

Bedside Ultrasound-Guided Percutaneous Needle Aspiration of Intra- and Extra-Axial Intracranial Hemorrhage in Neonates

Mehmet N. Cizmeci^{1,2} Liesbeth Thewissen³ Alexandra Zecic⁴ Peter A. Woerdeman⁵ Bart de Boer⁵
 Edward Baert⁴ Paul Govaert^{6,7} Jeroen Dudink^{1,2} Floris Groenendaal^{1,2} Maarten Lequin⁸
 Linda S. de Vries^{1,2}

¹Department of Neonatology, Wilhelmina Children's Hospital, University Medical Center Utrecht, Utrecht, The Netherlands

²Brain Center Rudolf Magnus, University Medical Center Utrecht, Utrecht, The Netherlands

³Department of Neonatology, University Hospitals Leuven, Leuven, Belgium

⁴Department of Pediatrics, University Hospital Ghent, Ghent, Belgium

⁵Division of Neuroscience, Department of Neurosurgery, University Medical Center Utrecht, Utrecht, Utrecht, The Netherlands

⁶Section of Neonatology, Department of Pediatrics, Sophia Children's Hospital, Erasmus MC, University Medical Center Rotterdam, Rotterdam, The Netherlands

⁷Section of Neonatology, Middelheim Ziekenhuis-ZNA, Antwerp, Belgium

⁸Department Pediatric Radiology, Wilhelmina Children's Hospital, University Medical Center Utrecht, Utrecht, The Netherlands

Address for correspondence Linda S. de Vries, MD, PhD, Department of Neonatology, Wilhelmina Children's Hospital, University Medical Center Utrecht, KE 04.123.1 Lundlaan 6, 3584 EA Utrecht, The Netherlands (e-mail: l.s.devries@umcutrecht.nl).

Neuropediatrics

Abstract

Intracranial hemorrhage is an important cause of brain injury in the neonatal population and bedside percutaneous needle aspiration has emerged as an alternative due to the major risks that can be caused by standard neurosurgical decompression. We aimed to assess the effectiveness of this minimally invasive bedside technique and conducted a retrospective analysis of all newborn infants with a large extra-axial hemorrhage associated with a parenchymal hemorrhage causing a midline shift, managed at three academic centers over a 15-year period. Collected data included clinical history, laboratory results, review of all imaging studies performed, and neurodevelopmental follow-up. Eight infants (3 preterm and 5 full-term) presented on day 1 to 2 with seizures ($n = 6$) and apneas ($n = 5$), signs of increased intracranial pressure ($n = 4$), and coning ($n = 1$). Risk factors were present in six. Cranial ultrasound and computed tomography showed a midline shift in all; two infants showed status epilepticus on amplitude-integrated electroencephalography with complete resolution after the procedure. Between 7 and 34 mL could be aspirated associated with a decrease in the midline shift as seen by ultrasonography performed during the puncture. No complications were seen related to the procedure and none of the infants required further acute neurosurgical intervention. On follow-up, three had mild sequelae, including motor coordination problems ($n = 1$) and hemianopia ($n = 2$); none developed cerebral palsy or postneonatal epilepsy. Neonates, presenting with severe symptoms, can be managed successfully using ultrasound-guided needle aspiration and this minimally invasive bedside method should be kept in mind before performing neurosurgical decompression.

Keywords

- intracranial hemorrhage
- needle aspiration
- newborn
- subdural

received
 January 18, 2018
 accepted
 February 24, 2018

© Georg Thieme Verlag KG
 Stuttgart · New York

DOI [https://doi.org/
 10.1055/s-0038-1641568.](https://doi.org/10.1055/s-0038-1641568)
 ISSN 0174-304X.

Introduction

Intracranial hemorrhage (ICH) is defined as the pathological accumulation of blood within the cranial vault and is associated with substantial devastating consequences on morbidity, mortality, and long-term neurodevelopmental outcome in neonates. In contrast to preterm infants who exhibit intraventricular hemorrhage (IVH) as the most common form of ICH, term neonates usually present with an extra-axial hemorrhage involving the meninges, commonly seen after a normal delivery, or more rarely with a lobar hemorrhage occurring within the brain parenchyma.^{1,2} Regardless of the gestational age of the infant, the combination of duration of compression of the brain tissue, the extent of bleeding, and often accompanying seizures can have detrimental effects on neurological outcome.^{3,4} Therefore, proper management of this condition, which usually includes neurosurgical intervention, is of paramount importance.

While a decision for conventional neurosurgical decompression is most often made in the neonatal population, this may not be the best option for every patient with a compromised clinical condition. Bedside percutaneous needle aspiration has emerged as an alternative due to the major risks that can be caused by standard neurosurgical decompression including, transfer of the neonate to the operating theater, and further clinical deterioration due to general anesthesia and craniotomy. However, data on percutaneous needle aspiration is limited to a few cases with an extra-axial epidural or subarachnoid hemorrhage.^{3,5-10} The aim of this study was to illustrate the use of ultrasound-guided percutaneous needle aspiration in eight newborn infants with a large extra-axial hemorrhage associated with a parenchymal hemorrhage causing a midline shift and to review the literature on this method.

Methods

A retrospective analysis of all newborn infants with a large extra-axial hemorrhage associated with a parenchymal hemorrhage causing a midline shift, managed with bedside ultrasound-guided percutaneous needle aspiration at three centers over a 15-year period, was conducted. Eight patients were included, all of whom were admitted due to neonatal seizures and/or apneas. Neuroimaging techniques including cranial ultrasound (cUS), computed tomography (CT), and magnetic resonance imaging (MRI) were used. The medical records were used for information on neurodevelopmental outcome. Signed parental informed consent for inclusion in this study was obtained.

