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Isolated cerebral cortical tears in children: aetiology, characterisation and differentiation from non-accidental head injury

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ABSTRACT. A wide spectrum of intracranial injuries has been described as complicating difficult birth, particularly following instrumental delivery. We describe five children in whom isolated cortical tears were observed on MRI. Four cases were characterised by a difficult instrumental delivery. None of the children developed long-term neurological sequelae. As far as we are aware, isolated cerebral cortical tears have not been reported previously although recognition of this injury pattern is important because of its possible misinterpretation as a marker of a non-accidental head injury. Other differential diagnoses that should be considered include cerebral infarcts, schizencephaly and accidental head injury. The importance of high-quality cross-sectional brain imaging in newborn infants with seizures is emphasised.

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The association between instrumental delivery and neurological injury has been recognised since the late 19th century [1]. Ventouse (vacuum-assisted delivery) and forceps extraction remain the two main modes of instrumental delivery in widespread use. A wide spectrum of birth-related injuries to the brain has been described in association with difficult delivery [2], including skull fracture, extra-axial haemorrhage, intraventricular haemorrhage, cerebellar haemorrhage, dural tears and cerebral infarcts.

Non-accidental head injury (NAHI) poses a considerable diagnostic challenge for both clinicians and radiologists. Head injury is a common manifestation of child abuse. When intracranial injury occurs, it can be difficult to differentiate between accidental head injury (AHI) and NAHI, particularly as parents will often attribute injury to the former [3]. Establishing a correct diagnosis of NAHI can be very challenging but is of great importance. If missed, the child may be put at risk if placed back into a potentially abusive environment. Conversely, incorrectly ascribing the injury as inflicted can cause great harm to the family unit.

This study describes a series of five children, all of whom had cortical tears diagnosed upon imaging by various modalities including ultrasound, CT and MRI. All histories were reviewed to attempt to identify aetiological factors and to determine the prognosis of such injuries. To our knowledge, this pattern of injury has not been previously described. Three of the children underwent instrumental delivery and one an emergency Caesarean section following failure to progress in the second stage of labour. Birth trauma seems a likely common aetiological factor.

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Methods and materials

All five patients included in the study were found to have isolated cortical tears on MRI scans. Their case notes were retrospectively reviewed in an attempt to elucidate the aetiology of their intracranial injuries. In particular, any trauma to the mother's abdomen whilst pregnant, birth trauma and instrumental delivery, Apgar scores, documented head trauma and bruising post delivery, neurological deficit at presentation and the most recent clinical follow-up were recorded. The results of clotting studies, neonatal jaundice and infection screening were also documented.

All imaging was reviewed by two experienced neuroradiologists (N.S.M and T.J), both of whom have extensive experience in paediatric neuroimaging and non-accidental injury and work in a major tertiary referral centre.

Cranial ultrasound scanning was undertaken using a combination of multifrequency sector and linear array transducers, employing Acuson, (Mountain View, CA) or Toshiba (Toshiba Medical Systems, Tokyo, Japan) ultrasound units. CT and MR imaging was performed following oral sedation (chloral hydrate 75–100 mg kg^{-1}). Multislice CT scanning was undertaken in all cases on a GE system (Lightspeed, GE Medical Systems, Milwaukee, WI), employing a standard protocol using 5 mm thick slices. Intravenous contrast was not administered. The MR imaging was undertaken on GE (Signa LX, GE Medical Systems) or Philips (Intera, Philips Medical Systems, Best, The Netherlands) systems operating at 1.5T, employing a circularly polarised head coil and using standard sequences (axial and coronal T_2 fast spin echo, axial fluid-attenuated inversion recovery (FLAIR), axial and sagittal T_1 SE, and T_2* susceptibility imaging). The images were reviewed independently by the two neuroradiologists, who were blinded to the clinical information and obstetric history. Lack of agreement on the imaging findings was discussed and a consensus opinion adopted for the final position.

Results

The individual case histories are given below. The demographics and clinical data for the five cases are summarised in Table 1. The ultrasound, CT and MRI findings are described in Table 2.

