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ORIGINAL ARTICLE

Clinical Manifestations of Symptomatic Intracranial Hemorrhage in Term Neonates: 18 Years of Experience in a Medical Center

Mei-Chen Ou-Yang[1,2,3], Chung-Bin Huang[4,5], Hsin-Chun Huang[2,3], Mei-Yung Chung[2,3], Chih-Cheng Chen[2,3], Feng-Shun Chen[2,3], Pei-Hsin Chao[2,3], I-Lun Chen[2,3], Mei-Hui Ou-Yang[6], Chieh-An Liu[2,3]*

[1]*Division of Neonatology, Department of Pediatrics, Chang Gung Memorial Hospital, Chiayi, Taiwan*
[2]*Division of Neonatology, Department of Pediatrics, Chang Gung Memorial Hospital, Kaohsiung, Taiwan*
[3]*Chang Gung University College of Medicine, Kaohsiung, Taiwan*
[4]*Department of Pediatrics, Kuang Tien General Hospital, Taichung, Taiwan*
[5]*Department of Nursing, Hung Kuang University, Taichung, Taiwan*
[6]*San An Hospital, Kaohsiung, Taiwan*

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Background: Intracranial hemorrhage (ICH) is an uncommon but important cause of morbidity and mortality in term neonates. We conducted a retrospective analysis of the clinical characteristics and developmental outcomes of symptomatic ICH in term neonates.
Methods: A retrospective chart review was conducted of all term neonates (less than 1 month old) diagnosed with ICH and admitted to the neonatal intensive care unit of Kaohsiung Chang Gung Hospital from December 1991 to December 2008. Demographic characteristics, mode of delivery, laboratory data, clinical presentation, and developmental status were recorded.
Results: Data for 24 term neonates (17 boys and 7 girls) with a diagnosis of ICH were collected for analysis. The clinical manifestations of ICH included anemia (13/24, 54%), seizure (11/24, 46%), cyanosis (7/24, 29%), tachypnea (5/24, 21%), fever (1/24, 4%), hypothermia (1/24, 4%), and poor feeding (1/24, 4%). Age at symptom onset ranged from 2 hours to 11 days following birth. The most common type of ICH was subdural hemorrhage. All ICHs resolved, except in one infant, who died from hypoxic-ischemic encephalopathy at 25 days. Ten children with symptomatic ICH were reported to have normal development, while the remainder (13/23, 57%) showed developmental delays or disabilities.
Conclusion: Unexplained anemia, seizure, and cyanosis were the major presenting signs in infants with symptomatic ICH. A diagnosis of ICH should be considered in term neonates who present with one or more of these signs. Although the mortality in term infants with symptomatic ICH was low, more than half the cases reviewed here had subsequent developmental delays or disabilities.

*Corresponding author. Division of Neonatology, Department of Pediatrics, Chang Gung Memorial Hospital at Kaohsiung, 123 Ta-Pei Road, Niao-Sung Hsiang, Kaohsiung Hsien 833, Taiwan.
E-mail: liuca@adm.cgmh.org.tw

1. Introduction

Intracranial hemorrhage (ICH) in full-term neonates usually occurs in the vicinity of the tentorium cerebella and the falx, resulting in posterior fossa hemorrhage in the dural space. The types of ICH seen in neonates include subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH), intraparenchymal hemorrhage (IPH), and intraventricular hemorrhage (IVH). IVH is common in preterm infants but is rare in term neonates.[1] The incidence of symptomatic ICH in newborns at a regional neonatal intensive care unit in the United States was reported to be 2.7/10,000 full-term live births.[2] ICH is associated with apnea, bradycardia, seizure, neurologic disability, and mortality.[3] Several risk factors have been reported to correlate with ICH in term neonates, including assisted vaginal delivery (forceps and vacuum extraction), prolonged second stage of labor, precipitous delivery, maternal parity, fetal weight, and coagulation disorders.[4,5] A high incidence of ICH has been reported in association with labors requiring assisted vaginal delivery or cesarean delivery; however, this may reflect abnormal labor rather than specific modes of delivery.[6] Its incidence is nonetheless consistently increased by the use of vacuum/forceps-assisted extraction,[7] which are associated with higher incidences of SDH and SAH, but not IVH.[5,6]

Early detection of ICH may allow treatment of hemorrhage (in clotting disorders), prevention of further injury (evacuation of subdural hematomas, placement of shunt for increased pressure), family counseling, and longitudinal developmental assessment of affected infants. However, the diagnosis of symptomatic ICH in term neonates may be difficult because of the variety of clinically nonspecific forms of presentation.

