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The echogenic thalamus in hypoxic ischaemic encephalopathy

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Abstract. This paper reports 16 term infants in whom an echogenic thalamus was identified on cranial ultrasonography. Fourteen of the patients suffered severe birth asphyxia. The prognostic significance of this finding and the underlying pathogenesis is assessed.

The radiological features of neonatal asphyxia in the term infant are results of a global insult to the brain [1, 2]. Generalised cerebral oedema resulting from hypoxia may be difficult to identify on cranial ultrasound (US). The finding of increased echogenicity in the thalamus in asphyxiated term infants, in an otherwise normal brain, is unusual [2–4]. This paper reports 16 term infants in whom an echogenic thalamus was identified on cranial US. The prognostic significance of this finding and the underlying pathogenesis is assessed.

Materials and methods

We reviewed the US, computed tomography (CT) and the clinical data of 16 infants with US evidence of increased echogenicity in the thalamus, all of whom were of more than 35 weeks' gestation, and who had suffered birth asphyxia between 1985 and 1992. US examinations were performed on a Diasonics machine, using a 6-MHz transducer. Non-contrast computed tomographic examinations were performed on a Siemens Somatom 2 machine, using axial 8 mm × 8 mm slices. All examinations were performed by a single operator. Post-mortem material was available in four of five infants who died, and was examined by an neonatal pathologist and a neuropathologist. Follow-up at a remedial clinic was obtained where possible.

Results

All infants were of more than 35 weeks' gestation with a mean birth weight of 3.2 kg, and a range of 2.0–4.15 kg. Pregnancy complications and mode of delivery are presented in Table 1. One infant had a normal birth but suffered a near-miss sudden infant death event at 4 days of age. Fourteen had severe birth asphyxia. One infant had no identifiable hypoxic episode. All infants developed abnormal neurological features in the postnatal period which included seizures, arching, fisting, abnormal eye movements, altered tone, and absent suck. Metabolic disorders infection and dehydration were excluded in all of the 16 infants. A chromosomal abnormality consisting of 5 % mosaic with triploidy was identified in one infant.

All infants had serial cranial US examinations, except one baby who died at less than 48 hours of age, in whom only one study was performed. Eleven infants showed an abnormal increase in echogenicity in either one or both thalami on initial scans (Fig. 1). Five infants had normal studies initially but subsequent examinations showed the development of bright echoes within the thalami. The abnormality was associated with sonographic evidence of cerebral oedema in four infants out of the total group as evidenced by very small ventricles, reduction in pulsations in the anterior and middle cerebral arteries, and mild diffuse increased parenchymal echo pattern. Increased echogenicity was bilateral in 14 infants and unilateral in two. The pattern was focal in three and diffuse in 13. In three of the latter group the pattern became more focal with time.

In 2 infants the abnormality resolved over a period of 6 weeks (Fig. 2). In the remaining 14 it became more

Table 1. Pregnancy complications and mode of delivery

Pregnancy complications	Type of delivery
Ovarian cystectomy at 25 weeks ($n = 1$)	Normal vaginal delivery ($n = 7$)
Hypertension at 35 weeks ($n = 1$)	Vaginal delivery assisted by forceps ($n = 7$)
Epilepsy and deep vein thrombosis ($n = 1$)	Vacuum extraction ($n = 1$)
Abruptio placentae ($n = 1$)	Caesarian section ($n = 1$)
No complications ($n = 12$)	