Selected Cases

Case 1

This male infant weighing 1050 g ($p = 3-10$) was born to a gravida 1, para 0 mother by emergency cesarean section (C/S) at $31\frac{1}{7}$ weeks. The mother presented in labor with decreased fetal movements and Doppler ultrasonography revealed reversed end-diastolic flow velocities. The parental history and the course of the present pregnancy were unremarkable and the mother received one dose of antenatal steroids before delivery. The infant required intubation in the delivery room

due to respiratory insufficiency and the Apgar scores were 1, 1, and 9 at 1, 5, and 10 minutes, respectively. His umbilical cord blood gas showed a pH of 7.10 with a base excess of -12 mmol/L and a lactate of 13.7 mmol/L. Upon admission to a level 2 hospital, the infant was started on glucose infusion due to a low blood glucose level of 0.1 mmol/L. A complete blood count (CBC) analysis at 6 hours of life showed a hemoglobin of 6.5 mmol/L with a platelet count of 42,000/ μ L for which a thrombocyte and packed red blood cell transfusion were given. A cUS scan was performed, which showed a large left-sided parieto-occipital hemorrhage with a subarachnoid component causing a midline shift (►Fig. 1A). He was started on mannitol infusion (0.5 g/kg) and transferred to our level three neonatal intensive care unit (NICU) for further evaluation. His head

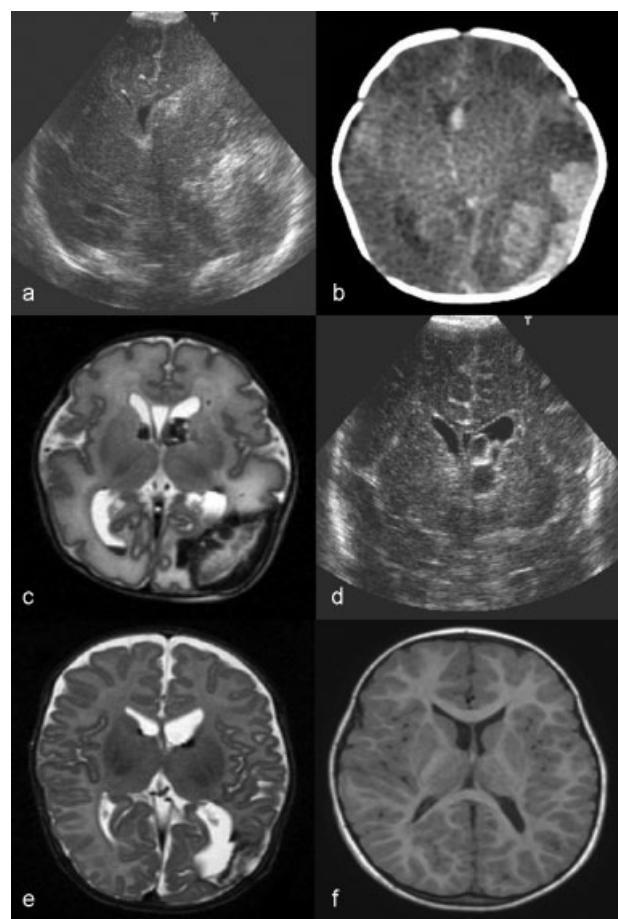


Fig. 1 Ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI) findings of case 1. (A) Cranial ultrasound (cUS) coronal view, showing a clear midline shift, a left-sided germinal matrix hemorrhage (GMH) and a large echogenic area in the left temporal-parietal lobe; (B) Axial CT confirming the midline shift and GMH, but showing both extra-axial and intraparenchymal hemorrhage as well as an area of low attenuation adjacent to the hemorrhage; (C) MRI-T2 sequence performed 8 days after the puncture no longer shows a midline shift, but shows a bilateral intraventricular hemorrhage, a subdural hemorrhage as well as intraparenchymal hemorrhage with adjacent increased signal intensity suggestive of ischemia; (D) cUS performed 2 weeks later showing left-sided residual hemorrhage; (E) MRI-T2 sequence at term equivalent age showing resolution of the hemorrhage with cystic evolution in the parenchyma, separate from the ventricle and mild ventricular dilatation; (F) T1-weighted sequence at 4 years showing normalization of ventricular size and progress in myelination.