The objective of this study was therefore to retrospectively review the clinical presentation, hospital course, and developmental outcome of ICH in term neonates admitted to a neonatal intensive care unit. We aimed to identify the clinical characteristics specific to ICH in term neonates to allow for its early diagnosis and prompt treatment.

2. Materials and Methods

We conducted a retrospective chart review of all term neonates (gestational age $\geq$37 weeks at birth) less than 1 month old diagnosed with ICH and admitted to the neonatal intensive care unit at Kaohsiung Chang Gung Hospital between December 1991 and December 2008. Neonates with IVH only were excluded.

Diagnosis and classification of ICH were based on the interpretation of cranial ultrasound and computed tomography (CT) or magnetic resonance imaging (MRI) studies, and standard radiologic criteria for the determination of ICH were used.[8] Brain ultrasonography was performed when patients presented with symptoms such as seizure, cyanosis, and tachypnea. If the ultrasound findings were positive for hemorrhage or suspected hemorrhage, then further imaging (CT or MRI) was arranged. CT or MRI was also performed at the suggestion of the examining neurologist. The final diagnosis of ICH was made based on interpretation of the imaging findings by an experienced radiologist. Ultrasound examinations were performed within 1 day of the onset of symptoms. If the ultrasonographic findings were positive for ICH, or hemorrhage could not be ruled out, then further evaluation with CT or MRI was performed. Thus CT or MRI studies were performed within 1–3 days or 2–8 days of symptom onset, respectively.

Cases with confirmed ICH were routinely evaluated by experienced neurosurgeons for clinical symptoms of increased intracranial pressure. Neurosurgeons also reviewed the radiographic findings. Radiographic evidence of mass effect or signs of elevated intracranial pressure may necessitate surgical hematoma evacuation. Surgical intervention was determined based on the neurosurgeon's evaluation of the clinical and radiographic findings.

Data on demographic characteristics, mode of delivery, laboratory data, and clinical presentation of ICH were collected for analysis. Developmental follow-up was conducted by telephone interview, written parental responses to questionnaires, and review of the charts from the developmental follow-up program at our institution.

3. Results

Twenty-four term neonates with ICH were identified during the study period. Demographic information on the mothers and infants, delivery information, clinical features, and outcomes are provided in Table 1. All mothers of infants with ICH received appropriate prenatal care. In terms of significant obstetric history, 71% (17/24) of mothers of neonates with ICH were documented as at least gravida 2.

The neonates with ICH had a mean birth body weight of 3090.33 ± 519.27 g. None of the neonates exhibited macrocephaly at birth. Only two of the neonates (cases 10 and 13) were born at Chang Gung Hospital; all others were transferred to our facility. Cesarean section was performed for 11 births (46%), and vacuum extraction was performed for 5 births (21%), of which only 1 case underwent cesarean section (case 16). Prenatal findings included fetal distress (2), placenta previa (1), abruptio placenta (1), preeclampsia (1), premature rupture of membranes

Table 1 Demographic characteristics and clinical features of term neonates with intracranial hemorrhage

