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Hydrocephalus after intraventricular hemorrhage in preterm and low–birth weight infants: analysis of associated risk factors for ventriculoperitoneal shunting

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Abstract

Background: Intraventricular hemorrhage and posthemorrhagic hydrocephalus are common causes of neonatal morbidity and mortality among preterm and low–birth weight infants (PT-LBWIs). Clinical management of posthemorrhagic hydrocephalus (PHH) is difficult and not well standardized. In this study, we aimed to determine the incidence of hydrocephalus after intraventricular hemorrhage (IVH) and the associated risk factors for ventriculoperitoneal (V-P) shunting in PT-LBWIs. We also wanted to identify the medical-care practices for these babies.

Methods: We reviewed the medical records of 42 babies with IVH diagnosed by cranial ultrasonography (classification of Papile et al, *J Pediatr* 1978;92:529-34). We compared 11 babies who required a V-P shunt with the 31 control subjects who did not require a V-P shunt or who died before discharge with respect to risk factors involved in V-P shunting. Maternal, perinatal, and neonatal risk factors, and therapies for IVH and PHH were studied as the V-P shunt–associated risk factors.

Results: The mean gestational age studied was 28.9 ± 2.7 weeks, and the mean birth weight was 1164 ± 391 g. This study revealed an incidence of 26% of PHH in PT-LBWIs with IVH. The most important risk factor for V-P shunt was found to be the severity of IVH ($P < .05$). Late gestational age and the time of IVH were found to be significant as well ($P < .05$). The length of hospitalization was found to be longer in patients with V-P shunt ($P < .05$). Therapies used for IVH and/or PHH were not significant as a risk factor for V-P shunt. In addition, the mortality rate was found to be 38% for all patients with IVH.

Conclusion: Intraventricular hemorrhage in PT-LBWIs remains a significant problem, particularly when it is associated with PHH leading to long-term neurological impairment and decreased survival rate.

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Keywords: Intraventricular hemorrhage; Low birth weight; Newborn; Posthemorrhagic hydrocephalus; Preterm; V-P shunt

1. Introduction

Intraventricular hemorrhage is one of the most common severe complications among PT-LBWIs. Despite a decline in recent years, there are still approximately 20% IVH incidences in infants born before 34 weeks of gestational age [7,25,33]. It is reported that higher-grade hemorrhages more commonly occur in low–birth weight infants [20,28,29]. Posthemorrhagic hydrocephalus with ventriculomegaly is a serious sequela of IVH. Posthemorrhagic hydrocephalus occurs in 35% of the patients with IVH, and 15% of the patients with PHH ultimately require treatment with a V-P shunt [33]. Although the risk for PHH correlates with the size and the location of

Abbreviations: CSF, cerebrospinal fluid; IVH, intraventricular hemorrhage; PHH, posthemorrhagic hydrocephalus; PT-LBWI, preterm and low–birth weight infant; V-P, ventriculoperitoneal.

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the original IVH, our ability to predict PHH severity and eventual need for shunt is poor [6,9,18,21]. Improved prediction could help clarify the complicated and controversial management practices of this condition. In this study, we aimed to determine the incidence of hydrocephalus after IVH and the associated risk factors for V-P shunting in PT-LBWIs. We also aimed to identify the medical-care practices for these babies.

2. Methods

2.1. Patients

Forty-two PT-LBWIs with IVH who were admitted to our neonatal intensive care unit between January 2000 and March 2004 were included in the study. The IVH diagnosis was based on ultrasonography examination supported with cranial computed tomography scan findings. Hemorrhages were classified based on Papile et al [29] in 4 grades: grade I, hemorrhage restricted to the germinal matrix; grade II, IVH without ventricular dilatation; grade III, IVH with ventricular dilatation; and grade IV, parenchymal hemorrhage. Posthemorrhagic hydrocephalus was defined as rapidly progressive when ventricular dilatation was accompanied by excessive increase in head circumference (>2 cm/wk) and/or when other clinical signs of increased intracranial pressure such as bulging fontanel, widening of the sutures, apnea, bradycardia, or seizures occurred.

