AJNR

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AJNR Am J Neuroradiol 1987, 8 (2) 291-295
http://www.ajnr.org/content/8/2/291

Periventricular Cavitations in the First Week of Life

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Eleven infants were encountered (nine premature, two term) in whom well-defined small periventricular cavitations were found by sonography in the first week of life. The sonographic findings bore remarkable similarity to subependymal pseudocysts in neonates previously described in autopsy specimens. The cavitations, which were identified predominantly along the superolateral aspects of the lateral ventricles, did not evolve in the manner of postnatally acquired periventricular leukomalacia. The location of the cavitations differed from the site of previously reported lesions of posthemorrhagic and postinfectious germinolysis along the medial aspect of the caudothalamic groove. Neurosonologists and neonatologists should be alerted to this finding and encouraged to follow these infants as a separate group to learn whether neurodevelopmental sequelae occur in these children.

The development of periventricular cavitations has been well documented by sonography in neonates and infants with periventricular leukomalacia (PVL) secondary to perinatal and postnatal hypoxic-ischemic brain injury [1–3]. In PVL, which has been shown to be infarction in the periventricular white matter of the brain [4–7], cavitation typically occurs 2–4 weeks after the precipitating insult [1–3]. Subependymal germinal cysts, another cystic encephalopathy, may evolve over several weeks in the course of resolving subependymal hemorrhage, or it may be found at birth in infants with congenital neurotropic infection without hemorrhage [8–12].

We report on 11 infants in whom periventricular cavities, predominantly along the superolateral aspects of the lateral ventricles, were noted by cranial sonography within the first week of life. Such early sonographic findings along with preliminary clinical and sonographic follow-up has not, to our knowledge, been previously reported.

Materials and Methods

Sonograms and medical records were reviewed in 11 infants who exhibited periventricular cavitations in the first week of life. Sonograms, performed with commercially available real-time scanners using 5.0-, 6.0-, and 7.5-MHz transducers, were obtained through the anterior fontanelle using coronal and parasagittal views. The rarity of these findings was evidenced by the small number of infants identified in the pooled neurosonographic experience of four neonatal centers over a 3½ year period from January 1983 to June 1986.

Results

Clinical and sonographic findings of week-old infants with periventricular cavitations are summarized in Tables 1 and 2.

Cavities were predominantly found along the lateral aspects of the frontal horns and/or bodies of the lateral ventricles (Figs. 1–3). Lesions, most often ovoid in shape, occurred singly or in linear clusters adjacent to the lateral ventricles.

Received June 30, 1986; accepted after revision September 22, 1986.

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AJNR 8:291–295, March/April 1987
0195-6108/87/0802-0291

TABLE 1: Summary of Clinical Findings in Infants with Periventricular Cavitations

Case No.	Gestation (weeks)	Birthweight (g)	Maternal History	Apgar Scores at 1, 5 min	Respiratory Status	Neurologic Findings	
						Neonatal Period	Postneonatal Period
1	32	1020	Preeclampsia CS	2, 8	Mild RDS	Normal	Lost to F/U
2	34	2070	None	4, 6	Mild RDS	Abnormal EEG	Normal at 7 mo; developmental delay and seizures at 16 mo
3	28	830	None	8, 9	RDS	Normal	Normal at 2 mo
4	32	1600	Urinary infection	9, 9	Mild RDS	Normal	Normal at 4 mo
5	31	1465	None	7, 9	Normal	Normal	Normal at 4 mo
6	40	3300	Intermittent leg and pedal edema	7, 9	Normal	Hypotonia, abnormal EEG (right parietal)	Lost to F/U
7	25	700	Twins	1, 6	RDS	Normal	Normal at 6 mo
8	40	3300	CS for fetal distress; nuchal cord	1, 3	PFC	Normal	Died at 11 days
9	31	1375	Seizures; CS for bleeding and fetal distress	1, 4	RDS	Normal	Normal at 2 mo
10	30	1760	None	7, 7	Mild RDS	Normal	Normal at 2 mo
11	29	1440	Meconium-tinged amniotic fluid	6, 7	RDS, PIE	Normal	Normal at 2 mo

Note.—CS = cesarean section, RDS = respiratory distress syndrome, F/U = follow-up, PFC = persistent fetal circulation, PIE = pulmonary interstitial emphysema.

