Cerebellar Hemorrhage in the Term Neonate: Developmental and Neurologic Outcome

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To elucidate the effects of cerebellar hemorrhage on the term neonate, neurodevelopmental assessments were conducted at a mean age of 32 months on six children. In addition to cerebellar hemorrhage, ventriculomegaly was present on each subject's initial computed tomographic scan. All were managed without surgical evacuation. Two patients required shunts for progressive ventriculomegaly. Five patients had follow-up computed tomography indicating mild atrophy of the superior anterior vermis of the cerebellum; however, none had abnormal ventricular size or abnormalities of the cerebrum. On detailed examination conducted between the ages of 18 and 48 months, five had hypotonia, truncal ataxia, and intention tremor; two had nystagmus. Only one patient walked independently. Intellectual performance of four patients was within the retarded range and two had mildly delayed development. Two patients had markedly disordered expressive language. These data suggest that term neonates surviving cerebellar hemorrhage have neurologic deficits related to the site of hemorrhage, and cognitive deficits related to more generalized cerebral insult.

Williamson WD, Percy AK, Fishman MA, Cheek WR, Desmond MM, LaFevers N, Thurber SD. Cerebellar hemorrhage in the term neonate: Developmental and neurologic outcome. Pediat Neurol 1985;1:356-60.

Introduction

The use of computed tomography (CT) has made possible the antemortem diagnosis of cerebellar hemorrhage in the neonate [1]. Early diagnosis has provoked discussion regarding therapy; both surgical and medical managements reportedly have been successful in promoting increased survival [2-6]. As a result, attention is now being directed at the neurodevelopmental progress of survivors. Detailed data describing long-term outcome are needed in order to compare therapeutic regimens and to provide parents with realistic prognoses.

The present report describes the neurologic and developmental status of six children, at a mean age of 32 months, whose neonatal cerebellar hemorrhages were managed medically.

Subjects and Methods

Detailed perinatal data are shown in Table 1. Patients 1-5 were born in Houston community hospitals and transferred to the Neonatal Intensive Care Unit, Texas Children's Hospital during the first 48 hours of life after exhibiting the central nervous system abnormalities described below. Patient 6 was born at a community hospital in another city, experienced clinical deterioration at five hours of age, and then was transferred to a Level III nursery for management.

Initial CT, performed in each patient between 12 and 48 hours of age, documented the cerebellar hemorrhage and associated findings. The presence of intraparenchymal cerebellar hemorrhage was defined by the following criteria: subtentorial location, posterior to the fourth ventricle, and evidence of neural tissue surrounding the hemorrhage (Fig 1).

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Presented, in part, at The Society for Pediatric Research; San Francisco, CA; May 1984

Received August 12, 1985; accepted September 20, 1985.

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Received July 9, 1985; accepted October 1, 1985

Table 1. Perinatal information for six neonates with cerebellar hemorrhage

Patient No.	Birth Wt (grams)	Gest Age (weeks)	Prenatal Risk Factor	Natal Factors	Apgar Scores 1 min/5 min	Status on Delivery
1	4100	40	Class A diabetes	Placenta previa, emergency C-section	9/9	Meconium stained nails, cord bilirubin 3.7 mg/dl, Coombs positive
2	2977	39	None	Elective low forceps	9/9	Normal
3	3174	37	Intermittent vaginal bleeding kept at bed rest	Rapid labor (1 hr 15 min); footling breech	8/10	Normal
4	3300	40	None	Prolonged second stage (2 hrs)	9/10	Normal
5	2807	39	Decreased fetal activity by maternal report	Frank breech, dark brown amniotic fluid	5/7	Pale, weak cry, normal tone, mask O_2, Hct 36.6%
6	2070	37	Pre-eclampsia, IUGR	Rapid labor, drop in fetal heart tones, frank breech, battledore placenta	6/8	Pale, hypotonic, mask O_2 given

Clinical course and management were as follows: Patient 1 experienced an apneic episode at 8 hours of age followed by hypertonia and lethargy. Initial CT scan revealed a midline cerebellar hematoma extending into both cerebellar hemispheres, blood in the extra-axial space, and marked lateral ventriculomegaly. Because of apneic episodes, mechanical ventilation was required for 24 hours. Medical management included treatment with dexamethasone, acetazolamide, and glycerol. For progressive ventriculomegaly, a ventricular drain was placed on day 7 and a ventriculoperitoneal shunt on day 24. At discharge on day 32 the patient was hypotonic and lethargic.

