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Periventricular Hemorrhage in Term and Late Preterm Neonates: Risk Factors and Outcomes

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ABSTRACT

Background: Periventricular hemorrhage (PVH) is common in early preterm neonates but much less common in term and late preterm neonates. The aim of this study was to identify the risk factors and outcomes of PVH in term and late preterm neonates in a tertiary care center in Upper Egypt.

Methods: A total of 33 term and late preterm (≥ 34 weeks of gestation) neonates (18 females and 15 males) admitted at the neonatal intensive care unit (NICU) of Sohag University Hospital and diagnosed with PVH from January 2016 to October 2018 were included in this prospective study. The neonates with major congenital malformation of the nervous system were excluded. The risk factors for PVH were ascertained, and the diagnosis was confirmed by transcranial ultrasound, computed tomography or magnetic resonance imaging. The outcomes were assessed at discharge time and the age of 6 months.

Results: The PVH was diagnosed in 5.9% of the neonates. The median of age was 12 days (age range: 2-28 days) at presentation, and convulsions were the most frequent presentations followed by poor suckling and pallor. The risk factors for PVH included prolonged prothrombin time, thrombocytopenia, non-administration of vitamin K, and prolonged labor in 54.5%, 30 %, 30 %, and 27.3% of the cases, respectively. Moreover, 26 (78.7 %) cases were discharged with good conditions, and 7 (21.2 %) subjects died in the NICU. The investigation of the patients' follow-up at the age of 6 months revealed neurological impairments in 36 % of the survivors. Grade IV and III PVH had mortality rates of 66.67% and 18.75%, respectively (P=0.009). Neurodevelopmental impairments were more frequent in Grade III and IV (P=0.002).

Conclusion: The incidence of PVH was 5.9% among term and late preterm neonates admitted to our NICU. Coagulation disorders and prolonged labor were the leading risk factors. Most patients had favorable outcomes, and one-third of the cases had neurodevelopmental impairments. In addition, mortality was reported in one-fifth of the neonates. Severe grades of PVH had the worst outcomes.

Keywords: Late preterm, Outcome, Periventricular hemorrhage, Risk factors, Term neonates

Introduction

Periventricular hemorrhage (PVH) is a generic term used to refer to germinal matrix hemorrhage and intraventricular hemorrhage (GMH-IVH) and parenchymal hemorrhage in the white matter adjacent to the ventricles (1). The GMH-IVH is common in preterm neonates born before the gestational age of 32 weeks as the source of bleeding mostly from the germinal matrix, which decreases at 32 weeks of gestation (2). Consequently, PVH is much less common in late preterm (34-37 weeks) and term neonates. Furthermore, there is a lack of knowledge about the risk factors and outcomes of PVH in this age group of neonates (3).

Intraventricular hemorrhage is grouped into four grades using ultrasound with the first three grades referring to hemorrhage inside the ventricles and the fourth grade, including intraventricular hemorrhage and parenchymal hemorrhage (4). Although PVH is related to prematurity and might be detected in half of the premature neonates, it is increasingly recognized in term neonates and has been reported in about 3.5% to 5.0% of term neonates (5, 6).

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Symptoms of PVH include convulsions or non-specific symptoms, such as apnea, respiratory distress, poor oral feeding, and fever or pallor (7, 8). Neurological impairments have been reported in 9-57% of the neonates with PVH, and most studies reported mortality rates from 1% to 25 % (9).

The occurrence and risk factors of PVH in term and late preterm neonates are still unclear (10). Maternal risk factors causing PVH includes the usage of drugs, pregnancy-induced hypertension, and placental abruption. Major perinatal risk factors are low Apgar score, resuscitation at birth, thrombocytopenia, breastfed infants with vitamin K deficiency, inherited coagulopathy, disseminated intravascular coagulopathy, increased cerebral venous pressure, prolonged labor, forceps delivery, and suction cup (11).

Identifying the risk factors for PVH has the potential to allow the development of good plans for the prevention of numerous neurodevelop-mental complications in neonates (12). This study aimed to identify the risk factors and outcomes of PVH in late preterm and term neonates admitted to the neonatal intensive care unit (NICU) of Sohag University Hospital.

Methods

Study Design

This prospective cohort observational study was carried out at the NICU in the Pediatric Department of Sohag University Hospital, Sohag, Egypt, from January 2016 to October 2018.

