Hypoxic Ischaemic Encephalopathy (HIE)

A Single Centre Audit of Incidence, Aetiology and Outcomes

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Introduction

HIE is a neonatal encephalopathy caused by systemic hypoxaemia and/or reduced cerebral blood flow resulting from an acute peripartum or intrapartum insult[1].

The etiology of such insults is variable. HIE can cause significant newborn morbidity. Acute management for infants ≥ 35 weeks includes therapeutic cooling and management from a Neonatal Intensive Care Unit (NICU) team[2].

Table 1: Population characteristics and risk of HIE for 38262 births at ≥ 35 weeks from July 1st, 2015 to December 31, 2022

	HIE (n = 49)	No HIE (n=38213)	p-value
MATERNAL*			
Maternal Age†	31.39 ± 5.07	31.18 ± 4.99	0.78
Advanced Maternal Age (age ≥ 36)	15 (31)	7492 (20)	0.07‡
Overseas-born	36 (74)	25322 (66)	0.36‡
Booking BMI †	25.87 ± 6.29	25.37 ± 5.74	0.58
Nulliparity	26 (53)	15491 (41)	0.08‡
Previous CS	7 (14)	6918 (18)	0.58‡
Multiple pregnancy	2 (4)	1032 (3)	0.38‡
Any diabetes	7 (14)	6605 (17)	0.71‡
Any hypertension	4 (8)	1659 (4)	0.16‡
Any smoking	1 (2)	1857 (5)	0.73‡
CS birth	31 (63)	11910 (31)	<0.01‡
Instrumental VB	10 (20)	4112 (11)	0.04‡ (<0.01 when excl CS)
Breech Vaginal Birth	2 (4)	345 (1)	0.07‡
Oxytocin	12 (25)	10980 (29)	0.64‡
Meconium liquor	20 (41)	6775 (18)	<0.01‡
Labour pyrexia ≥ 38°Celsius	5 (10)	1596 (4)	0.05‡
NEWBORN*			
Gestational Age in Days† (weeks+days)	276.31 ± 10.36 (39+3)	275.59 ± 8.70 (39+2)	0.63
Birthweight†	3323.51 ± 544.89	3319.06 ± 501.20	0.95
Male Gender	29 (59)	19508 (51)	0.32‡
5 min Apgar < 7	43 (82)	597 (2)	<0.01‡
Arterial cord lactate ≥ 6	38 (78)	2026 (n = 13241) (15)	<0.01‡
Born b/n 20:00 and 07:59 h	20 (41)	15578 (41)	1.0‡
Born AH (born outside 08:00-19:59 Mon-Fri)	27 (55)	20077 (53)	0.78‡

*Results are n (%) unless otherwise identified
†Mean +/- SD – relevance calculated with 2 tailed t-test
‡ Calculated values using Fisher's Exact Test with 0.05 significance– performed due to low incidence of HIE cases
Key: AH – after hours; BMI – body mass index; CS – caesarean; VB – vaginal birth

Methods

This single-institution retrospective study examined inborn babies ≥ 35 weeks, meeting HIE therapeutic cooling criteria between July 2015 - December 2022. Cases were identified by NICU records. Data was extracted from hospital databases (ObstetriX and eMaternity) and medical files of infants and mothers.

Table 2: Results from HIE Cases. Cases were separated into pre-admission HIE and Post Admission HIE based on admission CTG

Diagnosis/Cause and Outcomes*	Total N = 49	Pre-Admission HIE (CTG abnormal on admission) N = 19	Post-Admission HIE (CTG normal on admission) N = 30
Principal diagnosis / cause†			
Abruption	**8 (16)**	6 (32)	2 (7)
Fetal anaemia, no abruption	**5 (10)**	3 (16)	2 (7)
Fetal distress, no anomaly, without chorioamnionitis‡	**12 (24)**	6 (32)	6 (20)
Fetal distress, no anomaly, with chorioamnionitis‡	**12 (24)**	3 (16)	9 (30)
Fetal distress, major anomaly	**1 (2)**	1 (5)	-
Breech vaginal birth	**2 (4)**	-	2 (7)
Ruptured uterus - planned term VBAC attempt	**5 (10)**	-	5 (17)
Shoulder dystocia	**3 (6)**	-	3 (10)
Cord prolapse	**1 (2)**	-	1 (3)
Moderate Severe HIE	**31 (63)**	13 (68)	18 (60)
Neonatal death or moderate to severe abnormal outcome§ at follow up	**12 / 47¶ (26)**	6 / 17¶ (35)	6 / 30 (20)
Neonatal death	**3/49 (6)**	3 / 19 (16)	0 / 30 (0)
Moderate to severe abnormal outcome§ at follow up	**9/47¶ (19)**	3/ 17¶ (18)	6 / 30 (20)
Scope for improved care #	**3 yes, 2 possibly (10)**	1 yes, 1 possibly (10)	2 yes, 1 possibly (10)

*All statistics are number (%).
†Values may not add up to 100 due to rounding.
‡Chorioamnionitis diagnosed by intrapartum fever ≥ 38°C and/or moderate to severe acute inflammation on placental histopathology.
§Cerebral palsy, developmental delay, deafness requiring aids, blindness.
¶2 infants lack developmental follow-up, of which one also has Trisomy 21.
Failure to recognise abnormal CTG (1), delay in expediting birth (3), advising labouring VBAC woman to remain at home (1).
Key: CTG – cardiotocograph; HIE – hypoxic ischaemic encephalopathy; VBAC – vaginal birth (attempt) after previous caesarean

Conclusion

The rate of HIE was 1.3/1000 births. HIE causes were variable. Almost 40% presented with an abnormal CTG while 60% evolved after admission. Considerable infant morbidity occurred despite cooling. Scope for improvement in care was identified in 10% of cases.

Results and Discussion

Out of 38262 babies born at ≥ 35 weeks over 7.5 years, 49 received a diagnosis of HIE (1.3 per 1000). Prior to labour/birth there were no differences in characteristics between HIE and no HIE groups (table 1).

In 19 cases (39%), the fetus was significantly unwell at hospital presentation (Pre-admission HIE) whereas 30 were initially well (Table 2). HIE causes such as abruption, acute fetal anaemia and fetal distress with or without chorioamnionitis occurred in both groups, while uterine rupture, shoulder dystocia, breech birth and cord prolapse occurred only after admission. Time of day was not relevant. Twelve (26%) HIE infants died or had moderate-severe abnormal neurological outcome at follow up (4 months to 5 years)[2].

Five cases (10%) demonstrated definite or possible scope for improved care. These cases involved failure to recognise abnormal cardiotocograph features, delays in expediting birth and advising a labouring VBAC woman to remain at home longer than appropriate.

References

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2. Jacobs SE, Berg M, Hunt R et al. Cooling for newborns with hypoxic ischaemic encephalopathy. Cochrane Database of Systematic Reviews 2013, Issue 1. Art No CD003311.