2.2. Procedures

We compared the 11 babies who required a V-P shunt with 31 control subjects who had IVH but did not require a V-P shunt or who died before discharge. Maternal, perinatal, and neonatal risk factors, and treatments for IVH and ventricular dilatation were investigated as associated risk factors for V-P shunt. The following risk factors were analyzed: (1) birth weight (grams) and sex; (2) gestational age (weeks), determined by the date of last menstruation (whenever this differed by up to 2 weeks as indicated by the ultrasonography performed until the 20th week of gestation, New Ballard method was used as reference; whenever maternal information was absent or unconfirmed, definite gestational age was indicated by New Ballard method [1]); (3) labor/delivery; (4) multiplicity; (5) presence of acute perinatal asphyxia, considered as an Apgar score of 3 or less at 1 minute and/or 5 or less at 5 minutes of life; (6) apnea crisis, defined as breathing pauses of more than 20 seconds followed by bradycardia and/or cyanosis and/or oxygen saturation drop; (7) time of hemorrhage diagnosis (days); (8) grade of hemorrhage; (9) presence of respiratory distress syndrome; (10) presence of patent ductus arteriosus; (11) presence of mechanical ventilation; (12) hydrocephalus diagnosed by ultrasonography and computed tomography; (13) therapies for ventricular hemorrhages and/or ventricular dilatations ([*a*] medical treatment with acetazolamide and furosemide only, [*b*] medical treatment with lumbar puncture, [*c*] external ventricular drainage, and [*d*] external ventricular drainage with intraventricular streptokinase infusion; Fig. 1); (14) length of hospitalization; and (15) outcome.

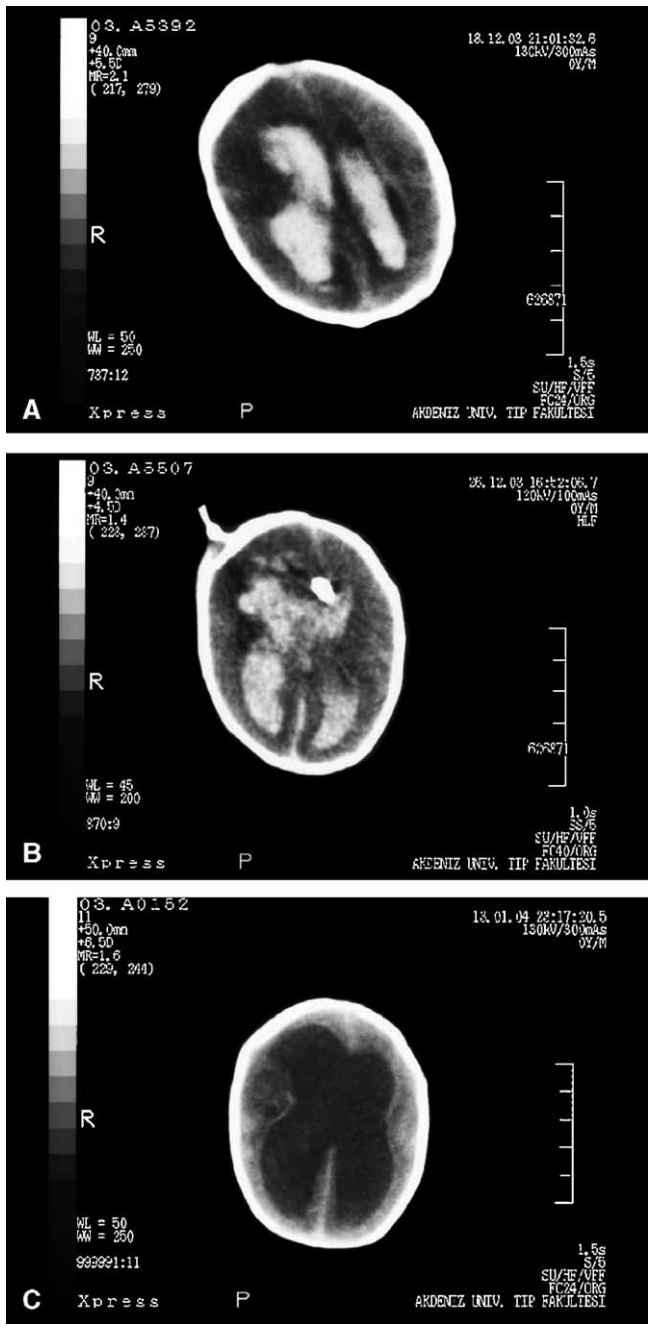


Fig. 1. Intraventricular fibrinolytic therapy with streptokinase infusion in our case. Computed tomography scans shows 1 (A), 8 (B), and 26 (C) days of IVH. This treatment modality did not prevent the progressive PHH.

2.3. Statistical analysis

All values are expressed as mean ± SD. Pearson, χ^2, and Mann-Whitney *U* tests were used to analyze the data from SPSS 11.0 for Windows (SPSS, Chicago, IL). Differences were considered significant when the *P* value was less than .05.