Case 1

Labour was induced at 36 weeks' gestation following 6 weeks of maternal pre-eclampsia. The pregnancy was otherwise uneventful. The child underwent a difficult delivery, first with ventouse (failed) then with forceps (successful). At birth, the baby did not require resuscitation; however, neck bruising and a lump at the base of the head were noted. He was discharged home at day 8, having undergone phototherapy for jaundice. He was re-admitted to hospital with apnoeas and following episodes of post-feed cyanosis at 3 weeks of age. A nasopharyngeal aspirate grew parainfluenza; a lumbar puncture, cardiac echocardiography and electroencephalogram (EEG) were normal. The child subsequently underwent departmental cranial ultrasound (Figure 1a,b), CT (Figure 2a) and MRI (Figure 3a) examinations. At 5-month follow-up, no developmental or neurological deficits were detected and he was discharged.

Case 2

This patient underwent a difficult forceps delivery following an uneventful pregnancy. He presented at day 2 of life with right-sided arm and leg seizures, which did not respond to phenobarbitone and were eventually controlled with paraldehyde. A septic screen, lumbar puncture and EEG were normal. Initial ultrasound imaging (undertaken by a neonatal intensive care unit physician) was interpreted as showing no significant abnormality. The child's symptoms settled and he was discharged. The child was electively admitted for CT scanning 8 weeks post-discharge (Figure 2b); MRI was performed as an outpatient at 8 months of age to assess the brain following appropriate brain development (Figure 3b). The child had no focal neurological deficits at outpatient follow-up examinations at 3 months and 1 year of age.

Case 3

This child was born following a difficult forceps delivery performed under general anaesthesia for shoulder dystocia. The baby had no respiratory effort at birth and absent recordable heart beat. She was intubated and ventilated. The first respiratory efforts were at 5 min. She was transferred to the neonatal intensive care unit and was put on continuous positive airway pressure (CPAP) immediately following extubation. No focal neurological deficit was observed and there were no signs of encephalopathy. A cranial ultrasound was undertaken by the neonatal unit paediatric intensivists and interpreted as normal, however MRI (Figure 3c) performed at 6 weeks of age proved abnormal. At 10 months of age, she had normal tone and movements and exhibited normal developmental progress. She was subsequently discharged.

Case 4

During pregnancy, there were two episodes of antepartum haemorrhage at 18 and 30 weeks' gestation. Delivery occurred at 35 weeks and was uncomplicated with no record of intrapartum difficulty or instrumental delivery. The child was referred at 50 days of age with increasing head circumference (75th percentile at birth, >98th at referral). There was no other neurological

Table 1. Obstetric and clinical features

Case	1	2	3	4	5
Maternal status[a]	P0 G1	P0 G1	P0 G2	P0 G1	P0 G1
Antepartum factors	None	Maternal hypertension	None	Two episodes of ante-partum haemorrhage	None
Birth weight (kg)	2.45	3.4	4.1	2.4	3.52
Sex	Male	Male	Female	Male	Female
Apgar scores (at 1, 5 and 10 min)	9/10/10	9/10/–	0/4/–	9/9/10	6/9/10
Gestational age at delivery	36 w	40 w	41 w 6 d	35 w	40 w
Delivery route	Vaginal	Vaginal	Vaginal	Vaginal	C-section
Instrumentation	V and F	F	F	No	No
Resuscitation or ventilation	No	No	Ventilation and intubation	No	No
Jaundice	Yes	No	No	No	No
Scalp abnormality	Yes	No	Caput and moulding	No	No
Neurology at presentation	Stiff upper limbs, eye deviation	Seizures	None	Rapid increase in head circumference	Seizures
Neurology at latest follow-up	None, discharged	None	None	None	None
Clotting	Normal	Not tested	Not tested	Normal	Not tested

P, para; G, gravida; V, ventouse; F, forceps; Y, yes; N, no; w, weeks.
[a]Gravida indicates the total number of times a woman has been pregnant, regardless of whether these pregnancies were carried to term. Para indicates the number of viable (>24 weeks) births.