circumference on arrival was 27.8 cm ($p = 40$) with a full fontanel and a cUS study revealed a small left germinal matrix-intraventricular hemorrhage (GMH-IVH) with a large focus of increased echogenicity in the left parieto-occipital lobe causing a midline shift, which was later confirmed to be a large parenchymal hemorrhage with a subdural and subarachnoid component on a CT scan (►Fig. 1B). After obtaining parental consent, an ultrasound-guided percutaneous needle aspiration through the parietomastoid suture was performed by the neurosurgeon. A 24-gauge needle was inserted with the guidance of a convex ultrasound probe placed on the anterior fontanel resulting in removal of 20 mL blood. The infant remained stable during the procedure and midline shift was no longer seen on subsequent cUS examinations. The amplitude-integrated electroencephalography (aEEG) showed a normal background pattern for age without seizure activity. Further etiologic studies including alloimmune antibodies, TORCH, and Parvovirus B19 were found to be negative and thrombocytopenia was considered to be due to fetal growth restriction and perinatal asphyxia. An MRI of the brain on day 9 showed no midline shift, presence of bilateral GMHs, and a large left-sided parieto-occipital hematoma communicating with the left lateral ventricle with some blood in the occipital horn. Punctate white matter lesions were also noted together with a left-sided subdural collection (►Fig. 1C); MR angiography and MR venography were unremarkable. cUS examinations demonstrated grade I GMH on the right and grade II IVH on the left (►Fig. 1D). Serial cUS scans showed development of mild posthemorrhagic ventricular dilatation (PHVD), especially on the left. He was started on oral isosorbide (8 g/kg/day) and required three lumbar punctures to stabilize the PHVD. The infant was mechanically ventilated for 9 days followed by continuous positive airway pressure for two more days before weaning to room-air and discharged home on day 36 on isosorbide and phenobarbital maintenance. Isosorbide therapy was discontinued 4 weeks after discharge and a repeat cUS scan showed no dilatation after cessation of the therapy. The follow-up examination at term equivalent age showed a restless, but otherwise well infant and the MR scan showed slightly asymmetrical myelination of the PLIC and mildly decreased size of cerebellar peduncle on the left, partial resolution of the hematoma leading to cystic appearance in the left parieto-occipital lobe, and ex-vacuo dilatation of the left lateral ventricle (►Fig. 1E). The infant received regular physiotherapy and at 6, 12, 18, 24, and 36 months corrected age, his Griffiths Scales of Mental Development (GMD) score was within the normal range. A follow-up MR scan at 4 years of age showed focal white matter loss and gliosis (►Fig. 1F). Follow-up until 5.5 years of age revealed a friendly boy with a small stature (< -2 SD) receiving growth hormone. The infant did not develop cerebral palsy; however, he was diagnosed with developmental coordination disorder and received a special education program for 6 months after which he started and still attends mainstream school.

Case 2

This male infant was born at $39^{4/7}$ weeks to a 34-year-old gravida 2, para 1 mother who had received routine prenatal

care. The infant was born by C/S due to failure to progress in labor. The Apgar scores were 9 and 10 at 1 and 5 minutes, respectively, and umbilical cord blood gas showed a mild metabolic acidosis with a pH of 7.15 and a base excess of -6 mmol/L. His birth weight was 3785 g ($p = 50-90$), head circumference 35.5 cm ($p = 50-90$), and the physical examination was completely normal. At the age of 30 hours, repetitive clonic movements of the left hand and leg were observed and the patient was admitted to the NICU for neuromonitoring. The CBC revealed a hemoglobin of 7.8 mmol/L with a platelet count of 362,000/ μ L and a C-reactive protein of 34 mg/L. Other laboratory studies including basic metabolic panel and coagulation profile were within the normal range. aEEG confirmed the presence of seizure activity in the right hemisphere (►Fig. 2A). cUS revealed a large parenchymal hemorrhage in the right parieto-occipital lobe compressing the right lateral ventricle causing a midline-shift (►Fig. 3A and B). After a loading dose of phenobarbital (20 mg/kg), seizures recurred electrographically and required add-on therapy with midazolam (loading dose: 0.05 mg/kg, followed by 0.15 mg/kg/h), lidocaine (loading dose: 2 mg/kg, followed by continuous infusion for 24 hours, tapered gradually), and levetiracetam (10 mg/kg/dose). An MRI on day 2 showed a massive hemorrhage in the right parieto-occipital lobe with a large occipitoparietal subarachnoid and subdural collection. Hemorrhage was also detected in the right germinal matrix and lateral ventricle (►Fig. 3C and D). MR angiography and MR venography were normal. Bedside ultrasound-guided percutaneous needle aspiration was performed by the neurosurgeon and 7 mL of blood was removed. aEEG after the procedure demonstrated complete cessation of the seizure activity and subsequently the anti-epileptic drugs were tapered (►Fig. 2B and C). Serial cUS studies displayed reduction in the size of the hematoma and disappearance of the midline shift with mild PHVD. Further etiologic evaluation yielded heterozygous carrier state for factor V Leiden mutation. The infant was discharged home on day 12 on phenobarbital maintenance therapy. A follow-up MR scan at 3 months of age showed resolution of the previously noted hematoma, leaving a large arachnoid cyst in the right parieto-occipital lobe and ex-vacuo dilatation of the right lateral ventricle (►Fig. 3E and F). He developed hemianopia of the left eye and a convergent strabismus and otherwise he showed favorable development with a GMD score normal for his age currently at 18 months.

Results

The clinical and outcome characteristics of all eight infants (3 preterm and 5 full-term) are shown in ►Table 1. All infants presented on day 1 to 2 with seizures ($n = 6$) and apneas ($n = 5$), signs of increased intracranial pressure ($n = 4$), and coning ($n = 1$). Six had etiologic risk factors for hemorrhage. cUS and CT showed a midline shift in all (►Fig. 1); two infants showed status epilepticus on aEEG with complete resolution after the procedure (►Fig. 2 of Case 2). All infants were intubated, sedated, and mechanically ventilated during the procedure and between 7 and 34 mL (median 20 mL) could