TABLE 2: Sonographic Findings in Infants with Periventricular Cavitations

Case No.	Gestation (weeks)	Birthweight (g)	Early Sonography (<1 week)		Late Sonography (>3 months)	Ventricular Size
			Periventricular location of cavitations	Intracranial hemorrhage		
1	32	1020	Left frontal horn	None	Lost to F/U	Normal
2	34	2070	Bilateral frontal horns	None	Normal at 6 mo	Normal
3	28	830	Body of left lateral ventricle, left frontal horn	None	Lost to F/U	Normal
4	32	1600	Left frontal horn	Left SEH	Normal at 6 mo	Normal
5	31	1465	Bilateral frontal horns	None	Normal at 4 mo	Normal
6	40	3300	Left frontal horn	Left SEH/choroid plexus hemorrhage	Lost to F/U	Mild enlargement of left frontal horn and body
7	25	700	Bilateral frontal horns	None	Two tiny cavitations identified at 6 mo	Normal
8	40	3300	Bilateral frontal horns and bodies of ventricles	None	Died; no autopsy	Normal
9	31	1375	Bilateral frontal horns	Bilateral SEH/IVH	Lost to F/U	Mildly enlarged initially; normal at 1 mo
10	30	1760	Bilateral frontal horns	None	Lost to F/U	Normal
11	29	1440	Bilateral frontal horns	Bilateral SEH, small left IVH	Lost to F/U	Normal initially; mildly enlarged at 2 mo

Note.—F/U = follow-up, SEH = subependymal hemorrhage, IVH = intraventricular hemorrhage.

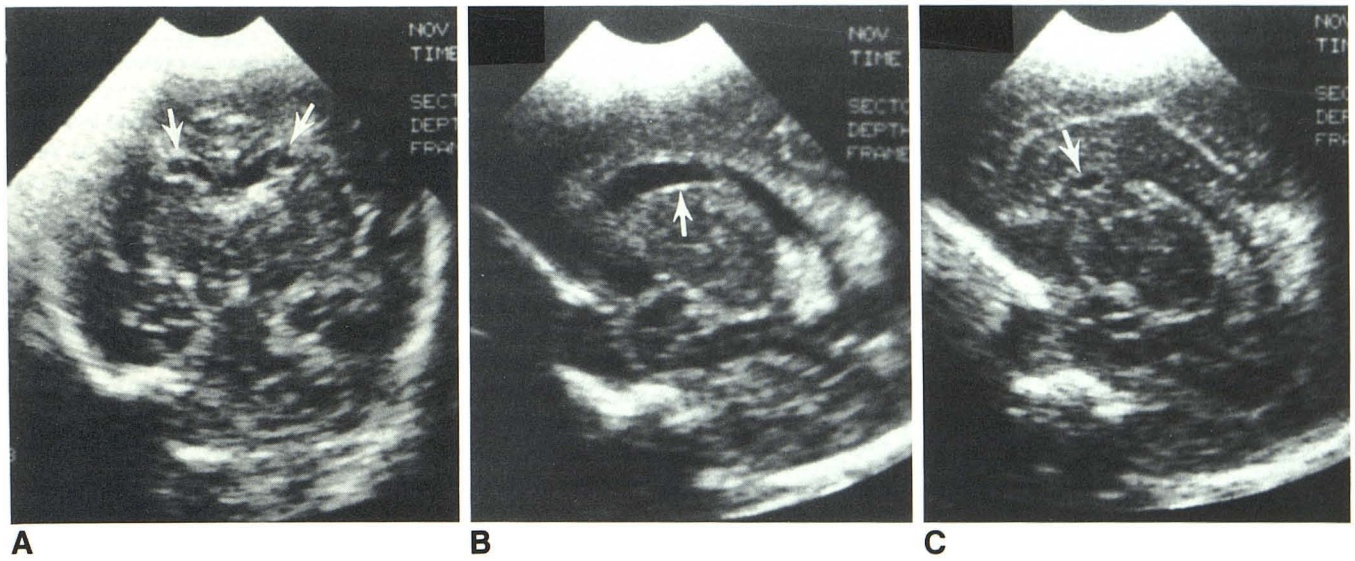

Fig. 1.—Case 2. Coronal semiaxial (*A*), left (*B*) and right (*C*) parasagittal sonograms show bifrontal periventricular cavitations (*arrows*).