Table 2. Imaging findings

Case	1	2	3	4	5
Ultrasound findings					
Figure	Figure 1a,b			Figure 1c,d	
Timing of scan	22 d	2 d	6 d	7 w	2 d
Echodensity	Mixed hyper/hypo- echoic	Normal	Normal	Hypoechoic	Normal
Subdural collection	No	No	No	No	No
Calvarial/scalp abnormalities	No	No	No	No	No
Cortical tear	Yes	No	No	Yes	No
Adjacent parenchymal changes	Hyperechoic	No	No	Hyperechoic	No
CT findings					
Figure	Figure 2a	Figure 2b		Figure 2c	
Timing of scan (days or weeks after birth)	23 d	8 w		7 w	
Attenuation	Low (CSF)	Low (CSF)		Low (CSF)	
Subdural collection	No	No		No	
Parenchymal haemorrhage	Deep white matter, punctate. None perilesional	No		No	
Calvarial or scalp abnormalities	Yes (scalp swelling)	No		No	
Cortical tear?	Yes	Yes		Yes	
Adjacent parenchymal oedema or gliosis	No	No		No	
Cerebral atrophy	No	Yes, local		No	
Dysmorphic features	No	No		No	
MRI findings					
Figure	Figure 3a	Figure 3b	Figure 3c	Figure 3d	Figure 3e
Time from birth	23 d	8 m, 15 d	1 m, 14 d	7 w	25 d
Location	Left SFG	Left SFG	Right SFG	Right SFG	Right anterior parietal
Cortical disruption	Yes	Yes	Yes	Yes	Yes
Blood products	Yes, deep white matter	No	No	Yes, marginal	Yes, marginal
T_1 characteristic	Low	Low	Low	Low	Low
T_2 characteristic	High	High	High	High	High
CSF characteristics	Yes	Yes	Yes	Yes	Yes
Adjacent gliosis	Yes	Yes	Yes	No	No
Subdural collection	No	No	No	No	No
Local atrophy	Yes	Yes	No	No	No
Dysmorphic abnormalities	No	No	No	No	No
Scalp abnormalities	Yes	No	No	No	No
Skull fracture?	No	No	No	No	No

CSF, cerebrospinal fluid; SFG, superior frontal gyrus; d, days; w,weeks; m, months.

abnormality at presentation. A lumbar puncture showed xanthochromia but was otherwise normal. A departmental cranial ultrasound examination demonstrated a cortical tear and prominent subarachnoid spaces (Figure 1c,d). CT (Figure 2c) and MRI (Figure 3d) were subsequently undertaken and showed no evidence of extra-axial haemorrhage. Skeletal radiographic survey and indirect ophthalmologic examination were normal. At three months of age, he was well and his head circumference was on the 50th percentile.

Case 5

This infant presented at 2 h of age with seizures. There were no other signs of encephalopathy or abnormal neurology. The blood sugar, serum sodium, calcium, magnesium and metabolic screen were all normal. There was no evidence of infection and a lumbar puncture was normal. The pregnancy had been normal but there was failure to progress in the second stage of labour associated with signs of fetal compromise, which led to an emergency Caesarean section. At birth, the baby did not require resuscitation and was admitted to the postnatal ward. The seizures were controlled with two doses of phenobarbitone and subsequently resolved. Cranial ultrasound undertaken by the neonatal intensive care physicians was interpreted as being normal. She was discharged home at 4 days of age and an MRI scan was performed on day 25 (Figure 3e). At follow-up at 2 months of age, there are no abnormal neurological signs.

Discussion

Five patients are described who presented with a similar pattern of brain abnormality on their brain