No.	Sex	BBW (g)	Apgar score at 1 min	Apgar score at 5 min	G/P/A	Obstetric and labor history	Delivery type	Vacuum	Clinical presentation	Age at symptom onset	Hospital course	Radiological evaluation (CT or MRI)	Outcome
1	F	2400	7	9	G1P1A0	–	NSD	–	Seizure	<1 d	–	SAH, SDH	CP (spasticity)
2	F	3100	8	9	G2P2A0	–	NSD	–	Seizure	4 hr	–	IPH, SDH	Subnormal cognitive status, Mixed DD
3	M	3250	2	6	G2P2A0	Emergent C/S (FD)	C/S	–	Cyanosis	Birth	HIE, infected cephalohematoma	IVH, SAH	Died, day 25
4	F	2950	8	9	G1P1A0	–	NSD	+	Seizure	1 d	Surgery	IPH, SDH	Moderate MR & DD
5	M	3400	7	9	G1P1A0	Placenta previa	C/S	–	Seizure	5 hr	HIE	IPH	Severe MR & DD
6	M	2500	6	8	G2P2A0	–	C/S	–	Seizure	<1 d	Dandy-Walker variant, gastric outlet obstruction	SDH	Mixed DD
7	M	3000	8	9	G2P2A0	–	NSD	–	Tachypnea	Birth	–	SDH	Normal
8	M	4200	9	10	G2P2A0	Protracted 3rd stage	NSD	+	Cyanosis	12 h	–	SAH	Normal
9	M	3920	2	7	G2P2A0	Placenta abruption, emergent C/S	C/S	–	Cyanosis	Birth	–	IPH	Mild motor DD
10	M	2660	7	9	G2P2A0	Prolonged labor	NSD	+	Cyanosis	2 hr	Hydrocephalus	SDH	Mixed DD
11	M	1900	8	10	G3P3A0	PIH, emergent C/S (FD)	C/S	–	Cyanosis	4 hr	–	SDH	Mixed DD
12	F	2800	8	9	G1P1A0	Meconium 2+	NSD	–	Tachypnea	<1 d	Hydrocephalus	SDH, IPH	Normal
13	F	3330	9	10	G2P1A1	IDDM, meconium 1+, nuchal loop	NSD	–	Seizure	2+d	Surgery	SDH, IPH	Normal
14	M	3750	8	9	G2P2A0	–	C/S	–	Seizure	2+d	–	IPH	Normal
15	M	2680	6	8	G2P1A1	Preeclampsia, emergent C/S (FD)	C/S	–	Tachypnea	Birth	DNR requested	SDH, IPH	Mild Motor DD
16	M	2800	7	8	G1P1A0	Meconium 3+	C/S	+	Poor feeding	2+d	–	SDH	Psychomotor DD
17	M	3480	8	9	G2P1A1	GDM, PIH, meconium 4+	NSD	–	Cyanosis	2 d	–	IVH, SAH, IPH	Normal
18	M	3360	9	10	G2P2A0	–	NSD	–	Tachypnea, fever	11 d	GBS meningitis	SAH	Right hemiparesis
19	F	2500	7	8	G4P1A3	Nuchal loop, meconium 3+	C/S	–	Tachypnea	14 hr	–	IPH	Normal
20	M	3428	8	9	G2P2A0	–	C/S	–	Seizure	10 d	–	IPH	Normal
21	M	3380	8	9	G3P3A0	–	C/S	–	Fever, seizure	3 d	Surgery	SDH	Mixed DD
22	M	2880	8	9	G3P1A2	–	NSD	–	Hypothermia, cyanosis	14 hr	–	SDH	Normal
23	F	3180	7	8	G1P1A0	–	NSD	–	Seizure	1 d	–	SDH	Normal
24	M	3320	7	9	G1P1A0	–	NSD	+	Seizure	3 d	–	SDH, SAH	Mild hypotonia

BBW=birth body weight; G/P/A=gravida/para/abortus; CT=computed tomography; MRI=magnetic resonance imaging; NSD=normal spontaneous delivery; SAH=subarachnoid hemorrhage; SDH=subdural hematoma; CP=cerebral palsy; IPH=intraparenchymal hemorrhage; DD=developmental delay; C/S=cesarean section; FD=fetal distress; HIE=hypoxic-ischemic encephalopathy; IVH=intraventricular hemorrhage; MR=mental retardation; PIH=pregnancy-induced hypertension; IDDM=insulin-dependent diabetic mellitus; DNR=do not resuscitate; GDM=gestational diabetes mellitus; GBS=group B streptococcus.

(1), pregnancy-induced hypertension (1), and protracted labor (2). Gestational diabetes and type 1 diabetes were noted in case 13 and case 17, respectively. In addition, cases in which vacuum-assisted delivery was performed primarily resulted in SDH, SAH, SDH+SAH, or any of these in combination with IPH.

Laboratory data for the 24 term neonates with ICH are presented in Table 2. All neonates had a pH $\geq$7.2 at initial arterial blood gas analysis. Thirteen (54%) had anemia with a hemoglobin level $<$140 g/L. Two neonates had thrombocytopenia, with platelet counts of 7.6×10^4/L and 1.9×10^4/L, respectively. All patients received intramuscular vitamin K following delivery. Prolonged prothrombin time (PT) was observed in two patients, and all study infants had normal activated partial thromboplastin time. Nine infants had anemia, but without abnormalities in platelet count, PT, or activated partial thromboplastin time (i.e., no coagulopathy). Lumbar punctures were performed in six neonates; one of the six was documented to have a group B *Streptococcus* infection.

Initial presentation of ICH included seizures in 11 (46%) neonates, cyanosis in 7 (29%), tachypnea in 5 (21%), and fever, hypothermia, and poor feeding (4%) in 1 each. Postnatal age at symptom onset ranged from 2 hours to 11 days. The types of ICH included SDH (15/24, 63%), IPH (10/24, 42%), SAH (5/24, 21%), and IVH (2/24, 8%). ICH often involved multiple compartments (9/24, 38%), which manifested frequently as SDH, IPH or SAH. Five of the 24 neonates required surgical intervention for ICH: two underwent ventriculoperitoneal shunting for hydrocephalus, and three underwent surgical evacuation of subdural hematomas. One infant died (case 3) from hypoxic-ischemic encephalopathy at 25 days old.