3. Results

Forty-two PT-LBWIs with IVH were studied. The mean gestational age was 28.95 ± 2.75 weeks, and the mean birth weight was 1164 ± 361 g (male-female, 18:24). The severity of IVH was graded as follows: 6 (14.28%) infants exhibited grade I, 11 (26.19%) infants exhibited grade II, 18 (42.85%) infants exhibited grade III, and 7 (16.66%) infants exhibited grade IV. The mean time of ventricular hemorrhage was found to be 6.26 ± 6.52 days of birth.

All patients underwent acetazolamide and furosemide as the initial medical treatment. Among those, 11 (26.19%) developed progressive PHH and required V-P shunt. The most significant risk factor for V-P shunt was identified as the severity of IVH (9 babies in grade III) ($P < .05$). Late gestational age (30.09 ± 2.34 vs 28.55 ± 2.85 weeks) and the time of IVH (11.45 ± 9.05 vs 4.42 ± 4.17 days) were also found to be significant ($P < .05$). The length of hospitalization was found to be longer in patients with V-P shunt ($P < .05$). Therapies for ventricular hemorrhages and/or ventricular dilatations were not significant as risk factors for V-P shunt. Birth weight, sex, labor/delivery, multiplicity, and presence of acute perinatal asphyxia, apnea crisis, mechanical ventilation, respiratory distress syndrome, and patent ductus arteriosus were also not found statistically significant as risk factors. The mortality rate was determined to be 38% in all patients with IVH.

4. Discussion

Intraventricular hemorrhage and posthemorrhagic hydrocephalus are the most important neurological complications in PT-LBWIs during the neonatal period. The incidence of IVH ranges from 5% to 90%, depending on the center, although it frequently oscillates between 30% and 40% [20,23,31,33]. Although the incidence of IVH increases as the gestational age decreases, higher-grade hemorrhages occur more frequently in low–birth weight neonates. Infants with IVH or PHH often have poor neurodevelopmental outcomes such as seizure disorders, IQ problems, severe motor handicaps, and visual problems with multiple handicaps [3,33,34].

Several factors have been implicated in IVH pathogenesis, including intravascular, vascular, and extravascular factors. Among the intravascular factors, any situation leading to an alteration in the cerebral blood flow and/or central nervous system blood pressure may develop into an IVH because of increases in cerebral venous pressure as consequences of mechanical ventilation, barotraumas, apnea crisis, congestive heart failure, sepsis, and others. Vascular factors relate to the microcirculation of the germinal matrix. These capillaries have a marked capillary fragility and are especially susceptible to ischemic lesions, promoting breakage and subsequent bleeding. Extravascular factors include the factors that are disruptive to the mesenchymal and glial supports of matrix vessels. In addition, alterations in local fibrinolytic activity have been observed in areas of hemorrhage [29,33,34]. In summary, prenatal, natal, and postnatal factors are considered contributory to the process.

Posthemorrhagic hydrocephalus with ventriculomegaly is a serious sequela of IVH. The blood may spread through the ventricular system and accumulate in the basilar cisterns at the posterior fossa. Particulate blood clots may impair CSF circulation through the fourth ventricle and the sylvian aqueduct, and block reabsorption sites at the arachnoid villi through the development of adhesions in the basal cisterns [12]. Experimental studies indicate that hydrocephalus has deleterious effects on cerebral white matter, initially involving the oligodendrocytes and later the axons [15,37]. The mechanisms appear to be related to attenuation of vessels in white matter with ischemia and altered energy metabolism [32]. Studies in human infants with PHH also suggest ischemia, alterations in myelin and axons in white matter, and physiological disturbances (impaired visual and somatosensorial evoked potentials), consistent with cerebral white matter injury [2,9,37]. Neonatal white matter damage (often called periventricular leukomalacia) is the major neuropathologic form of this brain injury and underlies most of the neurological morbidity encountered in survivors of preterm birth. Notably, however, experimental studies have also demonstrated the deleterious effects on cerebral cortical neurons, and findings on follow-up of human infants include cognitive deficits, supporting cerebral cortical neuronal effects [5,6,9,26,33,34].

The literature regarding the associated risk factors involved in progressive hydrocephalus in PT-LBWI with IVH is conflicting. A few studies have tried to discern the predictive variables in PHH [6,8,9,11,18,21,27]. The lack of uniformity in assessing a number of variables makes it difficult to compare series. Our present study revealed an incidence of 26% PHH in PT-LBWI with IVH. This result is similar to those reported by other institutions. Hansen et al [11] reported that previously identified antecedents and correlates of IVH also appear to be the antecedents and correlates of progression to V-P shunt among infants with IVH. Our findings did not support this report because, in our cases, previously identified antecedents and correlates for IVH in preterm babies were also associated with high mortality. On the contrary, we found that the late gestational age (average 30 vs 28 weeks) and the time of ventricular hemorrhage (average 11 vs 4 days), but not the previous antecedents and correlates of IVH, are critical for V-P shunt.