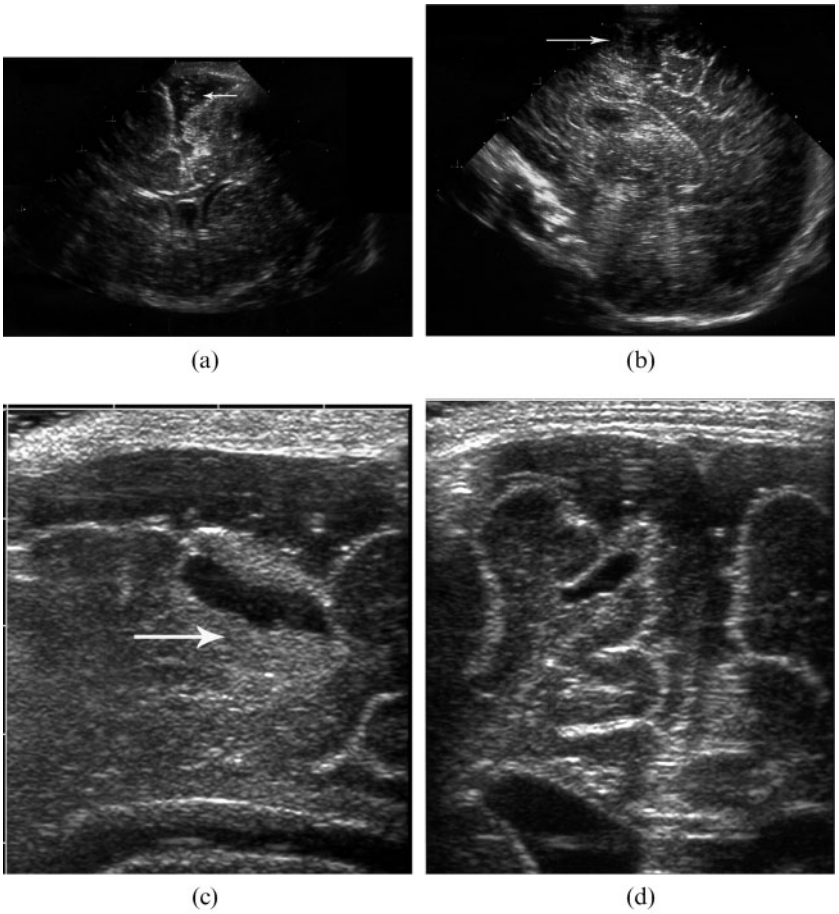

Figure 1. Ultrasound appearances of cortical tears. (a,b) Case 1. Cortical tear in left superior frontal gyrus. The adjacent brain parenchyma is mildly hyperechogenic on the coronal image. (c,d) Case 4. Hypoechoic cortical tear in right superior frontal gyrus. The surrounding parenchyma is hyperechogenic.

imaging. The central abnormality was a deep cortical tear or cleft, located in the frontal or parietal cortex. In all five cases, these lesions displayed cerebrospinal fluid (CSF)-like characteristics and were not associated with skull fracture or subdural haematoma. This pattern of isolated injury has not been previously described. Four out of the five patients underwent a difficult instrumental delivery (two with forceps, one with ventouse followed by forceps, one with forceps followed by Caesarean section).

In these children, birth trauma seems the most likely aetiology of the cortical tears. The clinical presentation of these children is similar to that described in the literature in children who have suffered birth-related intracranial injury. One study of cranial injuries in newborn infants showed that these children present most commonly with low Apgar scores, apnoeas and seizures [2]. These were all features of the children included in this study, suggesting the link to birth-related trauma.

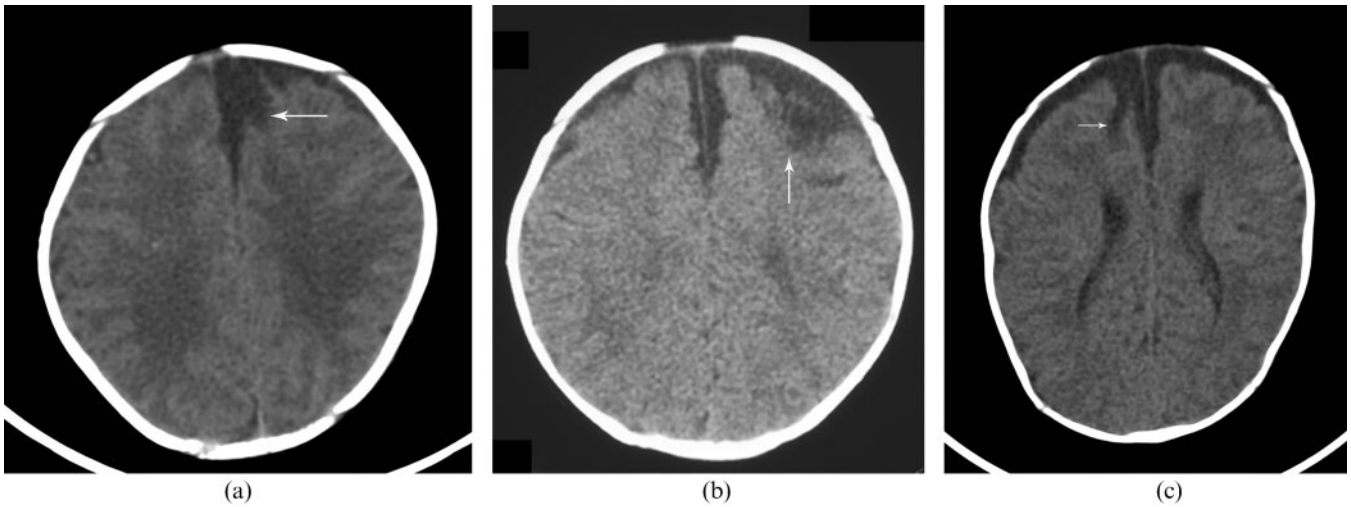

Figure 2. CT appearances of cortical tears. (a) Case 1. Cortical tear in the left superior frontal gyrus. There is deep white matter of high attenuation in the right centrum semiovale, which may represent either calcification or haemorrhagic change. (b) Case 2. Cortical tear in the left superior frontal gyrus with local cerebral atrophy. (c) Case 4. Cortical tear in right superior frontal gyrus.