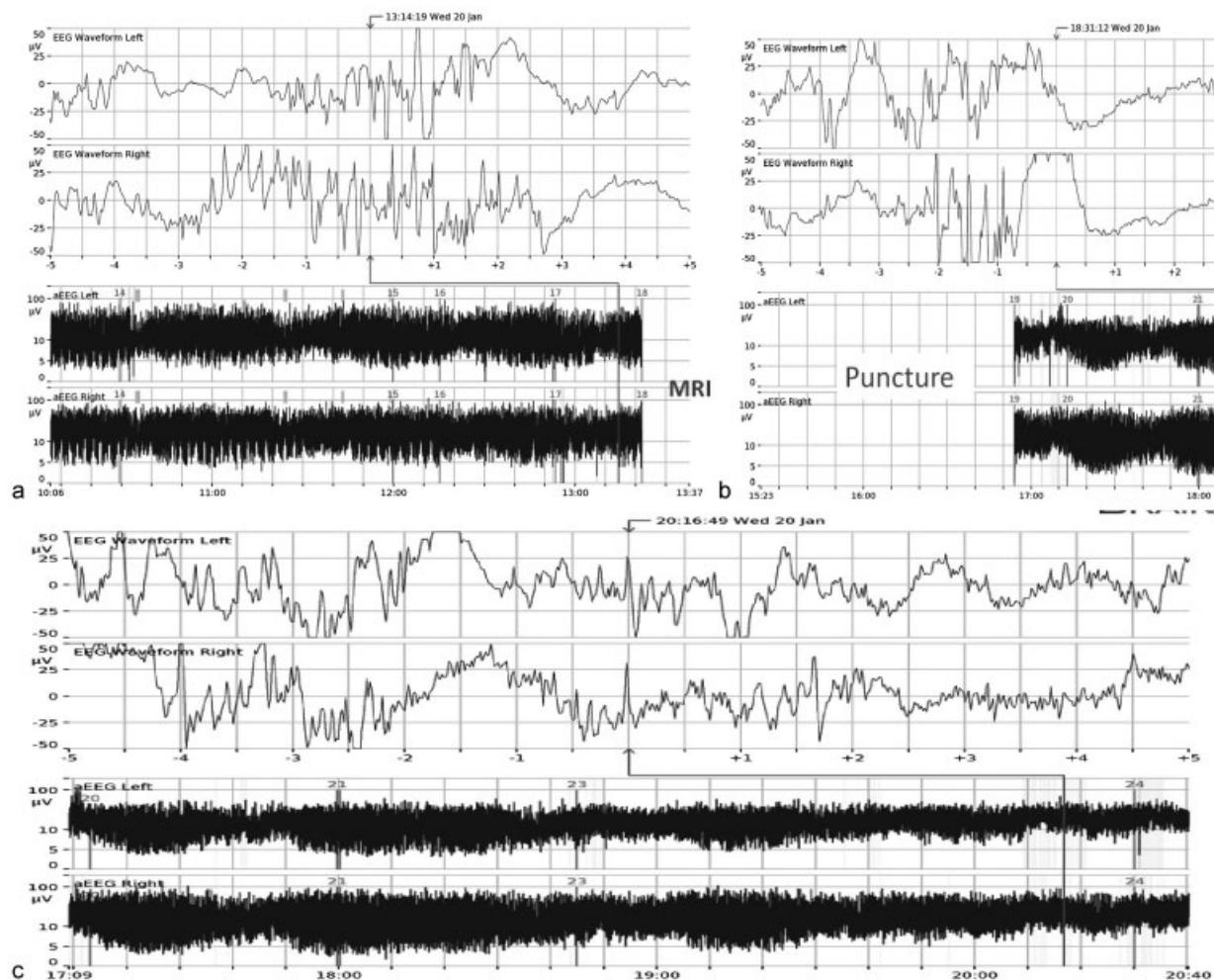


Fig. 2 Amplitude-integrated electroencephalography (aEEG) recording of case 2. (A) Status epilepticus in the right hemisphere refractory to multiple antiepileptic drugs. aEEG recording was interrupted during the magnetic resonance imaging and the puncture; (B) aEEG trace showing cessation of the seizure activity following the procedure on a continuous normal voltage background and presence of sleep–wake cycling; (C) no recurrence of the seizure activity was noted in the following hours, showing continuous normal voltage background and sleep–wake cycling.

be aspirated associated with a decrease in the midline shift as seen by ultrasonography performed during the puncture. No complications were seen related to the procedure and none of the infants required further acute neurosurgical intervention. One infant developed posthemorrhagic hydrocephalus and needed a ventriculoperitoneal shunt (Case 8). Median age at follow-up was 12 months (range 4–168 months). Three had mild sequelae, including motor coordination problems ($n = 1$) and hemianopia ($n = 2$); none developed cerebral palsy or postneonatal epilepsy.

Discussion

ICH is an important cause of brain injury in the neonatal population and despite the substantial advances in perinatal medicine in the past few decades, it continues to be a burdensome health issue in the NICU.² The infants reported in this case series all presented with severe symptoms due to a large intra- and extra-axial cerebral hematoma causing increased intracranial pressure with a midline shift and were managed at

the bedside with an ultrasound-guided needle puncture with an immediate improvement of their symptoms. To the best of our knowledge, this is the first series of newborn infants with a combined intra- and extra-axial hemorrhage presenting with a midline shift managed with this method. Bedside percutaneous needle aspiration is a nonsurgical minimally invasive technique in which the exact site and depth of needle insertion can be determined under ultrasound guidance. Given the major risks of a standard neurosurgical decompression procedure in an unstable neonate, including transfer of the infant to the operating theater, administering general anesthesia and further deterioration of the hemodynamic status, and possible severe changes in cerebral perfusion due to craniotomy, a less invasive method may inevitably be more favorable. Although there are anecdotal reports in the literature on needle aspiration of intracranial hematoma, the cases presented had extra-axial hemorrhages, without intra-axial components (►Table 2).^{5–8,11,12} In 1983, Aoki⁸ reported the association of an epidural hematoma with a depressed skull fracture and in his report, he stated that the successful management of this