Inclusion Criteria

All term and late preterm (≥ 34 weeks of gestation) neonates with a definite diagnosis of PVH by neuroradiological studies (e.g., transcranial ultrasound, computed tomography [CT] or magnetic resonance imaging [MRI]) in the first 28 days of life were included in the present study.

Exclusion Criteria

Early preterm neonates (≤ 34 weeks of gestation) and neonates with major congenital malformation of the nervous system (e.g., hydranencephaly, anencephaly, encephalocele, and hydrocephalus) were excluded from the present study.

Methods

The neonates with PVH were subjected to clinical history taking, including sex, gestational age, delivery type, Apgar scores, presentation age, presenting symptoms, vitamin K administration after delivery, as well as antenatal, perinatal, and postnatal events. All the neonates underwent full clinical examinations with detailed neurological examinations focusing on congenital anomalies, conscious level, cranial nerves, reflexes, as well as motor and sensory system.

The PVH risk factors were ascertained, including the presence of fetal distress, maternal factors, such as placental abruption, drug use during pregnancy, severe congenital heart disease, and sepsis or systemic anticoagulation. Laboratory investigations included complete blood count and complete coagulation profile (e.g., prothrombin time, prothrombin concentration, and activated partial thromboplastin time). Brain imaging included transcranial ultrasound, CT or MRI.

The neonates with PVH were subdivided into four grades according to the modified Papile classification. In grade I, hemorrhage was confined to the germinal matrix lining the ventricles. Grade II hemorrhage extended into normal-sized ventricle occupying less than 50% of ventricle volume, while grade III hemorrhage spilled into dilated ventricle and bleeding occupying more than 50% of ventricle volume.

Grade IV included intraventricular hemorrhage and parenchymal hemorrhage. Grades I and II were considered mild PVH, while grade III and IV were considered severe PVH (13). The outcomes were clinically assessed at discharge time and age of 6 months by detailed neurological examination and neurological imaging (e.g., CT, MRI).

Ultrasound examination

Transcranial ultrasound examination was performed within the first 24 h of admission to NICU when PVH was suspected using Siemens ACUSON X700 with linear and sector array multi-frequency transducers. It was the initial method for diagnosis through the anterior fontanels. By ultrasound, hemorrhage looks as homogeneous hyperechoic collections in early presentation, which later become heterogeneous. Hemorrhage was observed lining the wall of the ventricles, intraventricular according to its severity or associated with parenchymal extension. Hydrocephalus and parenchymal extension or mass effect were initially assessed by ultrasound, and then further assessment was performed by cross-sectional imaging (e.g., CT or MRI).

Computed tomography examination

The CT examination was carried out using a General Electric Lightspeed 8 Slice scanner. The

technique for brain examination was nonenhanced spiral examination. Most of the cases underwent CT examination to assess the severity and associated complication, such as hydrocephalus, mass effect or parenchymal extension.

Magnetic resonance imaging examinations

The MRI examinations were carried out using Siemens 1.5 Tesla MAGNETOM ESSENZA with routine and gradient sequences that attain the acute and subacute blood signals and allow better assessment of the parenchyma, ventricles, mass effect, and exclusion of congenital anomalies.

Ethical consideration

The protocol of the present study was approved by the Research Ethics Committee of Sohag Faculty of Medicine. Informed consent was obtained from the parents of all enrolled subjects.

Statistical analysis

The data were analyzed using STATA intercooled (version 12.1) software. The quantitative data were represented as mean, standard deviation, median, and range. The data were analyzed using the student's t-test to compare the means of two groups, as well as analysis of variance and Bonferroni post-hoc test for the comparison of the means of three groups or more. When the data were not normally distributed, the Kruskal-Wallis and Mann-Whitney U tests were used for the comparison of three or more groups and comparison of two groups, respectively. The qualitative data were represented as numbers and percentages and compared using either the Chi-square test or Fisher's exact test. P-value less than 0.05 was considered statistically significant.

Results

This study included 33 term and late preterm neonates diagnosed with periventricular hemorrhage in the NICU at the Pediatric Department of Sohag University Hospital, Sohag, Egypt, from January 2016 to October 2018. They represented 5.8% (33/563) of term and late preterm infants admitted to our NICU during the study period. The gestational age ranged from 34-39 weeks and the median of gestational age was 37 weeks. In total, 18 (54.5%) cases were females and 15 (45.5%) subjects were males with a male to female ratio of 1:1.2. The median age at presentation was 12 days (age range: 2-28 days).

