Neonatal Medicine

Real time ultrasound, arterial pulsation and neonatal cerebral infarction.

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Summary: This paper describes an infant whose cranial ultrasound scan showed marked unilateral cerebral arterial pulsation and enlargement without other abnormality. Subsequent computerized tomogram showed extensive cortical infarction in an area not readily accessed by ultrasound. It is concluded that the real time dimension of cranial ultrasound is of diagnostic value in the absence of demonstrable parenchymal or intraventricular abnormality.

Introduction

During the course of the first year of a diagnostic ultrasound service in a non-referral neonatal unit, 20 term neonates with suspected intracranial pathology were scanned using an A.T.L. 100 sector scanner with a 5 MHz in-line transducer. These came from a total of approximately 800 scans carried out by a consultant paediatrician and a senior registrar in paediatrics (B.D.). ‘Difficult’ scans were discussed with a consultant paediatric radiologist with experience of neonatal cranial ultrasound.

Prior to provision of the service a small study had been undertaken to assess correlation between ultrasound and post-mortem appearance of intracranial pathology. Eight infants of less than 1500 g were studied and excellent correlation existed according to the grading system of Levene & de Crespigny.1

Scans of the 5 infants considered to have non-ischaemic intracranial pathology showed one infant with hydrocephalus (myelomeningocele), and one infant with agenesis of the corpus callosum.

Ischaemic cerebral injury was suspected in the other 15 mature neonates. Scans of 11 infants were normal, 2 infants had the ‘bright brain’ appearance,2 one infant unilateral echodensity associated with diminished generalised arterial pulsation, and a further infant with the appearances described below.

Case history

On the fifth day an infant was noted to have generalized clonic movements occurring at the rate of one per second and more marked on the right side. She had been born to a healthy primiparous mother by spontaneous vertex delivery at term. There had been no evidence of asphyxia and head circumference was on the 50th percentile. Birth weight was appropriate at 3.82 kg and there was no history of maternal drug ingestion. Apart from overt seizure activity, clinical examination including carotid pulsation and auscultation of neck and skull was normal. Serial oscillometric blood pressure measurements were normal.

Screening for infection and metabolic abnormality of blood, cerebrospinal fluid and urine were negative. Electroencephalography showed low voltage, featureless activity over the left cerebral cortex. Skull X-ray was normal. Echocardiography showed normal anatomical relations with no evidence of intracardiac or ductal shunt. Cranial ultrasound showed an unusually large, pulsating vessel in the distribution of the left anterior cerebral artery from its junction with the internal carotid artery to the anterior horn of the left lateral ventricle (Figure 1).

Four ultrasound scans were performed over a 24 hour period from the onset of the seizures. The first three scans were performed in the neonatal unit and the fourth on transfer to the regional referral centre. The fourth scan was performed by a consultant radiologist who confirmed the above findings and noted no other ultrasound abnormality. The seizures

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occurred in clusters over 12 hours and responded to intravenous phenytoin. Transfer to the regional paediatric neurology unit had been made on the grounds of possible arteriovenous malformation.

Twenty four hours after the commencement of seizures a computed tomographic (CT) scan with contrast enhancement was performed (Figure 2) and the results reviewed by a consultant radiologist, neurologist and neurosurgeon. The diagnosis at this time was unclear and considered to be either ischaemic infarction or an arteriovenous malformation similar to that seen in the Sturge-Weber syndrome. Neurosurgical intervention was not considered to be an option at this time and no immediate further investigation was undertaken.

Repeat cranial ultrasound at 2 weeks and 3 months was normal. A CT scan at 8 months has shown cerebral atrophy only, consistent with perinatally acquired ischaemic cerebral injury.

The patient now exhibits global retardation with right hemiparesis. No further seizures have occurred.

Comment

Literature on neonatal cranial ultrasound contains little reference to arterial pulsation. Hill et al. observed changes in cerebral arterial pulsation associated with areas of increased echodensity in 3 infants. These mature neonates were considered to have cerebral ischaemic insult on clinical grounds and CT scan showed the echodensities to be likely areas of infarction.

Ultrasound has been shown to be of prognostic value in the low birth weight infant in whom haemorrhage is a common marker of intracranial pathology. Haemorrhage is a less common consequence of ischaemic cerebral injury in the mature neonatal brain, and diffuse parenchymal lesions are probably better identified by CT than by ultrasound.

Cerebral perfusion is a central issue in the pathophysiology of the neonatal brain and the dynamic quality of real time ultrasound permits imaging of arterial pulsation not apparent in other scanning techniques such as CT.

The ultrasound appearances in the case described are unique in our experience to date. Accurate measurement of regional cerebral perfusion is not possible in normal clinical practice but the appearances suggested a localized disturbance in perfusion later confirmed by the CT changes consistent with infarction.

Lassen has described relative hyperaemia of cerebral tissue surrounding infarction and has also reported increased cerebral blood flow, generalized or local, during seizures.

The true incidence of neonatal cerebral infarction cannot be accurately known, although it is the most common cause of subsequent handicap such as cerebral palsy. Aetiological factors related to infarction were not apparent in the case described. Prenatal asphyxia is not uncommonly missed by standard monitoring techniques. Evidence of disseminated intravascular coagulation, placental infarction, involving fetal circulation, vessel trauma and systemic hypotension were absent in this case. It is not possible to state whether the seizures observed in this infant preceded or followed the occurrence of cerebral infarction.
Although knowledge of perinatal hypoxic ischaemic injury remains incomplete, cranial ultrasound has contributed greatly to current understanding of the mechanisms involved. However, little comment is usually made when reporting scans in respect of arterial pulsation. Such comment, although subjective, may provide important information regarding pathology not immediately apparent.

References

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