Patient 2 had an apneic episode with cyanosis at 36 hours of age and a tense anterior fontanel was palpated. CT scan findings included a midline cerebellar hematoma with extension into both hemispheres, blood in the interhemispheric spaces and moderate ventriculomegaly. Dexamethasone and acetazolamide were given initially. Progressive ventriculomegaly required ventriculoperitoneal shunting on day 20, one week prior to discharge.

Patient 3 had an apneic episode with cyanosis and extensor posturing of the extremities at 24 hours of age. CT scan findings included a midline cerebellar hematoma with surrounding edema, blood in the interhemispheric sulcus, and mild ventricular dilatation. Oxygen was given by mask but mechanical ventilation was not required. Intermittent apneic and bradycardic episodes resolved spontaneously. Dexamethasone was administered for 10 days. At discharge on day 11, extensor posturing and uncoordinated suck were evident.

At 12 hours of age Patient 4 developed cyanosis with feeding followed by bradycardia, lethargy, full fontanel, and extensor posturing. Initial CT scan demonstrated a midline cerebellar hematoma extending into the right hemisphere, blood in the extra-

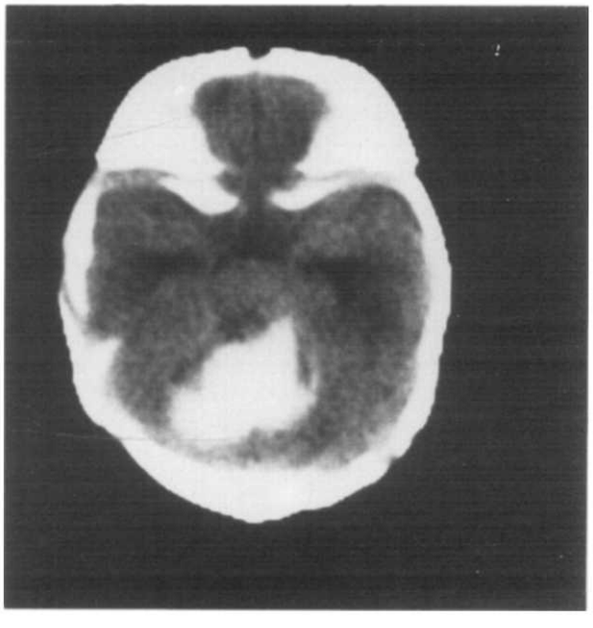

Figure 1. Unenhanced CT of a 36-hour-old term infant, demonstrating a high density lesion, interpreted as intraparenchymal cerebellar hemorrhage because of its subtentorial location, posterior to the fourth ventricle with evidence of neural tissue surrounding the hemorrhage.

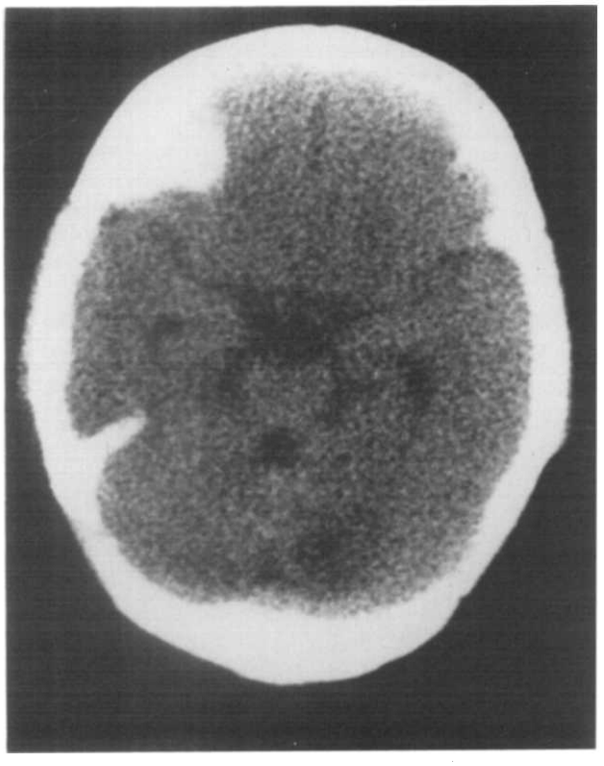

Figure 2. Unenhanced CT of a 2-month-old infant with a previously diagnosed neonatal cerebellar hemorrhage, now revealing a low density lesion in the left cerebellar hemisphere and mildly enlarged fourth ventricle suggesting loss of brain substance.