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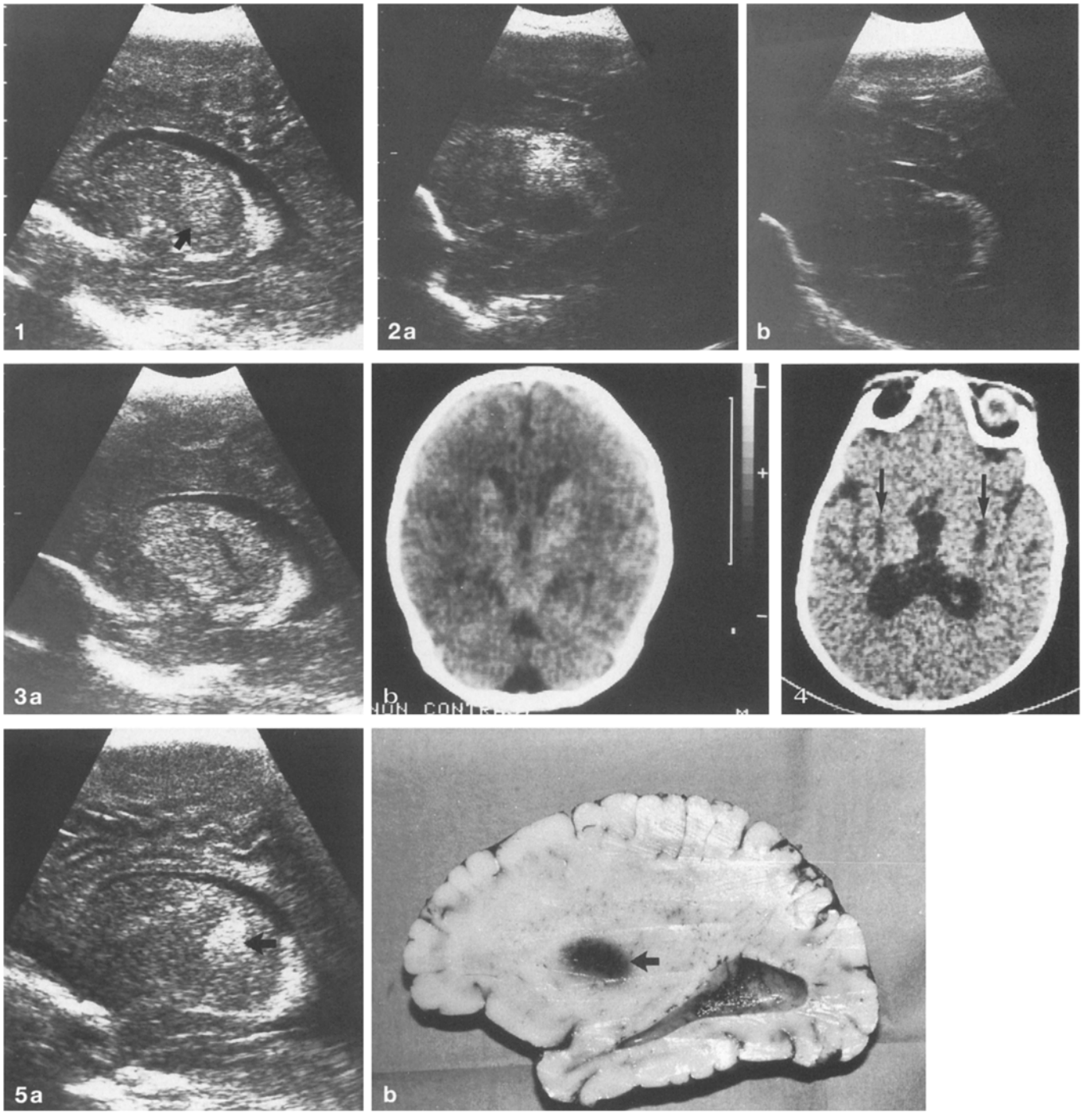

Fig. 1. Sagittal sonographic image. There is increased echogenicity of the thalamic region *(arrow)*

Fig. 2. a Sagittal sonographic image showing increased echogenicity at the anterior aspect of the right thalamus. **b** 6 weeks later, the appearances have resolved

Fig. 3. a Sagittal sonographic image showing increased echogenicity of the thalamus extending to involve the caudate nucleus. This was bilateral. **b** Axial CT slice on the same infant showing bilateral increased attenuation in both thalami and caudate nuclei

Fig. 4. Axial CT slice. There are bilateral linear low attenuation areas in the thalamic regions *(arrows)*

Fig. 5. a Sagittal sonographic image. There is a discrete area of increased echogenicity in the thalamus *(arrow)*. This was bilateral and symmetrical. **b** Post-mortem sagittal brain slice, gross appearance. There is a haemorrhagic area in the region of the thalamus *(arrow)* corresponding to the sonographic appearances

echogenic with time. Three of the four infants with cerebral oedema later showed mild ventricular dilatation.

