The 'bright brain'

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SUMMARY In this preliminary study we report on a new neonatal cerebral ultrasound appearance consisting of high amplitude echoes that arise diffusely throughout the brain and obscure the echoes of normal structures. This scan appearance was associated with probable cerebral insult and we believe that it is caused by cerebral oedema.

A study was set up to assess the feasibility of developing a neonatal cerebral scanning service in 2 neonatal intensive care units—one in a subregional referral centre and the other in the baby unit of a district general hospital. During the study a new neonatal cerebral ultrasound appearance was noted in 13 infants.

Patients and methods

Two identical ATL Mark 100 5 MHz ultrasound sector scanners were used to scan neonatal brains in the 2 intensive care units. Indications for scanning were not rigorous, and were low birthweight (particularly 1500 g or less), moderate or severe illness, and any clinical neurological abnormalities. One hundred and fifty one babies were scanned—56 from the district unit and 95 from the subregional unit. Twenty four (46%) babies from the district unit and 77 (81%) babies from the subregional unit weighed 2500 g or less at birth. We studied 13 babies, 8 from the district unit and 5 from the subregional unit.

Results

The cerebral ultrasound scans of these 13 babies showed an unusual pattern characterised by an excess of small high amplitude echoes, scattered diffusely, that made some major structures indistinct (Fig. 1(a)). Where these 2 elements—an increase in diffuse echogenicity and a consequent partial or complete obliteration of structures normally visible—were present, the appearance was recorded as positive. Figure 1(a) shows a positive scan of 1 of the babies (case 8); a 'snowstorm' of bright echoes hides the right lateral ventricle and makes other structures less clearly visible than normal. In the other 12 babies the degree of obliteration was more, or less, severe.

Fig. 1(a) Coronal scan through anterior fontanelle of 36 weeks' gestation infant (case 8) aged 15 hours: severe birth asphyxia and convulsions. Normal structures seen with difficulty.

Fig. 1(b) Same infant as in Fig. 1(a), scanned in same plane with the same machine settings, but 9 days later: neurologically improving; normal scan.

In 12 of the 13 babies the history or clinical findings, or both, suggested that a cerebral insult had occurred (Table). Eight babies had generalised convulsions (7 definite and 1 probable), 5 of whom had episodes likely to lead to hypoxic/ischaemic cerebral injury. Of these 5 infants, 4 also developed

Table *Clinical characteristics in relation to ultrasound scans*

Case	Gestational age (weeks)	Birthweight (g)	Cerebral insult	Abnormal clinical findings	Age scan abnormal (days)	Age scan normal (days)	Age clinical improvement (days)	No of scans
<i>District Unit</i>								
1	36	2130	None known	None	5	13	N/A	3
2	41	3350	1 min Apgar = 4	Convulsions day 1	0.5	5	1	2
3	33	1230	Blood glucose 1.0 mmol/l, day 1	Irritability, cyanotic spells day 1	1	>17	6	2
4	38	3000	None known	Convulsions, day 1	1	4	4	3
5	33	1930	Difficult forceps delivery	Irritable, day 1	0.3	15	1	4
6	39	2720	None known	Convulsion, day 1	1	Not repeated	2	1
7	33	1420	Blood glucose 0.2 mmol/l, day 1	Localised limb twitching, day 1	<1	4	4	3
8	36	1660	5 min, Apgar = 2	Convulsions requiring ventilation, day 1; ARF	0.5	9	6	5
<i>Sub-regional unit</i>								
9	40	4100	5 min Apgar = 4, breech delivery	Probable convulsions day 1: ARF	8	Not repeated	6	1
10	40	2770	Traumatic breech delivery, shock	Convulsions, day 1: ARF	4	None normal	None	6
11	40	3300	Birth asphyxia, meconium aspir.	Convulsions, day 1; ARF: died at 25 days	7	14	Gradual incomplete	3
12	40	1840	Blood glucose <0.5 mmol/l, day 1	SGA asymptomatic	0.1	Not repeated	N/A	1
13	40	3860	Group B, β -haemolytic strept. meningitis	Convulsions, days 8-22	13	16	22	3

ARF = acute renal failure; SGA = small for gestational age; N/A = not applicable.

Conversion: SI to traditional units—blood glucose: 1 mmol/l $\approx$ 18 mg/100 ml.

acute renal failure and 1 (case 10) needed peritoneal dialysis. One of the remaining 3 babies who suffered generalised convulsions had group B, β -haemolytic streptococcal meningitis and the other 2 had fits in the first day for no known reason.

Five babies did not have generalised convulsions: 3 had documented hypoglycaemia, blood glucose $\leq$ 1.0 mmol/l ($\leq$ 18 mg/100 ml)—2 had non-specific signs of irritability and 1 was asymptomatic; 1 had non-specific irritability after a difficult forceps delivery; and 1 was clinically normal with a normal previous medical history.

In 6 of the 8 babies in whom the time of cerebral insult could be estimated, diffuse hyperechogenicity was first seen within 24 hours of birth (range 4–24 hours). But it was a transient phenomenon, and had resolved completely in 8 of the 13 babies in 3 to 15 days. (Fig. 1(b) is an ultrasound scan of the same baby as in Fig. 1(a), but taken 9 days later.) Resolution of the ultrasound appearances was always accompanied by improvement in any abnormal neurological signs. Failure to improve clinically was associated with the persistence of ultrasound scan appearances in 1 baby and 3 had no follow up scans because they had been discharged home from the unit, clinically normal. Twelve of the 13 babies

survived to be discharged home, but 1, who had made some neurological improvement and had lost her diffuse hyperechogenicity scan appearance, died suddenly.

Discussion

In this preliminary study there was a high incidence of probable cerebral insult among those babies with a positive scan appearance, but we cannot determine the incidence of a positive scan appearance among those babies who were likely to have suffered cerebral insult, or the incidence, if any, of positive scan appearances among 'normal' babies. Babcock and Han¹ referred to diffusely abnormal echogenicity in the neonatal brain after severe asphyxia, but no other reference to the cerebral scan appearances described in this study has been found in published reports. There is, however, a sign known as the 'bright liver',² seen in diffuse chronic liver disease that seems analogous. Indeed, the original report² of the 'bright liver' describes both diffuse hyperechogenicity and a loss of clarity of vessel boundaries, which suggests that this neonatal cerebral scan appearance might even be referred to as the 'bright brain'.

There are several reasons why the image may appear diffusely hyperechogenic. Machine artefact may be excluded by careful attention to machine settings and by repeating the same settings on subsequent examinations. It seems that an excess of echoes is produced throughout the brain substance, swamping those produced by structures that are usually clearly visible so that they are no longer clearly defined. If the clinical associations are confirmed the cause may be a pathological process that develops soon after a cerebral insult, is often non-fatal, and resolves over a few days. Cerebral oedema is a possible cause, although its occurrence in the neonatal brain, particularly in that of the preterm infant, has recently been questioned.³

Diffuse neonatal cerebral hyperechogenicity is of interest as an ultrasound scan appearance. A prospective study will be necessary to determine its incidence, the correlation with cerebral insult, and the prognosis. Such a study may help to interpret the pathological importance of this scan appearance and further clues may be provided by the apparent differences and birthweight distribution of incidence

observed between the 2 units we studied. If the 'bright brain' scan appearance represents cerebral oedema it will help considerably in the diagnosis and management of this condition.

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