Infants with ICH had mean Apgar scores of 7$\pm$2 and 9$\pm$1 at 1 minute and 5 minutes, respectively. There were four infants with low 1-minute Apgar scores; all required resuscitation. Developmental follow-up (range, 0.3–17.3 years) revealed that 10 of the 23 (43%) surviving infants were considered developmentally normal, while 13 (57%) had mild-to-severe developmental delays, including spastic quadriplegia (1), hemiplegia (1), mixed developmental delay (5), moderate mental retardation (2), mild motor developmental delay (2), psychomotor developmental delay (1) and mild hypotonia (1).

Table 2 Laboratory data for term neonates with intracranial hemorrhage

No.	Initial pH	Hb (g/L)	Plt ($\times10^4$/L)	PT	APTT	Lumbar puncture
1	7.348	129	37.6	13.6	35.3	–
2	–	128	58.8	10.3	40	–
3	7.275	177	19.4	10.3	34.5	–
4	–	139	7.6	10.4	35.1	–
5	7.302	147	20.6	11.8	41.9	Negative
6	7.308	124	25.7	–	–	Negative
7	7.466	124	33.2	10	38.9	–
8	7.402	98	13	10	32.7	Negative
9	7.247	166	15.6	13.8	36.8	–
10	7.304	138	24.6	11.8	31.5	–
11	7.216	117	10.3	18.7	39	–
12	7.367	101	13.7	9.3	23.7	–
13	–	189	15.9	10.9	34.1	–
14	7.450	164	27.8	8.9	25.9	Negative
15	7.241	158	1.9	28.1	60.8	–
16	7.359	135	15.6	–	–	–
17	7.420	151	17.6	10	29.5	Negative
18	7.429	142	19.4	–	–	GBS
19	7.359	169	27.2	14.2	51.9	–
20	7.356	120	47.7	9.9	37.7	–
21	7.425	78	17.4	11.3	62.8	Bloody
22	7.385	129	39.3	9.1	32.4	–
23	7.359	196	16.1	13.1	62.1	–
24	7.547	158	21.0	–	–	–

Hb=hemoglobin; Plt=platelets; PT=prothrombin time; APTT=activated partial thromboplastin time; GBS=group B streptococcus. Dashes indicate missing data.

4. Discussion

ICH is a heterogeneous disorder with nonspecific clinical presentations, and varying etiologies and neurological outcomes. The incidence of SAH or SDH in symptomatic term neonates is associated with forceps and vacuum-assisted delivery.[4,5,9] Vertical molding, mechanical compression, and shearing and tearing of the falx and tentorium or bridging cortical veins secondary to difficult delivery or abnormal labor putatively contribute to the development of SDH, as even small amounts of blood in the posterior fossa may contribute to hydrocephalus or neurological deficits.[10–12] Infants born via assisted vaginal delivery are at particular risk for ICH, because prolonged mechanical compression during forceps delivery, and cup detachments or "pop-offs" during vacuum delivery, may result in rapid recompression and injuries to the scalp and associated blood vessels.[13] Such sudden changes in cerebral blood pressure, coupled with perinatal asphyxia induced by abruptio placentae, contribute to the rupture of the premature capillary–venous junction of the germinal matrix.

Of the 24 ICH cases in the present study, nine (37.5%) of the neonates with symptomatic ICH were delivered spontaneously. This suggests that factors other than mode of delivery are involved in the development of ICH. Looney et al[14] recently demonstrated that asymptomatic ICH in term neonates was significantly associated with vaginal delivery, with a prevalence of 26% in vaginal births. Another recent study[15] identified SDH in 46% of asymptomatic

term neonates, including those delivered both vaginally and by cesarean section. The study showed resolution of SDH within 4 weeks, with no neurologic sequelae. These result suggest that mode of delivery may not be the most important risk factor for ICH, and that other issues during the perinatal period may also be important risk factors.[6,15] Stressors, including maternal hypertension, as seen in case 11, and placenta previa and abruptio placenta, as seen in cases 5 and 9, respectively, may contribute to prenatal brain injury, prompting physicians to identify high-risk pregnancies and potentially difficult labors and deliveries promptly.