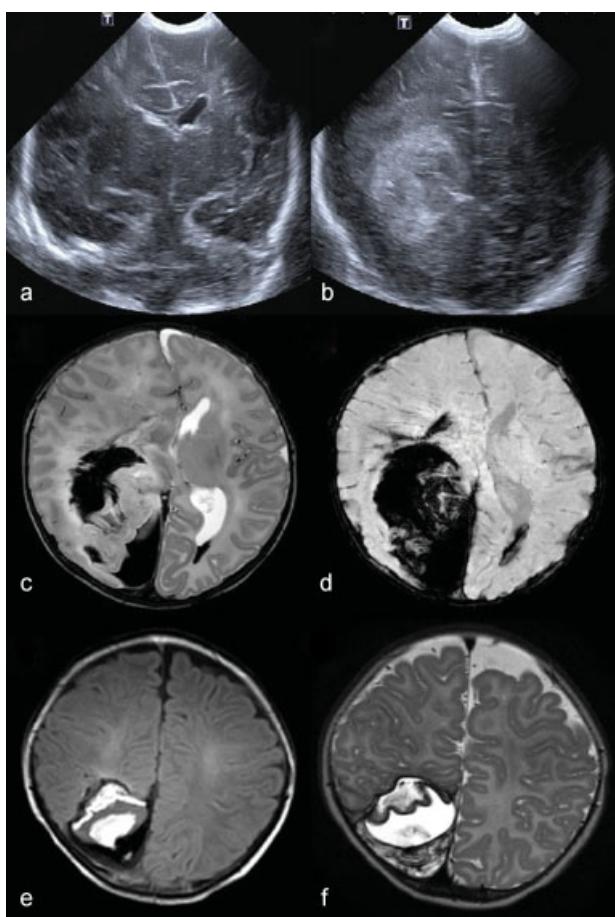


Fig. 3 Ultrasound and magnetic resonance imaging (MRI) findings of case 2. (A) Cranial ultrasound (cUS) coronal view, showing midline shift and a right-sided germinal matrix hemorrhage; (B) cUS image showing a large round-shaped echogenic area in the right parieto-occipital lobe consistent with the hematoma; (C) MRI-T2 sequence performed on day 2 before the procedure shows a midline shift, bilateral intraventricular hemorrhage, and small subdural and large subarachnoid hemorrhage associated with intraparenchymal hemorrhage and ischemia of the parenchyma; (D) MRI-susceptibility weighted imaging at the same level demonstrates a right-sided large intraparenchymal hemorrhage in association with extra-axial blood collection; (E and F) MRI T1 and T2 sequences at 3 months showing evolution of the parenchymal and extra-axial hemorrhage.

condition was achieved by aspiration of the liquefied hematoma rather than surgical removal. Later on, Negishi et al¹¹ and Yamamoto et al¹² reported further successful management of newborn infants with an extra-axial epidural hemorrhage with the aspiration technique and the latter stated that surgical procedures should be attempted only if aspiration turns out to be ineffective. One might wonder whether the thick and semisolid nature of the hematoma could prevent it from being aspirated; however, since the very early reports, it has been noted that the majority of the neonatal intracranial hematomas displayed liquid characteristics.^{6,8-10} Noguchi et al⁶ indicated that neonatal ICH was more liquefied in nature compared with its adult counterpart and showed a tendency to solidify over time, which allows needle aspiration. Consistent with the literature, all of the newborns described in our

series demonstrated a liquefied hematoma, which was easily aspirated. Parents were counseled on the risks, including failure of the technique to evacuate the hematoma, the risk of a new hemorrhage during or after the procedure and further damage to the cerebral tissue; yet none of the aforementioned complications were observed, although in case 8 brain tissue appeared to be part of the aspirated fluid, probably from the damaged occipital lobe due to the intracranial bleeding component. With cUS guidance during the procedure, both the assessment of the tip of the catheter and real-time visualization of the volume of the hematoma could be achieved.⁶ Improvement in the success rate of needle insertion under ultrasonographic guidance would prevent blinded multiple puncture attempts, thus controlling the symptoms more rapidly in a compromised infant, while reducing the risk of coalescence of the needle tracks to form a porencephalic cyst-like lesion and eliminating the risk of vascular injury with the addition of Doppler color flow.¹³ Additionally with cUS, the best puncture site in relation with the nearest suture could be assessed.

Anatomically, ICH is classified as epidural, subdural, subarachnoid, intraventricular, or parenchymal, and involvement of more than one compartment is possible.^{1,14} In the preterm infant, ICH occurs with an estimated incidence of 20% to 25% within the first 3 days after delivery in the form of GMH-IVH and the average incidence of a parenchymal hemorrhage is reported to be between 5% and 11%.^{15,16} However, in the term population, it is difficult to determine its true incidence, since not all term newborns with ICH present with clinical events and it is stated in the literature that asymptomatic ICH in term newborns is much more frequent than previously thought.¹ Looney et al¹⁴ reported the prevalence of asymptomatic ICH after vaginal delivery in term newborns to be as high as 26% in their MRI study. Whitby et al¹⁷ described nine neonates with an asymptomatic subdural hemorrhage and could show birth trauma as the responsible factor in only two of these infants. The authors stated that a subdural hematoma was not necessarily due to an evident birth trauma. Today, it is well-known that in majority of the neonates, ICH occurs spontaneously or related to a clotting defect such as allo- or isoimmune thrombocytopenia, a mutation in the *COL4A1* or *COL4A2* gene, hemorrhagic transformation after arterial stroke, sinovenous thrombosis, especially when associated with a thalamic hemorrhage, or as the result of a vascular malformation.^{1,17-22} The risk of ICH is higher in newborns with hemophilia if they were born by assisted vaginal delivery rather than C/S; whereas von Willebrand disease is not reported to be a common cause of ICH probably due to the physiologic rise in the factor level shortly after birth.^{23,24} We saw thrombocytopenia in two of our cases and in both cases, congenital causes of bleeding diathesis, perinatal infections and alloimmune thrombocytopenia were ruled out. Fetal growth restriction in both cases, as well as the perinatal asphyxia in one, might explain the thrombocytopenia. Dystocia necessitating vacuum extraction could well explain the intracranial events in two of our infants; however, we could not delineate any etiologic explanation for the 2nd and 4th case, although the former demonstrated mild metabolic acidosis and was heterozygous

