

Radiographic Findings as Indicators of Neurodevelopmental Outcomes in Abusive Head Trauma



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Gross Motor

Gross Motor

Developmental Domain

Developmental Domain

Visual Motor Problem Solving

Visual Motor Problem Solving

Language

Language

Background

Abusive head trauma (AHT) is known to have high mortality rates (10-40%), but studies on developmental outcomes and prognostic factors are limited. This study aims to identify clinical and radiographic markers of traumatic brain injury severity and neurodevelopmental outcomes.

Developmental Outcomes Following Abusive Head Trauma in Infancy
59 studies <--> 115 assessment tools

Human Brain Development Experience-dependent synapse formation Neurogenesis in the hippocampus Higher cognitive functions (-2 months to 5-10 years) Prefrontal cortex Synaptogenesis Synaptogenesis Synaptogenesis Synaptogenesis Synaptogenesis Adult Adult Age Months Months Years

Methods

Expert-confirmed AHT at TCH 3/2018 – 2/2021 (n= 188)

Demographic data, injury severity scales, radiographic data, neurodevelopmental assessment (n= 112)

2 pediatric neuroradiologists- independent review of brain and spine imaging with discrepancies resolved via consensus (n= 98)

Outpatient follow-up with longitudinal neurodevelopmental assessments via Capute scales and gross motor quotient (n= 56)

Fisher's exact test / chi-squared test:
Injury location and degree neurodevelopmental disability

Multivariate logistic regression for long-term outcome

Results

Figure 1. Percent of patients with specific developmental impairment and degree by hemorrhage location at first office visit following discharge

irge	at iirst	office visit	Norma	Norma	Mild	Modera	Severe, Profour	Norma	Mild	Modera	Severe, Profour	Norma	Mild	Modera	Severe, Profour
		Location n=	20	30	10	4	12	29	13	4	11	38	8	6	5
		Any	0.75	0.8	1	0.75	1	0.74	1	1	1	0.84	1	0.83	1
	96	Frontal	0.65	0.73	1	0.5	1	0.66	1	1	1	0.76	1	0.83	1
	Subdural Hemorrhage	Parietal	0.7	0.73	0.9	0.5	1	0.69	0.85	1	1	0.74	1	0.83	1
	bdı	Temporal	0.55	0.63	0.8	0.5	0.83	0.55	0.85	0.75	0.91	0.68	0.88	0.5	0.8
e e	Su dem	Occipital	0.65	0.7	0.7	0.5	1	0.62	0.85	0.75	1	0.68	1	0.67	1
ΙŽ	-	Tentorial	0.55	0.57	0.6	0.75	1	0.55	0.69	0.75	1	0.61	0.88	0.67	1
age		Cerebellar	0.3	0.27	0.4	0.75	0.75	0.31	0.38	0.5	0.73	0.37	0.63	0.33	0.6
Hemorrhage Type		Any	0.35	0.37	0.1	0	0.58	0.28	0.23	0.25	0.64	0.32	0.38	0.17	0.6
em	.e ë	Frontal	0.3	0.27	0.1	0	0.58	0.21	0.15	0.25	0.64	0.26	0.38	0	0.6
Ι≖	Subarachnoid Hemorrhage	Parietal	0.25	0.23	0.1	0	0.42	0.21	0.08	0	0.55	0.18	0.38	0.17	0.4
	rack	Temporal	0.05	0.1	0.1	0	0.33	0.03	0.08	0.25	0.45	0.11	0.38	0	0.2
	ıbaı Iem	Occipital	0.05	0.03	0	0	0.17	0.03	0	0	0.18	0.03	0.25	0	0
	S T	Cerebellar	0	0	0	0	0.17	0	0	0	0.18	0	0.25	0	0
		Basal Cistern	0.05	0.03	0	0	0.08	0.03	0	0	0.09	0.03	0.13	0	0
	. 0	Any	0.2	0.2	0.2	0.5	0.92	0.21	0.23	1	0.91	0.29	0.39	0.67	1
Hvnoxic	chemi	Cortex	0.05	0.2	0.2	0.5	0.02	0.03	0.15	0.5	0.91	0.29	0.39	0.67	1
3	schemic	Subcortical WM	0.05	0.07	0.1	0	0.5	0.07	0.23	0.5	0.45	0.11	0.17	0.33	0.6
	· <u>~</u>	Deep GM	0.2	0.03	0	0.5	0.5	0.21	0.23	1	0.36	0.11	0.17	0.5	0.6