Eleven infants had CT scans. The examination was normal in four. One of these four subsequently showed mild cerebral atrophy on a repeat CT examination at 8 months. Seven infants had abnormal CT scans. In three there was increased attenuation in both thalami, extending into the caudate nuclei in one (Fig. 3), suggesting bleeding into the thalami. One patient showed increased attenuation in the left thalamus as predicted by ultrasound, but in addition a supra-tentorial haemorrhage was detected which could not be seen by sonography. One infant had a CT scan performed at 5 months of age which showed bilateral dense thalamic calcification. The remaining two infants, whose CT examinations were performed at 2 and 5 months of age respectively, showed bilateral discrete linear low attenuation areas in the thalami, consistent with thalamic infarcts (Fig. 4).

Five infants died. Two infants died at less than 48 hours of age, and one at 1 month, at 5 months and at 3.5 years. Post-mortem examination was performed in four. Haemorrhage and oedema at the site of the sonographic abnormality was seen in the two infants who died at less than 48 hours of age (Fig. 5). The neurones were intact in both infants.

In the infants who died at 1 and 5 months the autopsies showed haemosiderin deposition, calcium encrustations and neuronal gliosis involving the thalami, basal ganglia, brain stem, cerebellum and spinal cord.

Eleven infants have survived to date. Nine have significant neurological sequelae, including mental and motor handicap. Two infants have normal development. One infant shows normal physical and mental development but suffers from epilepsy, and one child was normal at the 6 months' review but has since been lost to follow-up.

Discussion

The newborn thalamus is normally a medium-level echogenic structure. The significance of increased echogenicity in the thalamus has received little attention in the literature. The poor neurological outcome of the infants in our group stimulated follow-up of these children and prompted this study [3].

The findings of normal initial US examinations in several infants suggests this entity is a result of an immediate prepartum, intrapartum or early postnatal event, rather than an antenatal event [4]. All infants except one had suffered a significant hypoxic episode as evidenced by standard criteria. An antenatal event therefore appears less likely, given such a recognised and identified episode [1].

In all infants the abnormalities in the thalamus were more evident on sonography than on CT, indeed, in four patients the CT was entirely normal. In the positive CT examinations the degree of abnormality and laterality concurred with the sonographic findings.

We therefore feel that sonography is more sensitive than CT in detecting these alterations in the parenchymal pattern of the thalamus. CT, however, does have a role in identifying other abnormalities not visible on sonography as was the case in one of our infants with a supratentorial haemorrhage. We feel that serial US examinations are necessary to identify and follow these abnormalities, as the studies may show a normal thalamus in the early postnatal period.

Increased echogenicity is a non-specific finding and may be due to haemorrhage or contusion within the substance of the thalamus or indeed calcification or infarction [5]. The distinction between these cannot be made by ultrasonography alone, but can be inferred by serial US and CT scans, and confirmed by histology [2]. Identification of an abnormal area seen by sonography requires serial examinations to determine if it persists or resolves. Histological findings in two infants who died early showed haemorrhage and no evidence of infarction. The two surviving infants with a good neurological outlook were those in whom the sonographic abnormality resolved, which was very probably also due to haemorrhage without infarction [6].

Histology on the other two infants with abnormal neurological signs, who died, revealed established widespread neuronal loss [1]. In the eight surviving infants with significant handicap, the sonographic abnormality was persistent and was most probably due to infarction.

The findings on histology of haemosiderin and astrogliosis were more widespread than sonography predicted, as they were seen in the cerebellum, brain stem and as low down as the lumbar spinal cord, reflecting the severity and global nature of the hypoxic event.

Hypoxia is a global insult affecting the whole brain, yet in these infants the thalamus exhibited the greatest – and often only – abnormality. It is unclear why such a global event should result in apparently localised sonographic abnormality in the thalamus. In only four infants was cerebral oedema identified in addition to their thalamic abnormality. It is said that the thalamus may be very sensitive to hypoxia due to its high metabolic rate and its blood supply [7, 8]. But if so, it is unclear why this abnormality is not more commonly seen, even in more minor asphyxiating episodes. Histology supports a hypoxic-ischaemic aetiology, with evidence of haemorrhage or infarction, with or without calcium deposition.

In conclusion, in our experience, the finding of increased echogenicity in the thalamus in a term infant who has suffered asphyxia is of sinister significance. In this series it was associated with a 31 % mortality, and a further 56 % long-term morbidity. We feel the abnormality most probably represents hypoxia-ischaemia, with or without infarction. Resolution in our experience was associated with a good prognosis (12.5 %), and most probably represents haemorrhage without infarction. Serial US is the imaging modality of choice in this entity; CT does have a role in identifying other abnormalities not visible on US.