Table 1 Characteristics of the newborns with intracranial hemorrhage, their clinical features, follow-up aspects, and outcome

Case	Sex	GA	Weight (g)	Mode of delivery	Presenting symptom	Laboratory	Location of the hemorrhage	Amount aspirated (mL)	Follow-up neuroimaging	Etiology	Neurologic Outcome
1	M	31 ^{1/7}	1050	C/S	Respiratory failure, full fontanelle on day 1	Hb:6.5 mmol/L Plt:42,000 μ L PT/APTT:Normal	Left temporal-parietal, left subdural, left GM-IVH (+) midline shift (+)	20	Cystic evolution in the parenchyma, mild PHVD gradually normalizing on MRI	SGA, perinatal asphyxia, thrombocytopenia	Current status: 5.5-year-old, developmental coordination disorder, attending mainstream school
2	M	39 ^{4/7}	3785	C/S	Left-sided, clonic seizures on day 2	Hb:7.8 mmol/L Plt:Normal PT/APTT:Normal	Right parieto-occipital, subarachnoid, subdural, PWML, bilateral GM-IVH (+) midline shift (+)	7	Arachnoid cyst, ex-vacuo dilatation of right ventricle on MRI	Mild acidosis at birth, heterozygous for FV Leiden mutation	Current status: 1.5-year-old, hemianopia on the left eye, convergent strabismus, favorable outcome
3	M	31 ^{6/7}	1285	C/S	Irritability, recurrent apneas, full fontanelle on day 1	Hb:12.8 mmol/L Plt:14,000 μ L PT:22s, aPTT:5.5s	Left temporal and subarachnoid, bilateral GM-IVH (+) midline shift (+)	12	Arachnoid cyst in the left parieto-temporal region on MRI	SGA, arachnoid cyst, thrombocytopenia	Current status: 3-year-old, helmet for plagiocephaly favorable outcome
4	F	41 ^{0/7}	3270	NSD	Right-sided hemiconvulsions on day 1	Normal	Left parietal, left subarachnoid midline shift (+)	24	Residual hemorrhage in the left parietal lobe, mild left ventricular dilatation	Unknown	Current status: 10 months, old, favorable outcome
5	M	37 ^{0/7}	1900	C/S	Feeding intolerance, recurrent apneas, respiratory failure, clonic seizures on day 1	Hb:7.4 mmol/L Plt:Normal PT/APTT:Normal	Right subarachnoid, subdural right temporal lobe diffusion restriction on DWI, midline shift (+)	7	Resolution of the hematoma with volume loss in temporal lobe, ex-vacuo dilatation of right temporal horn on MRI	SGA, vacuum extraction during C/S	Current status: 6 months, old, slightly asymmetrical motor function on lower extremities
6	M	36 ^{6/7}	2555	NSD	Hyperexcitability, hypoglycemia, apnea, seizures on day 1	Hb:9.9 mmol/L Plt:138,000 μ L PT/APTT:Normal	Left subdural, periphery of the cerebellum, midline shift (+)	20	Resolution of the hemorrhage, right posterior cerebral artery infarct, residual right thalamic hemorrhage on MRI	Assisted delivery for breech presentation	Current status: 14-year-old, hemianopia on the right eye, slightly slower motor milestones compared with his twin brother, no cerebral palsy
7	F	38 ^{1/7}	2470	NSD	Recurrent apneas, rightsided hemiconvulsions on day 1	Hb:7.4 mmol/L Plt:161,000 μ L PT:18s, aPTT:38s	Left subdural, ischemia in frontal, parietal and temporal lobes, midline shift (+)	34	Cortical and subcortical abnormalities on DWI, reduction in the size of the hematoma on MRI	Assisted delivery with vacuum extraction	Current status: 6 months, old, cessation of seizures after the procedure, no asymmetry
8	F	40 ^{3/7}	3265	NSD	Recurrent apneas, rightsided hemiconvulsions, bradycardia and high blood pressure on day 2	Hb:7.9 mmol/L Plt:247,000 μ L PT:16s, aPTT:39s	Left subdural, ischemia in frontal, parietal and temporal lobes, midline shift (+)	23	Developed hydrocephalus on MRI	Unknown	Current status: 5 months, old, required a VP-shunt for hydrocephalus

Abbreviations: aPTT, activated partial thromboplastin time; BSID-III, Bayley Scales of Infant Development-III; C/S, caesarean/section; cUS, cranial ultrasonography; DWI, diffusion-weighted imaging; g, gram; F, female; GA, gestational age; GM-IVH, germinal matrix-intraventricular hemorrhage; GMD, Griffiths Mental Development Scales; Hb, hemoglobin; M, male; mo, months; MRI, magnetic resonance imaging; NSD, normal spontaneous delivery; PHVD, posthemorrhagic ventricular dilatation; Plt, platelet; PT, prothrombin time; PWML, punctate white matter lesion; SGA, small for gestational age; VP, ventriculoperitoneal; WML, white matter injury.