More than half of the patients presented with convulsions, while poor suckling and pallor were the presentations of one-quarter and one-fifth of the neonates, respectively. On the other hand, vomiting was the least common presentation (Table 1).

Table 1. Clinical presentations of cases with periventricular hemorrhage

Variable	Number (%)
Convulsion	17 (51.5%)
Poor suckling	8 (24.2%)
Pallor	7 (21.2%
Apnea	5 (15.1%)
Jaundice	4 (12.1%)
Irritability	3 (9%)
Tachypnea	2 (6%)
Vomiting	1 (3%)

According to the obtained results, >50%, 42.4%, and 6% of the neonates were delivered by cesarean section (CS), unassisted normal vaginal delivery (NVD), and NVD with instrumentation, respectively. Most of the neonates were full-term with median 5-minute Apgar score of 9 (range: 7-10). Non-administration of vitamin K was reported in 30% of the neonates, and history of prolonged difficult labor was reported in 27.3% of the cases.

History of neonatal sepsis, fetal distress, and pregnancy-induced hypertension were reported in 9%, 6%, and 3% of the cases, respectively. The results of laboratory investigations revealed prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT) in 18 (54.5%) cases. In addition, 10 (30%) cases had thrombocytopenia (Tables 2 and 3).

Transcranial ultrasound was the most

Table 2. Perinatal risk factors in studied cases

Type of delivery	
Cesarean section	17 (51.5%)
Normal vaginal delivery without instrumentation	14 (42.4%)
Normal vaginal delivery with instrumentation	2 (6%)
Apgar score	
Mean (Standard deviation)	8.8 (0.90)
Median (Range)	9 (7-10)
History of prolonged labor	9 (27.3%)
Instrumental delivery	2 (6%)
History of fetal distress	2 (6%)
Pregnancy-induced hypertension	1 (3%)

Table 3. Postnatal risk factors in studied cases

Prolonged PT and aPTT	18 (54.5%)
No vitamin K administration	10 (30%)
Thrombocytopenia	10 (30%)
Neonatal sepsis	3 (9%)

PT: Prothrombin time
a PTT: Activated partial thromboplastin time

commonly used radiologic modality in 56% of the cases followed by CT in 42% of the subjects, while MRI was performed in only 2 cases. With respect to grading of PVH, <50%, 25%, 18.2%, and 12.1% of the neonates were reported with grade III, grade II, grade IV, and grade I, respectively (Table 4, Figures 1-4).

Table 4. Grades of periventricular hemorrhage

Grades of Periventricular hemorrhage	Number (%)
Grade I	4 (12.1%)
Grade II	7 (21.2%)
Grade III	16 (48.5%)
Grade IV	6 (18.2%)

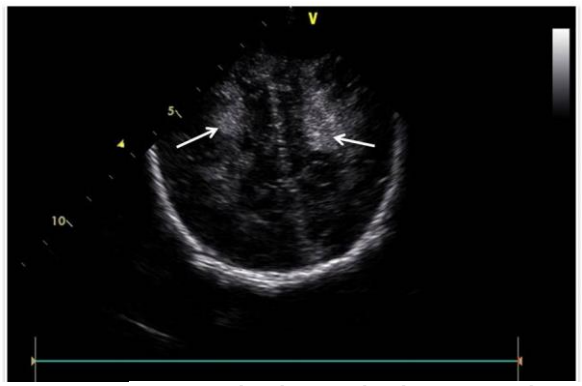

Figure 1. Transcranial ultrasound showing grade I hemorrhage and ventricular wall hyperechoic lesions consistent with hemorrhage [arrows]

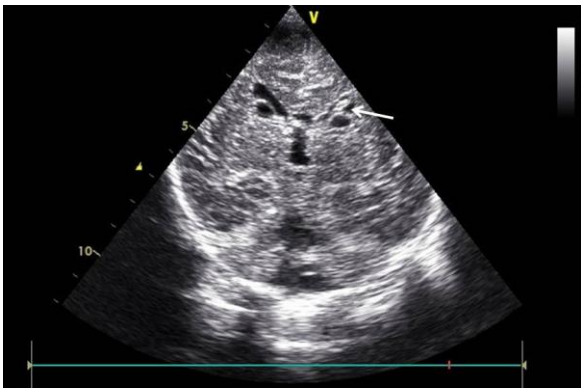

Figure 2. Transcranial ultrasound showing grade II intraventricular hemorrhage [arrows] (Note normal sized ventricles)