Clinical presentations of ICH in neonates are nonspecific. Any presentation of the core neurologic symptoms (e.g., seizure, fever, reduced consciousness, generalized hypotonia and increased intracranial pressure) may be indicative of ICH. Hanigan et al[2] reported that the most common presenting symptoms in term infants with ICH were seizures, respiratory distress, and apnea within 2 days of birth (in 24 of 33 infants, 72%); these findings were consistent with those of the current study. It should be noted that apnea without tonic eye deviation may often be overlooked as a manifestation of neonatal seizure, as opposed to an episode of neonatal seizure manifesting as apnea.

A low Apgar score can also be a marker of hypoxic-ischemic and/or hemorrhagic injury. Takahashi et al,[16] in a prospective study evaluating the effect of fetal distress on the neonatal brain, found that all three infants with 5-minute Apgar scores of ≤ 7 evidenced ICH. Jhawar et al[11,17] also found that lower Apgar scores, with or without resuscitation, were risk factors for ICH. Four infants in the present study exhibited low Apgar scores (<7) at 1 minute (cases 3, 6, 9, and 15); however, all but one (case 3) had 5-minute Apgar scores ≥ 7. This suggests that low Apgar scores are not reliable predictors of ICH and that, conversely, normal Apgar scores do not rule out the possibility of neonatal ICH.

Likewise, temperature instability may be a nonspecific sign of ICH. Fang et al[18] recently reported an association between hyperthermia and ICH. One of the patients in the current study had hyperthermia while another had hypothermia. It is possible that such disturbances in temperature regulation are suggestive of underlying neurological abnormalities.

Over half of the term infants with ICH in this study also exhibited anemia; anemia or abnormal hematologic findings may suggest the presence of internal hemorrhaging, including ICHs (i.e., large SDH). Among the 13 anemic neonates in the present study, only four had coagulation abnormalities (decreased platelet count, prolonged PT). A decreased platelet count may indicate alloimmune thrombocytopenia, while prolonged PT may indicate vitamin K deficiency. A diagnosis of intracranial SDH should be considered in such cases, especially when anemia is left unexplained, and a concomitant symptom cluster described by increased blood pressure, hypoxic-ischemic injury, and tachycardia is present (e.g., apnea, cyanosis). Increased vigilance is required when monitoring symptomatic infants.

After confirmation of a diagnosis of ICH, treatment should be symptom-based, beginning with aggressive correction of etiologic factors including thrombocytopenia, coagulopathy, and vitamin K deficiency, followed by close monitoring of cerebral function. Symptomatic ICH in term neonates is associated with a relatively low mortality (11% at 3-year follow-up),[11] but it remains a cause of neurological morbidity among survivors.[13] Our findings revealed a high rate of disability among survivors, with more than half (57%) exhibiting moderate-to-severe developmental delays, including cerebral palsy and mental retardation. This may vary with the type of ICH. Jhawar et al[11] performed a follow-up study of term infants with ICH and found that the most favorable outcomes were seen in those with SDH (80% had no reported problems in cognitive or motor development), while the worst outcomes were seen in infants with SAH and those with multiple compartment involvement. We also found that ICH involving multiple compartments (with SAH, SDH, or IVH) tended to be associated with poorer developmental outcomes: of the 9/24 (38%) infants with multiple compartment involvement, 6/9 (67%) exhibited abnormal outcomes, while of the 15/24 (62%) infants with single-compartment involvement, only 8/15 (53%) exhibited abnormal outcomes. The majority of patients in the current study with normal development had SDH and/or IPH.

SAH has been attributed to hypoxic-ischemic encephalopathy in addition to superficial cortical trauma caused by mechanical stresses from forceps or vacuum-assisted delivery. Although IPH is less frequently reported in term neonates, it is not uncommon in clinical practice. IPH may be attributed to a number of causes, including germinal matrix hemorrhage, a vascular malformation such as cavernous malformation, a coagulopathy such as vitamin K deficiency, hemorrhagic infarction, and ruptured aneurysm.[19,20] These findings suggest that term infants with SDH or IPH may be able to make better recoveries, and that lifelong disabilities may be more likely to occur in newborns with ICH when multiple compartments are involved.

In conclusion, the most frequent form of ICH in term neonates in this retrospective study was SDH, and the most common presenting signs were seizure, anemia, and cyanosis. There was a high rate of disability among survivors, with more than half exhibiting moderate-to-severe developmental delays,

including cerebral palsy and mental retardation. The presence of one or more of the common presenting signs in full-term neonates should alert physicians to the possibility of ICH. Early diagnosis, management, and counseling can then be undertaken to promote a favorable neurological outcome and minimize distress for families.

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