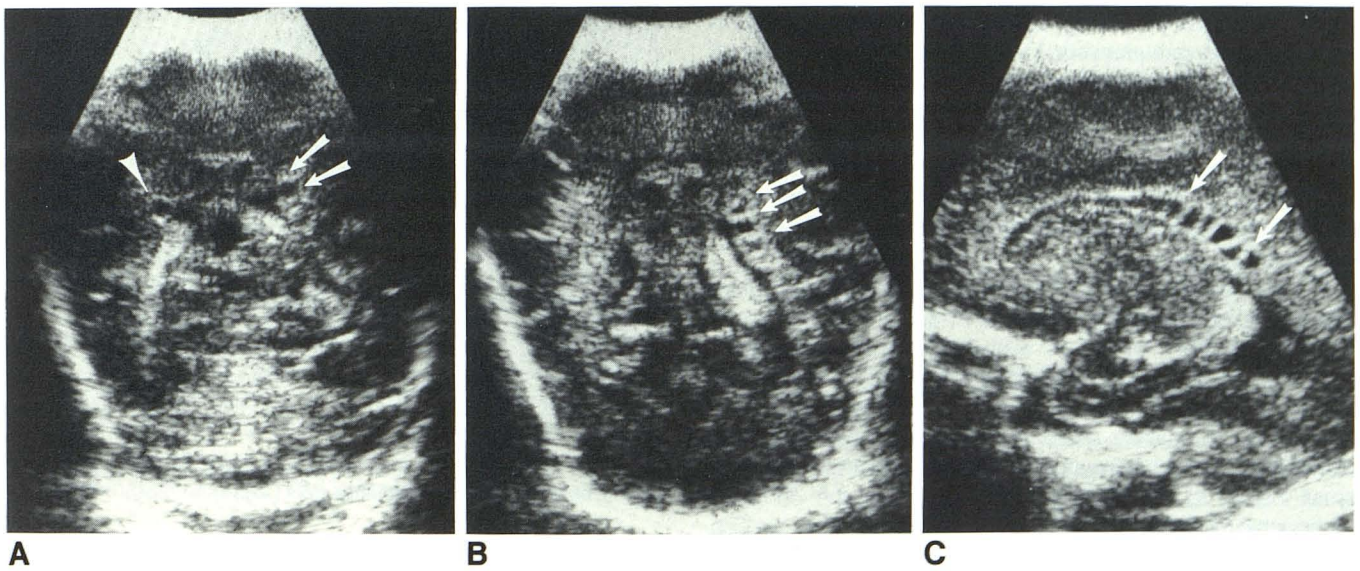

Fig. 2.—Case 6. Coronal semiaxial (*A* and *B*) and left parasagittal (*C*) sonograms show solitary right frontal cavitation (*arrowhead*) and linear cluster of polygonal cavitations along frontal horn and body of left lateral ventricle (*arrows*).

Occasionally, polygonal cavitations were grouped in linear clusters.

While some of the infants had notable events in their gestational histories, others did not. No prenatal or perinatal clinical pattern emerged from the sonographic findings in this small series.

Of the four infants who had follow-up studies, three (cases 2, 4, and 5) were seen at 6, 6, and 4 months of age, respectively, with normal neurosonograms. One infant (case 2) was examined at 16 months and found to have developmental delay and a seizure disorder but neither spasticity nor a vision disorder. Two infants (cases 4 and 5) appeared normal at 6 and 4 months of age. One infant with periventricular cavitations (case 7) had a twin who did not have them. At 6 months, both infants were developmentally normal, and the follow-up neurosonogram of the smaller twin with initially abnormal scans revealed two barely perceptible tiny residual cavitations along the lateral aspect of each frontal horn (Fig. 3C).

Six of the infants were lost to follow-up. One infant died, but permission for autopsy could not be obtained.

Discussion

The periventricular cavitations in our patients bear a remarkable similarity to some of those found in a group of 22

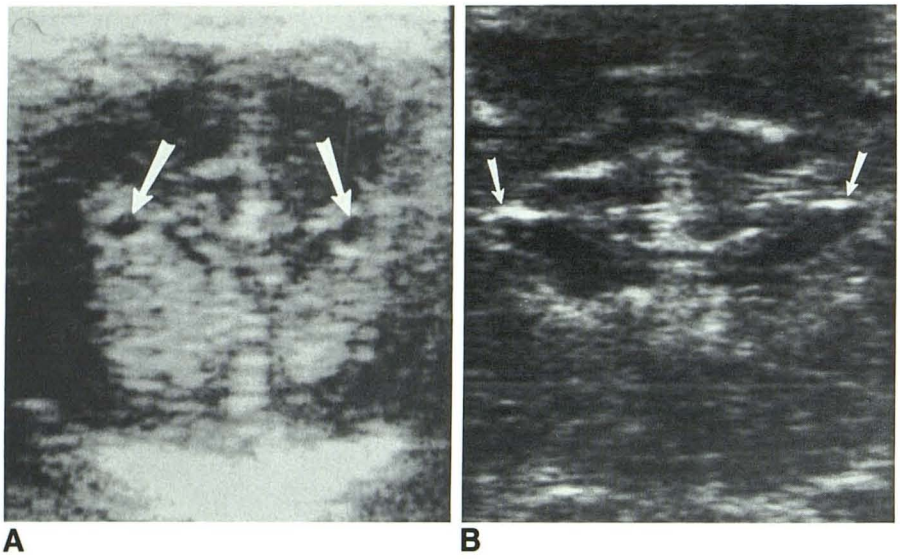

Fig. 3.—Case 7. Coronal sonograms.
***A*, Bifrontal cavitations (*arrows*).**
***B*, Follow-up coronal sonogram at age 6 months shows tiny residual cavitations (*arrows*).**