Posthemorrhagic hydrocephalus has been noted to occur at greater frequency in those infants with more severe degrees of IVH [11,18,30]. In agreement with those results, we also found the severity of IVH as the most important risk factor for V-P shunt. We believe it is also necessary to differentiate between progressive PHH and ventricular dilatation due to periventricular leukomalacia. Parenchymal injury due to periventricular leukomalacia may cause ventricular enlargement that does not benefit from V-P shunt. Du Plessis [8]

reported that the precise impact of secondary parenchymal injury on the overall neurological outcome of premature infants with PHH remains unclear in large part because of the vulnerability of the immature brain to other forms of injury (eg, periventricular leukomalacia) that may be difficult to distinguish from injury to distension.

There is also no consensus regarding the best treatment of PHH. All known treatments involve significant drawbacks. Conservative treatment including acetazolamide and furosemide for reducing the CSF production rate appears to be relevant only in infants with slowly progressive PHH but not in those in the acute state of increased intracranial pressure [8,33]. Kennedy et al [16] reported that the use of acetazolamide and furosemide in preterm infants with PHH is ineffective in decreasing the rate of shunt placement and is associated with increased neurological morbidity. Other treatment modalities include serial lumbar punctures, serial ventricular punctures, the placement of a subcutaneous ventricular catheter reservoir or shunt, the insertion of an external ventricular drainage, and the placement of a V-P or ventriculosubgaleal shunt [3,4,10,14,17,22,24,35]. Lumbar punctures are frequently associated with high rate of infection, and the amount of CSF drainage may also be insufficient [17]. Serial ventricular punctures would be at the expense of additional parenchymal damage and possible porencephaly at the aspiration site [35]. Ventricular subcutaneous reservoir or external ventricular drainage causes skin breakdown and wound infection. The most common therapy for PHH involves the permanent ventricular shunting. However, the placement of V-P shunts, subsequent to lumbar punctures, is associated with an overall infection rate of 50%, a revision rate of 68%, and a median number of 4 revisions in low–birth weight infants within the first weeks of life [14]. Intraventricular fibrinolytic therapy appears to be a promising method in the management of IVH and PHH [13,19,36]. In this study, therapies for ventricular hemorrhages and/or ventricular dilatations were not found significant as risk factors for V-P shunt. However, more studies with larger numbers of patients need to be conducted.

In conclusion, IVH in PT-LBWIs remains a significant problem, especially when it is associated with PHH leading to long-term neurological impairment and decreased survival.

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Commentary

Posthemorrhagic hydrocephalus (PHH) in premature babies remains a significant problem for the caretakers of these frail patients. Indeed, in most cases, the indication for the surgical treatment of ventriculomegaly remains doubtful in the absence of a clearly progressive macrocrania. Actually, the detection of signs and symptoms of increased intracranial pressure, and consequently, the decision for a treatment are considerably more difficult in this subset of patients as compared with older children. Furthermore, the long-term effectiveness of the placement of cerebrospinal fluid (CSF) shunt device is difficult to evaluate in this type of hydrocephalus where most of the cerebral damage (ie, destruction of the germinal matrix and development of periventricular infarcts) has already occurred at the time of the surgical treatment. The frequently associated complications, namely, infections of CSF shunt procedures, further contribute to the cautious attitude of pediatric neurosurgeons toward the surgical indication. Consequently, the choice for a conservative policy or excessively delayed surgical operation is not uncommon. All the aforementioned factors account for the extremely varying figures in the literature concerning the incidence of PHH and the results of its surgical management.

The value of this article is the attempt to evaluate the incidence of PHH and the relevance of some risk factors on an objective basis by taking into account some well-defined parameters. Therefore, the authors should be congratulated for this difficult task in a still relatively obscure field of clinical practice. They also briefly reviewed the current alternative treatment modalities for the placement of a ventriculoperitoneal shunt; however, endoscopic III ventriculostomy was not mentioned. Actually, although this last procedure often fails in treating PHH definitively, several authors have emphasized its value in postponing the placement of a permanent CSF drainage system. A final remark concerns intraventricular fibrinolytic therapy, which, according to the authors, appears to be a promising therapeutic measure. Unfortunately, in our experience, such a therapy did not result in a significant reduction in the incidence of PHH nor in the severity of the ventricular dilation.

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