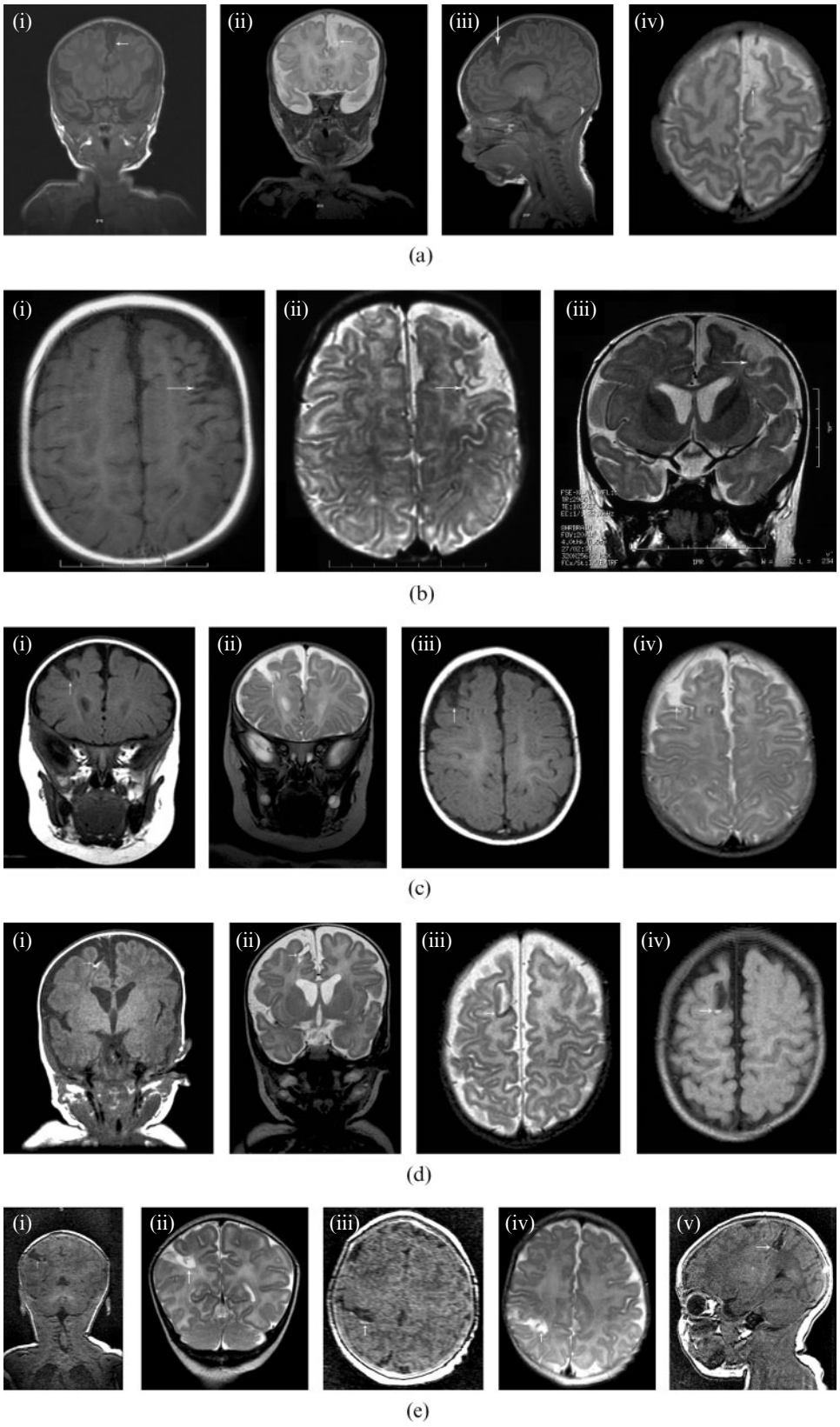

Figure 3. MRI appearances of cortical tears. (a) Case 1. (i) Coronal T_1 and (ii) coronal T_2 weighted images showing a cortical tear in the left superior frontal gyrus. (iii) Sagittal T_1 weighted image. (iv) Axial T_2 weighted image. (b) Case 2. (i) Axial T_1 and (ii) axial T_2 weighted images demonstrating a cortical tear in the left superior frontal gyrus. (iii) Coronal T_2 weighted image. (c) Case 3. (i) Coronal T_1 and (ii) coronal T_2 weighted images demonstrating a cortical tear in the right superior frontal gyrus. (iii) Axial T_1 and (iv) axial T_2 weighted images. (d) Case 4. (i) Coronal T_1 and (ii) coronal T_2 weighted images demonstrating a cortical tear in the right superior frontal gyrus. (iii) Axial T_2 weighted image and (iv) axial FLAIR image. Blood products can be seen at the edge of the tear as T_1/FLAIR (fluid-attenuated inversion recovery) hyperintensity and T_2 hypointensity. (e) Case 5. (i) Coronal T_1 and (ii) coronal T_2 weighted images demonstrating a cortical tear in the right anterior parietal lobe. (iii) Axial T_1 and (iv) axial T_2 weighted images. (v) Sagittal T_1 weighted image. Diffusion weighted imaging in this patient showed no evidence of restricted diffusion.

In all the children described in our series, there was no evidence of another cause for the cerebral lesions.

The link between instrumental delivery and intracranial injury and haemorrhage is well described. Two large studies showed that the risk of birth-related intracranial injury was markedly increased with instrumental delivery. Both studies showed little difference between forceps and ventouse delivery [2, 4]. Specific risk factors associated with ventouse-associated fetal neurological injury include nulliparity, poor placement of the vacuum cup and exceeding the safe limit for traction force [5]. A wide spectrum of injuries can complicate instrumental delivery, cephalhaematoma, skull fractures and extra-axial haemorrhage being the most common [2]. Intraparenchymal haemorrhage complicating birth trauma is relatively uncommon; where it is mentioned, it is generally poorly characterised in the literature. One detailed account of intracranial haemorrhage in term newborns describes a series of infants who suffered frontal lobe haemorrhage [6]. In these cases, this was largely associated with extracerebral haematoma and cortical sparing. This type of lesion is different to that seen in our series and no definite relationship with birth injury was demonstrated in these infants.