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References

1. Editorial (1992) Thalamic lesions in infancy. Lancet 339: 1143–1145
2. Hill A, Martin DJ, Daneman A, Fitz CR (1983) Focal ischaemic cerebral injury in the newborn: diagnosis by ultrasound and correlation with computed tomographic scan. Pediatrics 71: 790–793
3. Shen EY, Huang CC, Hung HY, Hsu CH, Huang FY (1986) Sonographic findings of the bright thalamus. Arch Dis Child 61: 1096–1099
4. Eicke M, Briner J, Willi U, Uehlinger J, Bolshauser E (1992) Symmetrical thalamic lesions in infants. Arch Dis Child 67: 15–19
5. Kanerek KS, Gieron MA (1986) Computed tomography demonstration of cerebral calcification in postasphyxial encephalopathy. J Child Neurol 1: 56–60
6. Trounce JO, Frawer CL, Punt J, Dodd KL, Fielder AA, Levene MI (1985) Primary thalamic haemorrhage in the newborn: a new clinical entity. Lancet I: 190–192
7. Vannucci RC and Voorhies TM (1984) Perinatal cerebral hypoxic-ischaemia: pathogenesis and neuropathology. Top Neonat Neuropathol: 27–82
8. Reilly M, Connolly S, Stack J, Martin EA, Hutchinson M (1992) Bilateral paramedian thalamic infarction: a distinct but poorly recognised stroke syndrome. Q J Med 297: 63–70

Literature in pediatric radiology

Continued from p. 265

Journal of Pediatric Surgery *(New York)*

Malignant small round cell tumor (Ewing's-PNET) of the chest wall in children. Shamberger, R.C. et al. (Dept. of Surg., The Children's Hosp., 300 Londwood Av., Boston, MA 02115, USA) 29:179 (1994)

Neuroimaging of brain injury in neonates treated with extracorporeal membrane oxygenation: lessons learned from serial examinations. Lazar, E.L. et al. (Stolar, C.J.H., Div. of Ped. Surg., College of Physicians and Surgeons, The Babies Hosp., Columbia Presbyterian Med. Center, 3959 Broadway, Room 212N, New York, NY 10032, USA) 29:186 (1994)

The spectrum of bile duct complications in pediatric liver transplantation. Peclet, M.H. et al. (Ryckman, F.C., Ped. Surg. Div., Children's Hosp. Med. Center, 240 Bethesda Av., Cincinnati, OH 45229, USA) 29:214 (1994)

Congenital high airway obstruction syndrome (CHAOS): a potential for perinatal intervention. Hedrick, M.H. et al. (Adzick, N.S., Fetal Treatment Center, UCSF Med. Center, HSW 1601, San Francisco, CA 94143-0570, USA) 29:271 (1994)

Ultrasonography is accurate enough for the diagnosis of intussusception. Shanbhogue, R.L.K. et al. (Dept. of Ped. Surg., Sophia Children's Hosp., Gordelweg 160, NL-3038 GE Rotterdam, The Netherlands) 29:324 (1994)

Medical and Pediatric Oncology *(New York)*

Radiotherapy in the SIOP (International Society of Pediatric Oncology) nephroblastoma studies: a review. Jereb, B. et al. (The Inst. of Oncol., Zaloska 2, 61000 Ljubljana, Slovenia) 22: 221 (1994)

Bone scintigraphy in nonsurgically treated Ewing's sarcoma at diagnosis and following-up: prognostic information of the primary tumor site. Hugenholtz, E.A.L. et al. (Piers, D.A., Dept. of Nucl. Med., Univ. Hosp., P.O. Box 30.001, NL-9700 RB Groningen, The Netherlands) 22: 236 (1994)

Esthesioneuroblastoma presenting as an orbital mass in a young girl. Bobele, G.B. et al. (Dept. of Neurol., Div. of Ped. Neurol., P.O. Box 26307, Oklahoma City, OK 73126, USA) 22: 269 (1994)