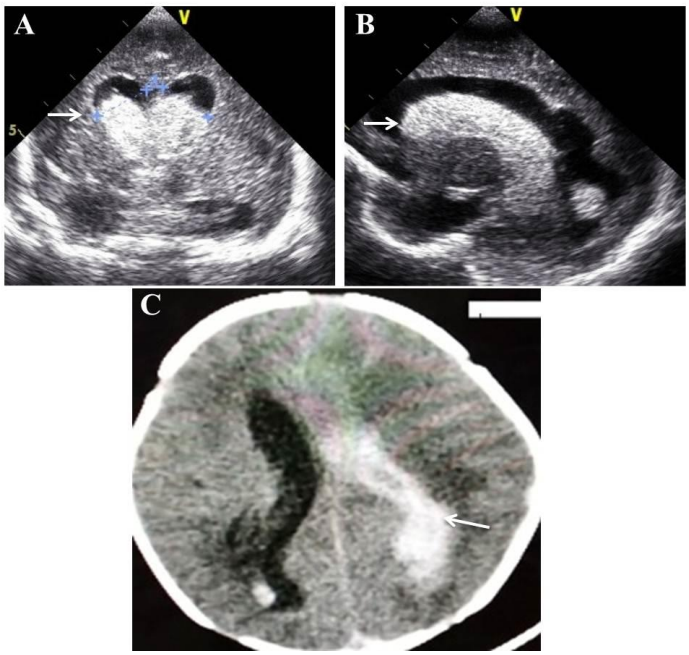

Figure 3. Transcranial ultrasound of A) coronal and B) sagittal views showing grade III intraventricular hemorrhage [arrows] (Note associated hydrocephalus); C) reconstructed non-enhanced computed tomography scan of brain showing grade III intraventricular hemorrhage filling left lateral ventricle [arrows] (Note associated hydrocephalus)

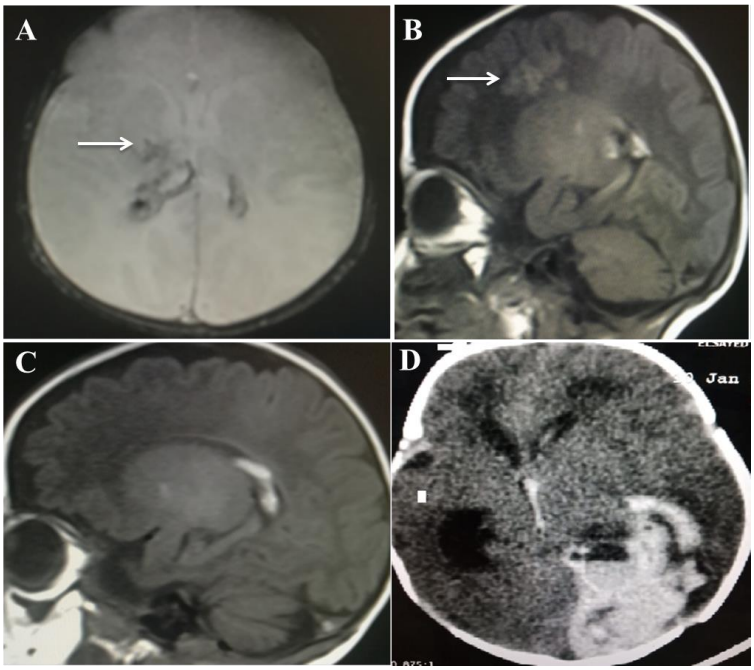

Figure 4. Magnetic resonance imaging A), B), and C) showing axial gradient; sagittal T1 WIs of brain showing grade IV periventricular hemorrhage with parenchymal extensions [arrows]; D) axial computed tomography image showing intraventricular hemorrhage more evident at left lateral ventricle side, as well as within third ventricle with parenchymal extension to left pareito occipital lobes and associated with hydrocephalus and midline shift

