children aged 0-2 years represents a diagnostic challenge requiring comprehensive evaluation beyond neuroimaging alone. While computed tomography (CT) dominates diagnostic approaches, laboratory investigations provide critical prognostic information and aid in detecting occult injuries. This editorial examines the essential role of laboratory testing in emergency department assessment of this vulnerable population.

Infants under two years cannot verbalize symptoms, presenting with non-specific manifestations such as vomiting, lethargy, or irritability that may mask life-threatening intracranial hemorrhage. Abusive head trauma remains undiagnosed in approximately 30% of initial presentations (1). The Pediatric Emergency Care Applied Research Network criteria achieve high sensitivity (100%) but modest specificity (53.8%), indicating that many children undergo unnecessary imaging while others with significant injury escape detection (1). One of the articles published in the current issue states that increased severity of head trauma is associated with greater reductions in hemoglobin and hematocrit levels, and elevated glucose levels. Moreover, these laboratory parameters may serve as useful indicators of prognosis and mortality risk in pediatric patients with moderate to severe head trauma.

Laboratory investigations complement imaging by revealing physiologic derangements, metabolic dysfunction, and systemic complications independent of structural lesions on CT scans. These findings fundamentally alter clinical management and prognostic assessment.

Hemoglobin concentration demonstrates remarkable prognostic significance in pediatric head trauma. Studies show that hemoglobin levels correlate inversely with injury severity and outcomes, with lower values predicting mortality and neurologic disability (1). The “delta-hemoglobin ratio” proportional change from admission to nadir hemoglobin independently predicts poor neurologic outcomes. Age-specific thresholds include delta-hemoglobin decrease exceeding 30.7% in infants 0-6 months and -20.6% in 6-12 month-olds (1).

The Pittsburgh Infant Brain Injury score (PIBIS), a validated clinical prediction rule incorporating hemoglobin <11.2 g/dL, achieves 93.3% sensitivity for detecting brain injury in well-appearing infants (1). Hemoglobin measurement serves as a surrogate for cerebral oxygen delivery in the context of impaired autoregulation characteristic of developing brains.

Coagulopathy represents both a consequence of severe brain injury and a critical determinant of outcome. Prospective studies reveal that 22% of children with severe traumatic brain injury demonstrate disseminated intravascular coagulation (2). Tissue factor released from injured brain parenchyma triggers systemic coagulation cascade activation, manifesting as hematoma expansion the leading cause of neurologic deterioration in the first 24-48 hours (2).

Coagulation abnormalities possess particular diagnostic significance in abusive head trauma. Research demonstrates that 54% of abused children with parenchymal brain damage exhibit prothrombin time (PT) prolongation, compared to only 20%



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without parenchymal injury (2). Among fatalities from abuse, 94% display PT prolongation. Critically, these abnormalities develop acutely following injury rather than representing pre-existing hemorrhagic diatheses a distinction with profound protective and legal implications (2).

Serum biomarkers represent a paradigm shift in traumatic brain injury diagnosis. The US Food and Drug Administration approved the Banyan Brain Trauma Indicator in 2018 the first blood-based diagnostic tool measuring glial fibrillary acidic protein and ubiquitin C-terminal hydrolase-L1 (3). These proteins, released following neuronal and glial cell damage, achieve 100% sensitivity and 67% specificity for detecting clinically important traumatic brain injury in children including those under 2 years (3).

Notably, biomarker elevations occur even in children with normal CT scans, detecting microscopic axonal injury invisible to conventional neuroimaging. This finding challenges the traditional “CT-positive” versus “CT-negative” dichotomy, revealing a continuum of neuronal damage with potential implications for cognitive development and post-concussive symptoms (3). Cost-effectiveness analyses indicate biomarker screening becomes economically advantageous when test cost remains below \$308.96, with favorable cost-effectiveness ratios compared to additional CT imaging (3).

Admission blood glucose concentration provides independent prognostic information. Hyperglycemia (glucose >200 mg/dL) correlates with injury severity and unfavorable outcomes, with persistent elevation beyond 48 hours showing strong association with mortality and neurologic disability (2). Elevated glucose exacerbates ischemic brain injury through lactate accumulation, oxidative stress, and cerebral edema formation. Protocols maintaining tight glycemic control (glucose ≤100 mg/dL) demonstrate reduced intracranial pressure and improved functional outcomes (2).