More recently, spontaneous superficial parenchymal hemorrhage with a predilection for the temporal lobes has been described in a small cohort of term infants [7]. Huang and Robertson [7] described seven otherwise healthy infants delivered vaginally who presented with apnoea and/or seizures within 48 h of birth. The imaging revealed superficial parenchymal haemorrhage involving the temporal lobe in five cases, the haemorrhages in the remaining two children being located in the frontal and parietal lobes. Common features in this series were accompanying leptomeningeal haemorrhage, overlying scalp soft tissue swelling and localisation of the haemorrhage close to a suture. Although apnoea or seizures occurred in three of the five infants in our case series, the other described features, in particular predilection for the temporal lobes, scalp swelling and leptomeningeal haemorrhage, did not feature in our cohort. Huang and Robertson [7] concluded that local trauma and contusion or venous compression or occlusion were aetiologically responsible for the injuries in their series.

No skull fractures were noted in our series. Although two children underwent delayed CT imaging, the interval between scanning and birth was sufficiently short (less than 7 weeks at most) to allow exclusion of a fracture, as fractures generally take 3–6 months to heal in infants. The cortical tear was located adjacent to a suture line in our series, which might implicate a traumatic sutural injury. Skull fractures were also absent in the cohort of children described by Huang and Robertson [7]. Venous compression adjacent to a suture, postulated as a possible mechanism by this group, may be a common aetiological factor in both series.

The differential diagnosis of this lesion pattern includes:

1. *Neonatal cortical infarcts*. Several causes exist such as bacterial meningitis, coagulopathies, and hypoxia-ischaemia [8]. They differ from the cortical lesions seen in our series in their morphology; deep clefts are not a recognised feature of infarcts. If a cortical tear is associated with a deep cleft, the diagnosis is more definite. A birth-related cortical tear that does not extend deep into the cortex is more difficult to differentiate from an old infarct. In addition, such aetiological conditions had been excluded in our cases. Cerebral infarction is recognised as a rare complication of instrumental delivery. It usually occurs on the side of vacuum application, often in association with cephalhaematomas, although variations on this have been reported, such as contralateral infarction [9].