Table 5. Neurological outcomes in six months

	Number (%)
Normal neurological outcome	16/25 (64%)
Abnormal neurological outcome	9/25 (36%)
Hydrocephalus	4 (16%)
Spastic four limbs	2 (8%)
Left side hemiparesis	1(4%)
Right upper limb hypotonia	1 (4%)
Bilateral lower limbs weakness	1 (4%)

Table 6. Comparison of different grades of periventricular hemorrhage as outcomes at discharge time and 6 months

Variables	Grade I Number (%)	Grade II Number (%)	Grade III Number (%)	Grade IV Number (%)	P-value
		Discharge time			
Convulsion	0	1 (14.29)	8 (61.54)	0	0.002
Ryle feeding	0	1 (14.29)	5 (38.46)	0	0.43
Death	0	0	3 (18.75)	4 (66.67)	0.009
		6 months			
Abnormal neurological outcome	0	1 (14.29)	10 (76.92)	1 (50)	0.002
No head support	0	0	5 (38.46)	1 (50)	0.03

The outcomes revealed that 26 (78.7 %) cases were discharged, and 7 (21.2%) cases died during the hospital stay. Out of the 26 survivors, 20 (76.9%) cases were neurologically normal, and 6 (23%) cases had convulsions at discharge time and took their food by ryle tube and not directly through the mouth. Follow-up was available for 25/26 (96%) of the survived neonates at the age of 6 months that revealed neurological impairments in 36 % (9/25) of the cases. Moreover, hydrocephalus was the most common complication in 4 (16%) cases and required surgical intervention with shunt placement (Table 5).

The comparison of the outcomes of different grades of PVH at discharge time and 6 months revealed that grade IV had the highest mortality rate (66.67%) followed by grade III (18.75%), while there was no mortality among the cases with grades I and II (P=0.009; Table 6). Moreover, neurodevelopmental impairments were more common in grade III and IV than in other grades (P=0.002).

Discussion

Periventricular hemorrhage is frequently reported in preterm neonates, especially those who were less than 32 weeks of gestation; however, it occurs less frequently in late preterm and term neonates associated with a variety of clinical settings, mainly due to perinatal asphyxia, trauma or coagulation disorders (14). There is scanty data about the risk factors and outcomes of PVH in term and late preterm newborns (5).

This study aimed to identify the risk factors and outcomes of PVH in a cohort of 33 term and late preterm neonates with a median gestational age of 37 (range: 34-39) weeks diagnosed with PVH at the NICU in the Pediatric Department of Sohag University Hospital, a tertiary care center in Upper Egypt, from January 2016 to October 2018.

In the current study, the neonates' age at presentation ranged from 2-28 days with median presentation age of 12 days. It has been reported that most cases of PVH present within the first week of life (15, 16) and rarely beyond the first month after birth (16). The relative delay in the presentation of our cases may be due to delayed referrals from primary and secondary care centers.

Convulsions were the most frequent presentation in this series followed by poor suckling and pallor. Other presentations included apnea, irritability, jaundice, and vomiting. This finding is consistent with the results of other studies (6, 15, 16). Several factors have been indicated as risk factors associated with the occurrence of PVH in term neonates, including instrumental delivery, prolonged difficult labor, low Apgar score, thrombocytopenia, preeclampsia, vitamin K deficiency, and disseminated intravascular coagulopathy (6, 10, 14).

Coagulation disorders in the form of prolonged PT and aPTT were encountered in 18.33% and 54.5% of the cases, respectively, which represented the most important risk factor for PVH in this series. They can be partially attributed to the non-administration of vitamin K in 10.33% and 30% of the cases, as well as other coagulopathies, which could not be diagnosed with available investigations.

Those neonates who did not receive vitamin K at birth were delivered at home by midwives in rural areas. Thrombocytopenia was found in 30% of the cases and may be caused by sepsis in 9% of the cases or may be related to asphyxia associated with prolonged difficult labor in 27.3% of the cases. This result is consistent with the findings of a study carried out by Afsharkhas et al. (15).

However, in the aforementioned study, it was reported that coagulation disorders, including thrombocytopenia, were observed in more than half of the neonates with PVH. In addition, Hubbard et al. reported that failure to administer vitamin K at birth may lead to intracranial hemorrhage as a result of hemorrhagic disease of the newborn (17).