Hyponatremia occurs in 13-20% of hospitalized pediatric head trauma cases, typically from syndrome of inappropriate antidiuretic hormone secretion or cerebral salt-wasting syndrome (2). Distinguishing between these entities proves clinically critical: syndrome of inappropriate antidiuretic hormone secretion requires fluid restriction while cerebral salt-wasting demands aggressive sodium repletion. Hyponatremia predicts poor neurologic outcomes independent of injury severity, likely through exacerbation of cerebral edema (2).

Systematic screening for injuries beyond the clinically apparent proves essential in suspected child abuse. Occult abdominal trauma occurs in 2-10% of physically abused children, presenting

with subtle findings (4). Measurement of hepatic transaminases (aspartate aminotransferase and alanine aminotransferase) and pancreatic enzymes identifies occult liver and pancreatic injuries with high positive predictive value (4).

Current recommendations advocate screening all children under 2 years with suspected abusive injuries using complete blood count, hepatic transaminases, pancreatic enzymes, coagulation studies, and urinalysis (4). Transaminase elevations above 80 IU/L warrant abdominal imaging to exclude solid organ injury (4). Alarming, screening rates remain low only 20-51% of eligible children undergo appropriate testing, yet 41% of screened children yield positive results identifying previously unsuspected injuries (4).

Optimal utilization of laboratory investigations requires integration with clinical findings and imaging within coherent decision-making frameworks. For well-appearing infants within 24 hours of reported minor trauma, PIBIS scores guide neuroimaging decisions. When non-accidental trauma enters the differential, comprehensive laboratory evaluation becomes mandatory (5). Detection of coagulopathy or transaminase elevation triggers additional imaging and subspecialty consultation.

For children with confirmed intracranial injury, serial hemoglobin measurements enable calculation of delta-hemoglobin ratios for prognostic stratification. Glucose monitoring maintains optimal ranges targeting 100-150 mg/dL. Daily electrolyte assessment identifies hyponatremia requiring intervention.

The evaluation of head trauma in children aged 0-2 years demands comprehensive assessment integrating clinical evaluation, neuroimaging, and laboratory investigation. Hemoglobin dynamics predict mortality and disability. Coagulation abnormalities herald hematoma expansion and provide forensic evidence. Biomarkers detect brain damage invisible to CT. Glucose and electrolyte derangements offer modifiable therapeutic targets. Screening panels identify occult injuries.

Conclusion

Professional societies should update clinical practice guidelines recommending laboratory screening protocols stratified by age and injury mechanism. Healthcare systems should invest in point-of-care testing infrastructure and electronic decision support tools facilitating appropriate utilization. For the vulnerable infant whose injury severity may be masked by normal imaging, laboratory investigation provides essential context that can alter outcomes and protect the most vulnerable among us.

References

1. Kuppermann N, Holmes JF, Dayan PS, Hoyle JD Jr, Atabaki SM, Holubkov R, et al. Identification of children at very low risk of clinically-important brain injuries after head trauma: a prospective cohort study. *Lancet*. 2009;374:1160-70. Epub 2009 Sep 14. Erratum in: *Lancet*. 2014;383:308.
2. Hymel KP, Abshire TC, Luckey DW, Jenny C. Coagulopathy in pediatric abusive head trauma. *Pediatrics*. 1997;99:371-5.
3. Puravet A, Oris C, Pereira B, Kahouadji S, Gonzalo P, Masson D, et al. Serum GFAP and UCH-L1 for the identification of clinically important traumatic brain injury in children in France: a diagnostic accuracy substudy. *Lancet Child Adolesc Health*. 2025;9:47-56. Epub 2024 Dec 2.
4. Lane WG, Dubowitz H, Langenberg P. Screening for occult abdominal trauma in children with suspected physical abuse. *Pediatrics*. 2009;124: 1595-602. Epub 2009 Nov 23.
5. Berger RP, Fromkin J, Herman B, Pierce MC, Saladino RA, Flom L, et al. Validation of the Pittsburgh infant brain injury score for abusive head trauma. *Pediatrics*. 2016;138:e20153756.