tion in general. They assumed that the heights of children had not changed significantly during the observation period; this assumption, however, might be invalid. Indeed, the mean of the standard deviation scores was greater than 0 in the present study, though there was no difference between the means of the scores of patients and controls. Therefore, comparison with matched controls is essential in this type of study.

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Late onset cystic leucomalacia

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SUMMARY The ultrasound findings of eight premature infants who developed extensive cystic leucomalacia after the first two weeks of life are reported.

Ultrasound examinations should be considered after any clinical deterioration in preterm infants up to 40 weeks' postmenstrual age to avoid missing cases of extensive cystic leucomalacia.

Cranial ultrasonography was first introduced to neonatal intensive care units in 1979 and has now gained widespread acceptance as the diagnostic method of choice in the recognition of intracranial pathology.[1] The diagnosis of periventricular-intraventricular haemorrhages during life was initially seen as the main asset. These haemorrhagic lesions were noted to be common, especially in infants weighing 1500 g or less.[2] Most studies have shown that periventricular-intraventricular haemorrhages tend to occur within the first few days of life and, even in the presence of major problems, rarely after the first week of life.[2]

More recently we have become aware that ischaemic lesions can also be diagnosed by this technique.[3] The ischaemic lesions seem to be less common than the haemorrhagic ones.[3] In the past it has been assumed that this lesion occurs due to either prenatal insults or major problems in the immediate postnatal period.

We have been able, however, to document eight preterm infants who had normal ultrasound scans during the first two weeks of life and subsequently suffered an 'insult', which led to the development of extensive cystic leucomalacia.

Methods

Infants admitted to the Hammersmith Hospital Regional Intensive Care Unit were routinely scanned, daily during the first week of life and twice weekly thereafter until discharge, using an ATL Mark III sector scanner with a multifrequency scan head (3–5–7·5 MHz crystals).

Extensive cystic leucomalacia was defined as areas of increased echogenicity followed by cystic degeneration seen both in the coronal and the parasagittal view in the periventricular or subcortical areas.

Results

Between January 1982 and October 1985, 20 infants were diagnosed as having extensive cystic lesions, either in the periventricular or in the subcortical areas.

In 12 infants the cystic lesions were related to an antenatal or perinatal insult. In the remaining eight infants, however, evidence of leucomalacia, as noted by areas of increased echogenicity, was first noted between 16 days and 10 weeks after birth. The initial ultrasound scans were normal in seven of the eight infants, and the remaining infant was noted to have a small intraventricular haemorrhage (Table).

Table *Results of initial ultrasound scan and outcome of eight infants with development of leucomalacia due to an 'insult' first noted after the first two weeks of life*

Case no	*Gestation (wks)*	*Sex*	*Time of insult postmenstrual age age (wks)*	*Type of insult*	*Initial ultrasound scan*	*Type of lesion*	*Outcome*
1	30	M	32·5	Unknown	n	PVL	Severe cerebral palsy
2	27	M	31	Apnoeic spells	IVH	PVL	<6 months
3	28	M	30	NEC	n	PVL	<6 months
4	30	F	34	NEC	n	PVL	Died
5	30	F	34	NEC+sepsis	n	SCL	Severe cerebral palsy
6	27	F	36	Shock	n	SCL	Died
7	28	F	32	NEC	n	PVL	Died
8	30	F	32	Unknown	n	IVH+PVL	<6 months

IVH=intraventricular haemorrhage; NEC=necrotising enterocolitis; PVL=periventricular leucomalacia; SCL=subcortical leucomalacia; n=normal.

Discussion

Extensive cystic leucomalacia is less common than periventricular-intraventricular haemorrhage, but because of its poor outcome, its recognition is of great importance.[4] The present data suggest that leucomalacia, unlike periventricular-intraventricular haemorrhage, which rarely occurs beyond the end of the first week, can develop after a clinical deterioration at any time up to 40 weeks' postmenstrual age. This was also noted by Rushton, who recently reviewed 18 infants with a variety of echodense lesions, two of whom showed changes consistent with leucomalacia around 60 days of life, after a period of collapse.[5]

In our study eight out of 20 infants with extensive cystic lesions developed these lesions beyond the second week of life. This was associated with acute problems, like necrotising enterocolitis or sepsis, in six of these eight infants, but the other two showed no acute clinical deterioration.

Due to the natural evolution of cystic leucomalacia as seen by ultrasonography, it is evident that the diagnosis can be easily missed.[6] If the infant is scanned shortly after the collapse the first changes indicating leucomalacia (areas of increased echogenicity) may not yet have had time to develop. If the scan is postponed for several days, however, the cysts may not yet be present. In both situations the scan, having yielded normal results, may not be repeated.

We suggest, therefore, that as many as 40% of the infants with extensive cystic lesions may be missed unless all infants are scanned on a regular basis, especially those who suffered a late 'insult'.

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