In the current study, prolonged difficult labor was reported in 27% of the cases. It has been reported that perinatal asphyxia associated with difficult prolonged labor is considered one of the risk factors for PVH in term neonates (16, 18). Neonatal sepsis was recognized as a risk factor for PVH in 9% of the cases in this series. This finding is in line with the results of a study conducted by Gupta et al. In the aforementioned study, it was reported that sepsis can lead to PVH by causing thrombocytopenia and disseminated intravascular coagulopathy (19).

In this study, >50%, 42.4%, and 6% of the neonates were delivered by CS, NVD, and NVD with instrumentation, respectively. These findings were similar to the results of a study carried out by Bruno et al. (16), whereas they demonstrated that most cases were delivered by unassisted vaginal delivery and CS without instrumentation. Fetal distress was recognized as an important risk factor for PVH (20).

In this study, fetal distress was reported in 6% of the cases, which is consistent with the findings of a study performed by Bruno et al. However, in the aforementioned study, it was demonstrated that fetal distress was a risk factor for PVH in neonates (16). Maternal hypertension is a well-recognized risk factor for PVH hemorrhage in term neonates (19, 21). In the current study, pregnancy-induced hypertension was the least common associated risk factor for PVH and was encountered in 3% of the cases.

In the current study, grade III of PVH was reported in near half of the neonates followed by grade II and grade IV in 21.2% and 18.2% of the patients, respectively. Grade I was the least frequent grade in 12.1% of cases. Therefore, two-thirds of the neonates in this study had severe grades (III and IV) of PVH. A different pattern was reported in a study conducted by Afsharkhas et al. demonstrating that grade I was the most common (33%) grade followed by grade II (30%), grade IV (20%), and grade III (16.7%) (15).

The outcomes at discharge time revealed that 21.2 % of cases died in NICU due to life-threatening consequences of PVH. This finding is

in line with a mortality rate of 24.5% reported by Brouwer et al. (8) but lower than 12% reported by Bruno et al. (16) and 6.7% reported by Afsharkhas et al. (15). This may be explained by the high prevalence of severe grades of PVH accounting for two-thirds of the cases probably due to the referral of severe cases to our center from other hospitals.

In the current study, neurodevelopmental impairments were reported in 36% of the survivors the most common of which was posthemorrhagic hydrocephalus reported in 16% of the cases, and they were referred to the department of neurosurgery for shunt placement. This finding is in line with the results of a study conducted by Afsharkhas et al, (15), whereas neurologic deficit was reported in 35.7% of the survivors. In addition, 10% of the neonates had posthemorrhagic hydrocephalus.

Contrary to our results Bruno et al. (16) reported neurological deficit in 50% of the survivors, and in a study carried out by Hernandez et al. (22), neurological deficit was demonstrated in 44.4% of the survivors. This variation can be explained by differences in the severity of hemorrhage among neonates in different studies.

Observations in this study demonstrated a statistically significant higher incidence of neurodevelopmental impairments among the neonates with grades III and IV PVH than that among the patients with other grades. In addition, Grade IV PVH had the highest mortality rate followed by Grade III, and there was no mortality among the neonates with Grade I and II PVH. This was in line with the results of a meta-analysis conducted by Mukerji et al. (9), whereas they concluded that severe grades of PVH hemorrhage result in worse neurodevelopmental outcomes than mild grades, and severe grades have the worst outcomes.

Limitations

This study was limited by being performed in a single center and relatively short duration to detect PVH in term and late preterm neonates since the disease is less frequent in this age group of the neonates.

Conclusion

The incidence of PVH was 5.9% among term and late preterm neonates admitted to our NICU. Coagulation disorders and prolonged labor were the leading risk factors. Most patients had favorable outcomes, one-third of the cases had neurodevelopmental impairments, and mortality was reported in one-fifth of the neonates. Severe grades of PVH had the worst outcomes.

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Conflicts of interests

The authors declare no conflict of interest in this study.