Table 2 Characteristics of reported cases in the neonatal literature that were managed with needle aspiration

Author, year	No. of patients	Delivery method	Assist in delivery	Type of hemorrhage	Location of hemorrhage	Other lesions	Ultrasound guidance	Complication after procedure	Outcome
Aoki 1983 ⁸	1	SD	Vacuum	Epidural	Parietal	Cephalhematoma, skull fracture	No	–	Favorable
Negishi et al 1989 ¹¹	3	SD in all	Forceps in 1	Epidural	Parietal in 2, Frontoparietal in 1	Cephalhematoma, skull fracture in all	No	–	Favorable in all
Yamamoto et al 1995 ¹²	1	SD	–	Epidural	Parietal	Cephalhematoma, skull fracture	No	–	Favorable
Vachharajani and Mathur 2002 ⁵	1	C/S	Vacuum prior to C/S	Epidural and PH	Frontal	Subgaleal hemorrhage, depressed skull fracture	Yes	–	Favorable
Vinchon et al 2005 ³	5	SD in all	Vacuum in 2, forceps in 1	Epidural in 1, subdural in 4, PH in 2	Variable	Skin swelling in 3, skull fracture in 1, cheek wound in 1	No	–	Favorable in 4, poor in 1 with severe hypotonia
Noguchi et al 2010 ⁶	1	SD	Vacuum	Epidural	Parietal	Cephalhematoma, depressed skull fracture	Yes	–	Favorable
Smets and Vanhauwaert 2010 ⁷	1	SD	Vacuum	Epidural	Parieto-occipital	Cephalhematoma	No	Required second puncture	Favorable
Present study	8	SD in 4 C/S in 4	Vacuum in 2	PH in 5, GMH-IVH in 3, subarachnoid in 5, subdural in 6	Variable	Midline-shift in all infants	Yes	–	Favorable in 1, mild sequelae in 3, 4 below 12 months old

Abbreviations: C/S, cesarean section; GMH-IVH, germinal matrix hemorrhage-intraventricular hemorrhage; PH, parenchymal hemorrhage; SD, spontaneous delivery; No, number.

for factor V Leiden mutation. Occasionally, a parenchymal hemorrhage is accompanied by a subdural and/or subarachnoid extra-axial component and in most cases, it involves a single hemisphere.^{1,18} This was most prominent in the second and the third case with a large hemorrhage into the arachnoid cyst accompanying the parenchymal hemorrhage (►Table 1). Arachnoid cysts are reported in the literature either as a developmental process or secondary to trauma, hemorrhage and inflammation. Palin et al²⁵ reported a neonate who had ICH after the development of an arachnoid cyst and suggested a reverse causal relationship. Regardless of the primary focus, a therapeutic intervention to remove an intra- or extra-axial hematoma becomes essential when it causes neurologic findings such as seizures and compression of the adjacent cerebral tissues, leading to a midline shift or herniation.^{6,11} We saw seizure activity on aEEG and a midline shift on cUS in all of our cases and dramatic improvement in these findings on serial monitoring after the intervention. Cessation of the status epilepticus was evident in two cases and is noteworthy as a potential major benefit of this bedside intervention.

ICH has also the potential for severe long-term adverse neurological outcomes. Prematurity, presence of perinatal asphyxia, the extent of injury, early onset of repetitive seizures or status epilepticus, and the need for more than one antiepileptic drug to control seizures were associated with an unfavorable outcome.^{1,26} Klinge et al²⁷ showed that ICH is a major risk factor for neurodevelopmental morbidity including psychomotor retardation and cerebral palsy in infants with hemophilia. The newborns in our series presented with one or more of these risk factors; however, we saw a surprisingly favorable clinical outcome during the follow-up of these infants, normal scores for their ages on the neuro-developmental tests, and apparent improvements in their subsequent neuroimaging findings. We speculate that the relatively good outcome of these infants despite the presence of such risk factors might be due to the timely intervention and effective removal of the extra-axial component of these large space-occupying lesions with a minimally invasive method at the bedside without further compromising the clinical condition.

In summary, we described eight newborn infants presenting with overt clinical symptoms including seizure, apnea, and respiratory failure within the first 2 days after birth. All showed a clear midline shift due to an intra- and extra-axial ICH on neuroimaging. We saw a dramatic improvement in the symptoms with this minimally invasive aspiration technique guided by cUS performed at the bedside. We observed no complications during or after the procedure in our infants and the subsequent scans showed disappearance of the midline shift and resolution of the hematoma after the procedure. The neurologic outcome of the infants was rather favorable, all achieving results within the normal range on neurodevelopmental tests.

Conclusion

Bedside ultrasound-guided percutaneous needle aspiration technique should be considered for the management of large

hemorrhages with both intra- and extra-axial component, especially in infants with ongoing seizures, before performing neurosurgical trepanation.

Disclosure

The authors have no financial relationship or any other conflict of interest to declare.