infants autopsied and described by Larroche [13]. Ten of those infants were 2–3 days old, and 11 of the 22 cases had subependymal pseudocysts located lateral to the external angles of the lateral ventricles near the coronal plane of the foramina of Monro similar to those of most infants in our series (Fig. 4). Four of the 17 cases in Larroche's series involved the subependymal germinal matrix adjacent to the caudate nuclei and had evidence of previous hemorrhage. The subependymal portions of the occipital lobes were involved in two cases.

In early gestation the germinal layer abundantly surrounds the lateral ventricles until 30 weeks of gestation, when involution begins [14]. By 32–34 weeks, the germinal matrix is limited to the area medial to the caudate heads. By 40 weeks, the germinal matrix is largely involuted [13]. Because of the occurrence of similarly sized cavitations in a periventricular distribution, Larroche grouped all cases together and ascribed small insults to the fetal germinal matrix as the common pathogenic mechanism. Larroche admitted that some cavities in her series were lined with macrophages, implying malacic change, while in four other cases, positive tests for iron pigment suggested resolved subependymal hemorrhages. Thus, her case population appeared heterogeneous.

Sonographic findings of subependymal germinal matrix hemorrhages evolving into subependymal cysts along the medial aspect of the caudothalamic groove have been described [8, 10]. Congenital neurotropic infection may result in similarly located subependymal cysts [8–12] sonographically indistinguishable from posthemorrhagic subependymal cysts but without a documented prior hemorrhage [8]. Among pathologists, the cause of cysts of the matrix zone has been a subject of debate [15, 16]. While some of Larroche's cases exhibited subependymal cysts medial to the caudate nuclei, none were found in our patients who had no clinical or serologic evidence for congenital infection and who were too young to have cavitation within a neonatal subependymal hemorrhage. Nevertheless, it is still possible that both the cavitations and the high rate of prematurity in our series represent sequelae of an unidentified intrauterine infection or insult.

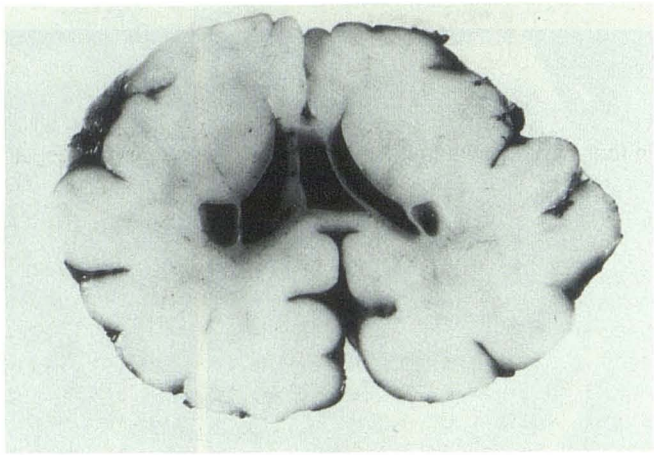

Fig. 4.—Coronal brain section shows two subependymal pseudocysts as described by Larroche. (Reprinted from [13].)

Periventricular cysts may be a manifestation of PVL, and the significant neurodevelopmental sequelae of PVL have been described [5, 7, 17]. Early recognition of PVL by sonography helps to provide health care personnel with the ability to counsel parents about an infant's guarded neurologic outlook.

The pathogenesis and evolution of PVL is thought to relate to the circulation in the periventricular cerebral white matter [5, 18]. In premature infants, the periventricular regions are watersheds between ventriculopetal and ventriculofugal circulations. These areas are therefore susceptible to infarct in situations causing hypoxia and ischemia [5, 18]. The earliest sonographic finding in PVL is irregular, coarse periventricular hyperechogenicity [1–3]. Over 2–4 weeks, the infarcted brain develops cavitations that can be seen both sonographically

and pathologically [1–7]. As PVL often involves the long motor tracts and optic radiations, affected infants commonly exhibit varying degrees of spasticity and cortical blindness [7, 17].