axial spaces, and moderate enlargement of the third and lateral ventricles. Separated sutures with a full fontanel were present on day 2. Following elective intubation, positive pressure ventilation was used for 4 days. Dexamethasone and glycerol also were given. The patient was discharged on day 13.

Patient 5 became pale and experienced bradycardia at 1 hour of age accompanied by a generalized seizure and nystagmoid eye movements. A CT scan indicated a midline cerebellar hematoma, a small hemorrhage in the interhemispheric sulcus and the third and lateral ventricles, and moderate ventriculomegaly. Occasional bradycardic and apneic episodes with extensor posturing required no mechanical ventilation. Dexamethasone was used for 1 day. Mild hypotonia was present at discharge on day 10.

Patient 6 had a generalized seizure with apnea and cyanosis at 5 hours of age. A full fontanel was palpable. A CT scan delineated a midline cerebellar hematoma, hemorrhage into the right occipital lobe, and moderate dilatation of the lateral ventricles. Mechanical ventilation was required for 2 days for recurrent apnea. The patient received phenobarbital, remained seizure-free after day 1, and was discharged on day 13.

Each of the six patients had one or more follow-up CT scans performed between the ages of 2 months and 3 years. Patient 3 had a radiolucent area in the cerebellum (Fig 2) and Patients 1-5 had evidence of mild atrophy of the superior anterior vermis. All had normal ventricular size. No patient had evidence of parenchymal abnormality of the cerebral hemispheres on follow-up scan.

All patients were seen in the Meyer Center for Developmental Pediatrics, Texas Children's Hospital for neurologic examination and multidisciplinary developmental assessment. The examiners performing the assessments were unaware of the exact location of lesions on CT. Cognitive functioning was assessed using the Bayley Scales of Infant Development (for those younger than 30 months) or the McCarthy Scales of Children's Abilities (for those older than 30 months) [7,8]. The following test battery was used to assess speech and language skills, depending upon chronologic age and developmental functioning: Peabody Picture Vocabulary Test, Expressive One Word Picture Vocabulary Test, Preschool Language Scale, Sequenced Inventory of Communication Development and clinical assessment of articulation, voice quality and fluency [9-12].

Results

The six children were evaluated at a mean age of 32 months (range: 18 to 48 months). Developmental and neurologic findings are noted in Table 2. Abnormal neurologic signs included hypotonia, truncal ataxia, intention tremor, and nystagmus. At the time of evaluation, only Patient 6 ambulated without aid; Patients 2-5 ambulated with aids. Patient 1 had not ambulated. Cognitive abilities were markedly impaired in 4 patients (>2 SD below mean) and mildly delayed (between 1 and 2 SD) in two patients.

Language comprehension was commensurate with level of cognition in all patients. Patients 4,5, and 6 had developmental skills sufficient to allow detailed assessments of expressive language and connected speech. Patient 6 had no expressive language delays or disorders. However, Patients 4 and 5 demonstrated marked disorders of expressive language which included dysarticulation, alterations in word and syllabic stress, dysarthric syllable repetition, slow rate, breathy or harsh voice quality, and monotony of pitch and loudness.

Discussion

Until the advent of CT, cerebellar hemorrhage in the term neonate was diagnosed primarily at autopsy [1]. As exemplified in these six term neonates, cerebellar hemorrhage now can be documented by CT, usually done to investigate the cause of central nervous system dysfunction. After delivery, a quiescent period of normality or mild, nonspecific findings (pallor, lethargy) is followed at 1 to 36 hours by an acute episode of clinical deterioration (apnea, seizures, full fontanel) suggesting central nervous system involvement. Initial CT documents not only the cerebellar hematoma, but also the early onset of ventriculomegaly.