References

- 1 Gupta SN, Kechli AM, Kanamalla US. Intracranial hemorrhage in term newborns: management and outcomes. *Pediatr Neurol* 2009;40(01):1-12
- 2 Inder TE, Perlman JM, Volpe JJ. Preterm intraventricular hemorrhage/posthemorrhagic hydrocephalus. In: Volpe JJ, Inder TE, Darras BT, de Vries LS, du Plessis AJ, Neil J, Perlman JM, eds. *Neurology of the Newborn*. Philadelphia, PA: Elsevier/Saunders; 2017:637-698
- 3 Vinchon M, Pierrat V, Tchofo PJ, Soto-Ares G, Dhellemmes P. Traumatic intracranial hemorrhage in newborns. *Childs Nerv Syst* 2005;21(12):1042-1048
- 4 Bassan H. Intracranial hemorrhage in the preterm infant: understanding it, preventing it. *Clin Perinatol* 2009;36(04):737-762, v
- 5 Vachharajani A, Mathur A. Ultrasound-guided needle aspiration of cranial epidural hematoma in a neonate: treating a rare complication of vacuum extraction. *Am J Perinatol* 2002;19(08):401-404
- 6 Noguchi M, Inamasu J, Kawai F, et al. Ultrasound-guided needle aspiration of epidural hematoma in a neonate after vacuum-assisted delivery. *Childs Nerv Syst* 2010;26(05):713-716
- 7 Smets KJ, Vanhauwaert D. Treatment of cranial epidural hematoma in a neonate by needle aspiration of a communicating cephalhematoma. *Eur J Pediatr* 2010;169(05):617-619
- 8 Aoki N. Epidural hematoma communicating with cephalhematoma in a neonate. *Neurosurgery* 1983;13(01):55-57
- 9 Takagi T, Nagai R, Wakabayashi S, Mizawa I, Hayashi K. Extradural hemorrhage in the newborn as a result of birth trauma. *Childs Brain* 1978;4(05):306-318
- 10 Merry GS, Stuart G. Extradural hematoma in the neonate. Case report. *J Neurosurg* 1979;51(05):713-714
- 11 Negishi H, Lee Y, Itoh K, et al. Nonsurgical management of epidural hematoma in neonates. *Pediatr Neurol* 1989;5(04):253-256
- 12 Yamamoto T, Enomoto T, Nose T. Epidural hematoma associated with cephalohematoma in a neonate—case report. *Neurol Med Chir (Tokyo)* 1995;35(10):749-752
- 13 Brouwer M. Treatment and outcome of neonatal haemorrhagic brain injury [Ph.D. dissertation]. Utrecht: University Medical Centre Utrecht; 2011
- 14 Looney CB, Smith JK, Merck LH, et al. Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. *Radiology* 2007;242(02):535-541
- 15 De Vries LS, Van Haastert IL, Rademaker KJ, Koopman C, Groenendaal F. Ultrasound abnormalities preceding cerebral palsy in high-risk preterm infants. *J Pediatr* 2004;144(06):815-820
- 16 Groenendaal F, Termote JU, van der Heide-Jalving M, van Haastert IC, de Vries LS. Complications affecting preterm neonates from 1991 to 2006: what have we gained? *Acta Paediatr* 2010;99(03):354-358
- 17 Whiby EH, Griffiths PD, Rutter S, et al. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. *Lancet* 2004;363(9412):846-851
- 18 Brouwer AJ, Groenendaal F, Koopman C, Nieuwstein RJ, Han SK, de Vries LS. Intracranial hemorrhage in full-term newborns: a hospital-based cohort study. *Neuroradiology* 2010;52(06):567-576
- 19 de Vries LS, Koopman C, Groenendaal F, et al. COL4A1 mutation in two preterm siblings with antenatal onset of parenchymal hemorrhage. *Ann Neurol* 2009;65(01):12-18
- 20 Carpenter AM, Singh IP, Gandhi CD, Prestigiacomo CJ. Genetic risk factors for spontaneous intracerebral haemorrhage. *Nat Rev Neurol* 2016;12(01):40-49
- 21 Sandberg DI, Lamberti-Pasculli M, Drake JM, Humphreys RP, Rutka JT. Spontaneous intraparenchymal hemorrhage in full-term neonates. *Neurosurgery* 2001;48(05):1042-1048, discussion 1048-1049
- 22 Wu YW, Hamrick SE, Miller SP, et al. Intraventricular hemorrhage in term neonates caused by sinovenous thrombosis. *Ann Neurol* 2003;54(01):123-126
- 23 MacLean PE, Fijnvandraat K, Beijleveldt M, Peters M. The impact of unaware carriership on the clinical presentation of haemophilia. *Haemophilia* 2004;10(05):560-564
- 24 Chalmers EA. Neonatal coagulation problems. *Arch Dis Child Fetal Neonatal Ed* 2004;89(06):F475-F478
- 25 Palin M, Anderson I, O'Reilly G, Goodden JR. A suprasellar arachnoid cyst resulting from an intraventricular haemorrhage and showing complete resolution following endoscopic fenestration. *BMJ Case Rep* 2015;2015:1
- 26 Pisani F, Cerminali C, Fusco C, Sisti L. Neonatal status epilepticus vs recurrent neonatal seizures: clinical findings and outcome. *Neurology* 2007;69(23):2177-2185
- 27 Klinge J, Auberger K, Auerswald G, Brackmann HH, Mauz-Körholz C, Kreuz W. Prevalence and outcome of intracranial haemorrhage in haemophiliacs—a survey of the paediatric group of the German Society of Thrombosis and Haemostasis (GTH). *Eur J Pediatr* 1999;158(Suppl 3):S162-S165