Figure 2. Percent of patients with persistent
deficits at last office visit by specific
developmental impairment and associated
hemorrhage location

npairment and associated tion			Normal	Normal	Mild	Moderate	Severe/ Profound	Normal	Mild	Moderate	Severe/ Profound	Normal	Mild	Moderate	Severe/ Profound
Location n=			20	30	10	4	12	29	13	4	11	38	8	6	5
		Any	N/A	0.13	0.2	0	0.67	0.18	0.38	0.75	0.73	0.13	0.75	0.6	1
Hemorrhage Type	96	Frontal	N/A	0.14	0.2	0	0.67	0.16	0.38	0.75	0.73	0.14	0.75	0.6	1
	Subdural Subdural Hemorrhage	Parietal	N/A	0.14	0.22	0	0.67	0.15	0.27	0.75	0.64	0.14	0.75	0.6	1
		Temporal	N/A	0.15	0.13	0	0.6	0.11	0.27	0.67	0.73	0.12	0.71	0.67	1
		Occipital	N/A	0.14	0.14	0	0.67	0.13	0.27	0.67	0.73	0.15	0.75	0.75	1
		Tentorial	N/A	0.06	0.17	0	0.67	0.19	0.33	0.67	0.73	0.13	0.71	0.75	1
		Cerebellar	N/A	0	0	0	0.6	0.22	0.2	0.5	0.75	0.14	0.6	1	1
		Any	N/A	0.1	0	N/A	0.71	0	0	0	0.78	0.09	0.67	0	1
		Frontal	N/A	0.13	0	N/A	0.71	0	0	0	0.78	0.1	0.67	N/A	1
		Parietal	N/A	0.14	0	N/A	0.8	0	0	N/A	0.67	0.14	0.67	0	1
		Temporal	N/A	0.33	0	N/A	0.75	N/A	0	0	0.6	0.25	0.67	N/A	1
		Occipital	N/A	0	N/A	N/A	1	N/A	N/A	N/A	0.5	0	1	N/A	N/A
		Cerebellar	N/A	N/A	N/A	N/A	1	N/A	N/A	N/A	0.5	N/A	1	N/A	N/A
		Basal Cistern	N/A	0	N/A	N/A	1	N/A	N/A	N/A	1	0	0.5	N/A	N/A
	U	Any	N/A	0.17	0.33	0	0.7273	0.17	0.33	0.75	0.8	0.18	1	0.75	1
Hvboxic	lschemic Injury	Cortex	N/A	0.17	0.33	0	0.7273	0.17	0.33	0.75	0.8	0.18	1	0.75	1
Ž	inj.	Subcortical WM	N/A	0	0	0	0.67	0	0.5	0.5	0.6	0	1	1	1
	<u> </u>	Deep GM	N/A	0	0	0	0.67	0	0.33	1	0.75	0.25	1	1	1
Independent of Imaging		NI/A	0.03	0.2	0.25	0.67	0.14	0.38	0.75	0.73	0 11	0.75	0.5	1	

Discussion/Conclusions

Subdural hemorrhage is the most common brain injury associated with AHT with limited overall specificity for long-term outcome

Tentorial and cerebellar SDH correlate with increasing severity of gross motor and visual motor problem solving but not language delay

Language deficits are not correlated to hemorrhage location and non-specifically seen with brain injury

Subdural and subarachnoid locations demonstrate statistically significant increases in visual motor problem solving severity

Hypoxic ischemic injury in any location correlates with increasing severity of neurodevelopmental deficit with increased likelihood of persistent deficits for initial severe/profound deficits

More severe deficits regardless of brain injury are associated with higher risk of persistent deficits

Neuroimaging coupled with early developmental assessments can be utilized as cursory markers of likelihood of neurodevelopmental delay and longitudinal persistence

References

correlation

Radiographi

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