Risk factors for cerebellar hemorrhage in term infants probably are similar to those described by Volpe for

Table 2. Developmental and neurologic outcome of evaluation between 18 and 48 months of age

Patient No.	Age Last Evaluated (Months)	MDI*/GCI†	Neurologic Findings			
			Hypotonia	Truncal Ataxia	Intention Tremor	Nystagmus
1	18	40	+ +	+	+	+
2	29	56	+ +	+	+	—
3	21	53	—	+	+	—
4	32	75	+ +	+	+	—
5	48	52	+ +	+	+	+
6	44	84	+	—	—	—

*MDI = Mental Developmental Index, Bayley Scales of Infant Development
†GCI = General Cognitive Index, McCarthy Scales of Children's Abilities

subdural hemorrhage [13]. These may include maternal (primagravida, small birth canal), fetal (large head), labor (prolonged or precipitous), or delivery (breech, difficult, or forceps) characteristics. The mechanism is assumed to be a compressive force to the occiput which causes a contusion or laceration of the underlying cerebellum [1]. Injury or obstruction to the venous system draining this area also may occur [6]. Occipital diastasis has been associated with such lesions, although usually this finding is not detected until autopsy [14].

Cerebellar hemorrhage traditionally has been managed by neurosurgical evacuation of the hematoma [2-4]. Although survival rates of patients receiving such management apparently have been good, information on the survivors' subsequent neurologic and developmental function is limited. Scotti described varying combinations of "quadriplegia", "hemiparesis", and "mental retardation" in four patients; however, few details were provided concerning the functional skills of the patients or their ranges of intellectual performance [4].

In the present series of patients managed medically, the disturbances of tone, balance, and coordination are compatible with a significant cerebellar insult [15]. The disorders of fluency, phonation, and articulation found in two patients are also compatible with lesions in the cerebellum, although such disorders characteristically have been reported in older patients with traumatic or degenerative lesions of the cerebellum [16].

In contrast, the impairment in intellectual functioning of this patient group reflects a generalized cerebral insult rather than an isolated cerebellar lesion. A review of each subject's history reveals risk factors possibly related to generalized impairment (Table 1) [17-20]. These factors include intrauterine growth retardation, apnea, seizures, and need for mechanical ventilation. All subjects had, in addition to the cerebellar hemorrhage, abnormalities on initial CT. Patient 6 had an intracerebral hematoma and all had ventriculomegaly. Such neuroradiologic findings, at least in the premature infant with intraventricular hemorrhage, may be associated with impaired outcome [19,20]. Based on the lack of either severely depressed Apgar scores or need for active resuscitation in the delivery room, there is no clear evidence that these subjects experienced birth asphyxia. However, it is possible that subsequent hypoxic-ischemic episodes, coupled with the above-mentioned risk factors, played a role in producing the generalized cerebral insult resulting in intellectual impairment.

From this series of patients managed medically, as well as from previous reports of neurosurgically managed patients, the neurologic and developmental outcomes for term neonates with documented cerebellar hemorrhage reveal significant disabilities. Poor outcomes associated with this lesion are contrary to the relatively good outcome reported for infants with posterior fossa subdural hematomas [4,21]. This

contrast underscores the importance of differentiating the two lesions. Discussions of prognosis with the family regarding cerebellar hemorrhage in the term neonate should include a careful consideration of this information. Perhaps the patients thus far reported represent the more severe end of the spectrum since their cerebellar hemorrhages were diagnosed subsequent to significant neurologic disturbances in the neonatal period and their outcomes suggest an accompanying generalized cerebral insult. Neonates with less significant and isolated cerebellar injury may go undetected, only to be found at a later age to be clumsy or to have unexplained minor abnormalities on neurologic examination [22,23]. Further follow-up of the patients in this report is needed to determine the evolution of their motor and language disturbances.

The authors thank the Meyer Center staff for their participation in this study, Dr. Paul Gerson for reviewing the CT scans, Dr. Geraldine Wilson for reviewing this manuscript, and Lou Peterson for manuscript preparation. Supported in part by MCT No. 000,436.

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