The infants in our series did not appear to have PVL as all had well-defined periventricular cavitations within the first week of life, and none of the four infants seen in follow-up had any neurologic abnormality associated with PVL. Even though several of our patients had cavitations in or near areas often involved by PVL, there was never any extension into the centrum semiovale. Moreover, the early sonographic detection of such well-defined cavities would preclude postnatally acquired PVL.

Difficulty with interpretation of both the data of Larroche [13] and of our study is caused by sample bias. Larroche's autopsy study was performed before the advent of any high-resolution brain imaging, and all infants obviously had fatal illnesses. Our patients were identified because of clinical requests for neurosonography in infants with either prematurity, low Apgar scores, or possible neurologic abnormality. Therefore, one cannot comment on the prevalence of these findings in infants but can state only that these findings are rare even in a group of selected high-risk infants. All but one of our 11 patients have survived, so a histologic comparison with Larroche's cases is not possible.

In our limited experience, the postnatal history of the cavitations appears to be resolution, as follow-up examinations in four of our patients have enabled us to see periventricular cavitations disappear in three infants and diminish in size and number in the other (Fig. 3C).

We call attention to small, lateral periventricular cavitations found in the first week of life. Whether they represent prenatal ischemia or infection with development of small areas of leukomalacia, or whether they represent degeneration of periventricular germinal matrix in early gestation, or whether they reflect a multiplicity of causes cannot be determined in this report. Neurosonologists and neonatologists should be aware that well-defined, small periventricular cavitations seen in infants in the first week of life is separate and distinguishable from postnatally acquired PVL. Identification of these infants as a separate group will enable long-term neurodevelopmental follow-up that may ultimately give predictive clinical value to this finding.

REFERENCES

1. Bowerman RA, Donn SM, DiPietro MA, D'Amato CJ, Hicks SP. Periventricular leukomalacia in the pre-term newborn infant: sonographic and clinical features. *Radiology* **1984**;151:383–388
2. Schellinger D, Grant EG, Richardson JD. Cystic periventricular leukomalacia: sonographic and CT findings. *AJNR* **1984**;5:439–445
3. Schellinger D, Grant EG, Richardson JD. Neonatal leukencephalopathy: common form of cerebral ischemia. *Radiographics* **1985**;5:221–242
4. Banker BQ, Larroche JC. Periventricular leukomalacia of infancy. *Arch Neurol* **1962**;7:386–410
5. DeReuck J, Chatta AS, Richardson EP Jr. Pathogenesis and evolution of periventricular leukomalacia in infancy. *Arch Neurol* **1972**;27:229–236
6. Leech RW, Alvord EC Jr. Morphologic variations in periventricular leukomalacia. *Am J Pathol* **1974**;74:591–602
7. Shuman RM, Selednik LJ. Periventricular leukomalacia. A one-year autopsy study. *Arch Neurol* **1980**;37:231–235
8. Shackleford GD, Fulling KH, Glasier CM. Cysts of the subependymal germinal matrix: sonographic demonstration with pathologic correlation. *Radiology* **1983**;149:117–121
9. Clair MR, Zalneraitis EL, Baim RS, Goodman K, Perkes EA. Neurosonographic recognition of subependymal cysts in high-risk neonates. *AJNR* **1984**;5:761–764, *AJR* **1985**;144:377–380
10. Butt W, Mackay RJ, deCrespigny LC, Murton LJ, Roy RND. Intracranial lesions of congenital cytomegalovirus infection detected by ultrasound scanning. *Pediatrics* **1984**;73:611–614
11. Shaw CM. Subependymal germinolysis. *J Neuropathol Exp Neurol* **1973**;32:153
12. Shaw CM, Alvord EC. Subependymal germinolysis. *Arch Neurol* **1974**;31:374–381
13. Larroche JC. Subependymal pseudo-cysts in the newborn. *Biol Neonate* **1972**;21:170–183
14. Friede RL. *Developmental neuropathology*. New York: Springer-Verlag, **1975**:7
15. Rorke LB. *Pathology of perinatal brain injury*. New York: Raven, **1982**:37
16. Gilles FH, Leviton A, Dooling EC. *The developing human brain. Growth, epidemiology, neuropathology*. Boston: Wright, **1983**:235–237
17. Armstrong D, Norman MG. Periventricular leukomalacia in neonates. Complications and sequelae. *Arch Dis Child* **1974**;49:367–375
18. Takashima S, Tanaka K. Development of cerebrovascular architecture and its relationship to periventricular leukomalacia. *Arch Neurol* **1978**;35:11–16