References

1. McKechnie L, Levene M. Prevention of periventricular haemorrhage. Paediatr Child Health. 2010; 20(8):362-6.
2. Scorrer T, Drayton M. Preventing periventricular haemorrhage. Curr Paediatr. 2001; 11(3):187-91.
3. Saver JL, Warach S, Janis S, Odenkirchen J, Becker K, Benavente O, et al. Standardizing the structure of stroke clinical and epidemiologic research data: the national institute of neurological disorders and stroke (NINDS) stroke common data element (CDE) project. Stroke. 2012; 43(4):967-73.
4. Levene M. The sequelae of periventricular haemorrhage. Curr Paediatr. 2005; 15(5):375-80.
5. Wu YW, Hamrick SE, Miller SP, Haward MF, Lai MC, Callen PW, et al. Intraventricular hemorrhage in term neonates caused by sinovenous thrombosis. Ann Neurol. 2003; 54(1):123-6.
6. Hong HS, Lee JY. Intracranial hemorrhage in term neonates. Childs Nerv Syst. 2018; 34(6):1135-43.
7. Takenouchi T, Kasdorf E, Engel M, Grunebaum A, Perlman JM. Changing pattern of perinatal brain injury in term infants in recent years. Pediatr Neurol. 2012; 46(2):106-10.
8. Brouwer AJ, Groenendaal F, Koopman C, Nievelstein RJ, Han SK, De Vries LS. Intracranial hemorrhage in full-term newborns: a hospital-based cohort study. Neuroradiology. 2010; 52(6):567-76.
9. Mukerji A, Shah V, Shah PS. Periventricular/ intraventricular hemorrhage and neurodevelop-mental outcomes: a meta-analysis. Pediatrics. 2015; 136:1132-43.
10. Sirgiovanni I, Avignone S, Groppo M, Bassi L, Passera S, Schiavolin P, et al. Intracranial haemorrhage: an incidental finding at magnetic resonance imaging in a cohort of late preterm and term infants. Pediatr Radiol. 2014; 44(3):289-96.
11. Blankenberg FG, Loh NN, Bracci P, D'Arceuil HE, Rhine WD, Norbash AM, et al. Sonography, CT, and MR imaging: a prospective comparison of neonates with suspected intracranial ischemia and hemorrhage. AJNR Am J Neuroradiol. 2000; 21(1):213-8.
12. Xu FL, Duan JJ, Zhang YH, Zhang XL, Guo JJ. Risk factors for periventricular-intraventricular hemorrhage in premature infants treated with

mechanical ventilation. Zhongguo Dang Dai Er Ke Za Zhi. 2012; 14(11):838-41.
13. Papile LA, Burstein J, Burstein R, Koffler H. Incidence and evolution of subependymal and intraventricular hemorrhage: a study of infants with birth weights less than 1,500 gm. J Pediatr. 1978; 92(4):529-34.
14. Szpecht D, Frydryszak D, Miszczyk N, Szymankiewicz M, Gadzinowski J. The incidence of severe intraventricular hemorrhage based on retrospective analysis of 35939 full-term newborns-report of two cases and review of literature. Childs Nerv Syst. 2016; 32(12):2447-51.
15. Afsharkhas L, Khalessi N, Karimi Panah M. Intraventricular hemorrhage in term neonates: sources, severity and outcome. Iran J Child Neurol. 2015; 9(3):34-9.
16. Bruno CJ, Beslow LA, Witmer CM, Vossough A, Jordan LC, Zelonis S, et al. Hemorrhagic stroke in term and late preterm neonates. Arch Dis Child Fetal Neonatal Ed. 2014; 99(1):F48-53.
17. Hubbard D, Tobias JD. Intracerebral hemorrhage due to hemorrhagic disease of the newborn and failure to administer vitamin K at birth. South Med J. 2006; 99(11):1216-20.
18. Al Yazidi G, Srour M, Wintermark P. Risk factors for intraventricular hemorrhage in term asphyxiated newborns treated with hypothermia. Pediatr Neurol. 2014; 50(6):630-5.
19. Gupta SN, Kechli AM, Kanamalla US. Intracranial hemorrhage in term newborns: management and outcomes. Pediatr Neurol. 2009; 40(1):1-12.
20. Armstrong-Wells J, Johnston SC, Wu YW, Sidney S, Fullerton HJ. Prevalence and predictors of perinatal hemorrhagic stroke: results from the kaiser pediatric stroke study. Pediatrics. 2009; 123(3): 823-8.
21. Sims ME, Halterman G, Jasani N, Vachon L, Wu PY. Indications for routine cranial ultrasound scanning in the nursery. J Clin Ultrasound. 1986; 14(6):443-7.
22. Hernandez JL, Anton JM, Cardona AU. Etiological factors and evolution of intracranial hemorrhage in term newborns. J Pediatr Neurol Med. 2016; 1:1-3.