2. *Cerebral venous infarction*. Although cortical infarcts may be seen, which do not conform to an arterial territory, the ischaemic lesions are usually subcortical in location and do not cause the deep cortical clefts observed in our series [10].

3. *Schizencephaly* is characterised by a deep cortical cleft. In contrast to the cases presented in our series, however, the cleft extends to the ventricular system and is lined by dysplastic grey matter [11]. Other associated features of schizencephaly, such as polymicrogyria, heterotopic grey matter and pachygyria, are absent in our children. The presence of blood products in our children's clefts on MRI also supports an alternative aetiology.

4. *Prenatal head injury*. Trauma to the mother's abdomen during pregnancy can also rarely lead to head injury *in utero* [12], including both intra- and extra-axial intracranial haemorrhage and cerebral contusion.

5. *Postnatal head injury*. It is important for clinicians and radiologists to be aware of the existence of isolated cerebral cortical tears, particularly as they may potentially be confused with brain injuries associated with NAHI. Of note, all of the children in our series had relatively mild neurological symptoms at presentation and all had no residual symptoms at follow-up.

Cerebral contusional tears of subcortical white matter are a recognised phenomenon in non-accidental injury [13]. A similar imaging abnormality is shared by all of the children in our series except that the lesions in these cases transgressed the cerebral cortex. In cases of NAHI, additional injuries (particularly subdural haematoma) will be present in most if not all cases.

Careful assessment is required to exclude conditions that may mimic non-accidental injury. Beyond the newborn period, severe traumatic intracranial injury in infancy is most commonly non-accidental in aetiology [14]. Domestic falls and minor accidental injuries do not usually cause severe intracranial events [15]. Cortical contusion is recognised as a complication of accidental trauma, which results from impact of the brain against bone at the site of impact (coup injury) or opposite to it (contracoup injury) [16]. A history of an appropriate major accidental injurious event will, however, be forthcoming in these cases.

Although some of the scans in our series were obtained after the neonatal period, none of the children in this study demonstrated evidence of subdural haemorrhage (SDH) on their imaging. One recent study has shown intracranial haemorrhage to be quite uncommon, even in instrumental deliveries, with an incidence of 1 in 664 of forceps deliveries [17] and 1 in 2750 spontaneous deliveries. However, two more recent

studies have demonstrated a higher incidence of occult SDH. Whitby et al [18] performed MRI at birth following normal vaginal delivery, instrument-assisted delivery or Caesarean section, recording 9 (8.1%) cases of small posteriorly located SDHs in 111 babies. No parenchymal haemorrhages were recorded in this study. Looney et al [19] found intracranial haemorrhage on comprehensive MR imaging in 17 (17.3%) of 88 neonates following vaginal delivery (65 cases) or Caesarean section (23 cases), imaged between 1 and 5 weeks after birth. In all cases, the intracranial haemorrhages occurred following vaginal delivery, giving a prevalence of 26% in vaginal births. The intracranial haemorrhages in this study consisted of 16 subdural, 2 subarachnoid and 6 parenchymal haemorrhages. The parenchymal haemorrhages in this study all occurred in the temporal lobes, occipital lobes, periventricular region or germinal matrix, and were associated with subdural haematomas in five out of six cases.

Routine cranial ultrasound scanning at birth is of limited value, particularly when undertaken by neonatology unit physicians whose primary goal is to detect periventricular haemorrhage and ventricular size in the context of prematurity. Although high-resolution ultrasound scanning undertaken by an experienced neuroradiologist was able to detect these lesions in two of the cases in our series, the other three cases had ultrasound examinations that were interpreted as being normal by a non-radiologist. Nevertheless, ultrasound remains a useful cot-side tool for the first-line evaluation of the intracranial contents in the unstable or critically ill neonate. The cortical lesions in our study are likely to be situated in an acoustically blind area of the brain if off-centred or if tangential plane scanning is not employed in addition to high-resolution near-field scanning.