Choroid plexus carcinoma: report of one case with favourable response to treatment. Aricò, M. et al. (Dept. of Ped., Univ., IRCCS Policlinico S. Matteo, I-27100 Pavia, Italy) 22: 274 (1994)

Journal of Neurosurgery *(Baltimore)*

Intracranial meningiomas of the first two decades of life. Germano, I.M. et al. (Edwards, M.S.B., Dept. of Neurol. Surg., c/o The Editorial Office, 1360 Ninth Av., Suite 210, San Francisco, CA 94122, USA) 80:447 (1994)

Radiology *(Easton PA)*

Pediatric knee MR imaging: pattern of injuries in the immature skeleton. Zobel, M.S. et al. (Borrello, J.A., Mallinckrodt Inst. of Rad., Washington Univ. School of Med., 510 S Kingshighway Blvd., St. Louis, MO 63110, USA) 190:397 (1994)

Juvenile rheumatoid arthritis of the knee: evaluation with US. Sureda, D. et al. (Dept. of Rad., Hosp. General Vall d'Hebron, Av. Vall d'Hebron s/n, E-08035 Barcelona, Spain) 190:403 (1994)

Three-dimensional sonographic evaluation of developmental dysplasia of the hip: preliminary findings. Gerscovich, E.O. et al. (Dept. of Rad., Univ., Davis Med. Center, 2516 Stockton Blvd., TICON II, Sacramento, CA 95817, USA) 190:407 (1994)

Splenic enlargement in neonates during ECMO. Klippenstein, D.L. et al. (Zerin, J.M., Dept. of Rad., Riley Hosp. for Children, 702 Barnhill Dr., Indianapolis, IN 46202–5200, USA) 190:411 (1994)

Vesicoureteral reflux: an accurate predictor of acute pyelonephritis in childhood urinary tract infection? Ditchfield, M.R. et al. (Dept. of Rad., Children's Hosp., 4480 Oak St., Vancouver, British Columbia, Canada V6H 3V4) 190:413 (1994)

Chondroblastoma: classic and confusing appearance at MR imaging. Weatherall, P.T. et al. (Rogers Magnetic Resonance Center, 5801 Forest Park Rd., Dallas, TX 75235–9085, USA) 190:467 (1994)

Archives of Disease in Childhood *(London)*

Renal scarring after acute pyelonephritis. Jakobsson, B. et al. (Dept. of Paed., Univ. Hosp., Karolinska Inst., S-14186 Huddinge, Sweden) 70: 111 (1994)

Metacarpal index in Marfan's syndrome and in constitutional tall stature. Nelle, M. et al. (Dept. of Paed., Univ., Im Neuenheimer Feld 150, D-69120 Heidelberg, FRG) 70:149 (1994)

British Journal of Radiology *(London)*

Plain chest radiographic changes of the absent pulmonary valve syndrome. Owens, C.M. et al. (Shaw, D., Dept. of Rad., The Hosp. for Sick Children, Great Ormond St., London WC1N 3JH, UK) 67:248 (1994)

Case report: calcification within aneurysmal bone cyst. Sharma, P. et al. (Dept. of Radiodiagnosis, Jawaharlal Inst. of Postgraduate Med. Education and Research (JIPMER), Pondicherry (605 006), India) 67:306 (1994)

Clinical Radiology *(Edinburgh)*

Case report: extravasation of Lipiodol. A complication of dacryocystography. Mansfield, D.C. et al. (Mackenzie, J.R., Dept. of Rad., Royal Hosp. for Sick Children, Yorkhill, Glasgow G3 8SJ, UK) 49:217 (1994)

International Journal of Pediatric Otorhinolaryngology *(Shannon)*

Holoprosencephaly in neonates. Ram, S.P. et al. (Dept. of Paed., Hosp. Univ. Sains, 16150, Kubang Kerian, Kelantan, Malaysia) 29:65 (1994)

Swelling over the face: infantile cortical hyperostosis. Jakubiková, J. et al. (Ped. ENT Clinic, Bardosova 21, 83101 Bratislava, Slovak Rep.) 29:147 (1994)

Congenital nasolacrimal duct mucocele: a cause of respiratory distress. Yee, S.W. et al. (Dept. of Otolaryngol.-Head and Neck Surg., Univ. for Med. Sciences, 4301 West Markham/Slot 543, Little Rock, AR 72205, USA) 29:151 (1994)

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