More definitive imaging is required when there is significant concern regarding encephalopathy or neurological disturbance in the setting of birth-related trauma. CT is quicker to perform than MRI and is useful in the acute setting but has the disadvantage of carrying a significant radiation dose burden. MRI is clearly the most sensitive modality for diagnosing pathology, particularly when subtle, and for differentiating the causes of intracranial abnormality described above [9].

Conclusions

We present the imaging findings in five children who had cerebral cortical tears on MRI examination. None of the children demonstrated subdural collections on their imaging and none were accompanied by any clinical or social risk factors relating to inflicted injury. Birth trauma is likely to be an important aetiological factor. Awareness of the existence of this phenomenon may be important in the assessment of children with suspected NAHI who undergo cranial imaging. The need for a detailed assessment of the birth history is emphasised should any such assessment be undertaken. We emphasise the need for high-quality cerebral imaging to exclude craniocerebral trauma and other significant associated pathology in newborn infants presenting with seizures.

References

1. Little WJ. On the influence of abnormal parturitions, difficult labours, premature birth and asphyxia neonatorum, on the mental and physical condition of the child especially in relation to deformities. Trans London Obstetrical Soc 1862;3:293–344.
2. Pollina J, Dias MS, Li V, Kachurek D, Arbesman M. Cranial birth injuries in term newborn infants. Pediatr Neurosurg 2001;35:113–9.
3. Oehmichen M, Meissner C, Saternus K-S. Fall or shaken: traumatic brain injury in children caused by falls or abuse at home — a review on biomechanics and diagnosis. Neuropediatrics 2005;36:240–5.
4. Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. N Engl J Med 1999;341:1709–14.
5. Doumouchtsis SK, Arulkumaran S. Head injuries after instrumental vaginal deliveries. Curr Opin Obstet Gynecol 2006;18:129–34.
6. Govaert P. Cranial haemorrhage in the term newborn infant. Cambridge: Cambridge University Press, 1993.
7. Huang A, Robertson RL. Spontaneous superficial parenchymal and leptomeningeal hemorrhage in term neonates. Am J Neurorad 2004;25:469–75.
8. Miller V. Neonatal cerebral infarction. Semin Pediatr Neurol 2000;7:278–88.
9. Kumar M, Avdic S, Paes B. Contralateral cerebral infarction following vacuum extraction. Am J Perinatol 2004;21:15–7.
10. Wasay M, Azeemudin M, Neuroimaging of cerebral venous thrombosis. J Neuroimaging 2005;15:118–28.
11. Oh KY, Kennedy AM, Frias AE, Byrne JL. Fetal schizencephaly: pre- and postnatal imaging with a review of the clinical manifestations. Radiographics 2005;25:647–57.
12. Breysem L, Cossey V, Mussen E, Demaerel P, Van de Voorde W, Smet M. Fetal trauma: brain imaging in four neonates. Eur Radiol 2004;14:1609–14.
13. Jaspan T, Narborough G, Punt JAG, Lowe J. Cerebral contusional tears as a marker of child abuse — detection by cranial sonography. Pediatr Radiol 1992;22:237–45.
14. Billmire ME, Myers PA. Serious head injury in infants: accident or abuse? Pediatrics 1985;75:340–2.
15. Kleinman PK Barnes PD. Head trauma. In: Diagnostic imaging of child abuse. St. Louis: Mosby; 1998.
16. Woodcock RJ, Davis PC, Hopkins KL. Imaging of head trauma in infancy and childhood. Semin Ultrasound CT MR 2001;22:162–82.
17. Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. New Eng J Med 1999;341:1709–14.
18. Whitby EH, Griffiths PD, Rutter S, Smith MF, Sprigg A, Ohadike P, et al. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. Lancet 2004;363:846–51.
19. Looney CB, Smith JK, Merck LH, Wolfe HM, Chescheir NC, Hamer RM, et al. Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. Radiology 2007;242: 535–41.

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References:
1. Gacci M et al. Bladder Instillation Therapy with Hyaluronic Acid and Chondroitin Sulphate Improves Symptoms of Postradiation Cystitis: Prospective Pilot Study. Clin Genitourin Cancer 2016; Oct;14(5):444-449. 2. Giannessi C et al. Nocturia Related to Post Radiation Bladder Pain can be Improved by Hyaluronic Acid Chondroitin Sulfate (iAluRil). Euro Urol Suppl 2014; 13: e592. 3. UK IQVIA data (accessed August 2024)

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