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Cerebral contusional tears as a marker of child abuse – detection by cranial sonography

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Abstract. A series of 6 infants subjected to child abuse is presented in whom contusional tears of subcortical white matter were detected during life by intracranial sonography. The sonographic appearances of this highly pathognomonic marker of shaking injury are described for the first time and their significance discussed. On the basis of our experience we suggest that high resolution cranial sonography is an extremely valuable part of the diagnostic work up in cases of suspected non-accidental injury.

Skull vault fractures and injury to the intracranial contents are common sequelae of child abuse and account for the greatest associated morbidity and mortality [1–5]. The post mortem appearances of white matter contusional tears in young infants subjected to child abuse have been described by pathologists for many years [5–8]. Computerized tomography (CT) has, over the past two decades, been the commonest method of assessing the brain in cases of suspected non-accidental injury (NAI) [9–12]. Magnetic resonance imaging (MRI) with it's greater imaging potential has been employed more recently [13, 14]. The CT findings mainly consist of subdural collections, subarachnoid haemorrhage, cerebral oedema and cerebral contusions. Cerebral lacerations not associated with an adjacent fracture have rarely been identified by CT, though recently they have been documented by MRI [14]. It has been stated that ultrasound is unable to demonstrate these lesions [15]. At our institution, a detailed ultrasound examination is performed routinely in all head injured infants. Near field imaging of the brain surface, with a spatial resolution of less than one millimeter, is now possible with the current generation of ultrasound scanners. We present our findings in six cases of verified child abuse who exhibited highly characteristic contusional tears which were detected by high resolution cranial sonography.

Patients and methods

All six patients in this report were under six months of age at presentation. Five of the patients were referred to the regional paediatric neurosurgeon as cases of suspected child abuse in whom there was evidence of a skull fracture or intracranial injury. The sixth case was suspected of having suffered non-accidental injury retrospectively.

In cases 1 to 5 a skeletal radiographical survey was performed on admission and external injuries documented or photographed. The sixth case underwent a skeletal survey prior to the post mortem examination. All cases were examined by sonography on an Acuson 128 unit (Mountain View, California) within 24 h of admission and at varying intervals during their hospital stay thereafter. A routine examination was initially performed with a 5 MHz sector transducer through the anterior fontanelle, imaging in conventional coronal and sagittal planes. A transcranial study was then performed with a 3 MHz sector transducer. Images in the transverse axial and coronal planes were obtained, specifically looking for subdural or extradural collections over the opposite cerebral convexity and in the infratentorial compartment. Finally a high resolution examination of the near field underlying the anterior fontanelle was undertaken in the coronal and sagittal planes with a 7 MHz linear array transducer.

In 4 cases the examinations were performed at the cot side in the paediatric intensive care unit, the infants being too ill to be transported to the radiology department. In 3 cases sonography had been performed at the referring hospital prior to transfer. In each case a high resolution study had not been performed and the only abnormality detected was a subdural haematoma in two of the patients.

Acute CT scans (Picker 1200 SX scanner) were obtained in 2 of the cases one of whom also underwent acute MR imaging (Picker Vista 0.15 T resistive unit). In one patient an acute phase MR scan alone was obtained, CT scanning being performed 5 months later and in a further case, in whom the child was felt to be too ill to transfer to the imaging suite, only a delayed CT scan was obtained four months after the injury. One infant was too ill to be transferred to the radiology department for CT scanning and died 52 hours following admission. CT or MR imaging was not performed in case 6, in whom NAI was not initially suspected, as he was initially felt to be too ill to be transferred for further imaging. The child eventually recovered and was discharged from hospital and lost to follow up. The child was subsequently brought to the hospital and pronounced dead on arrival 6 months later.

Results

Table 1 documents the presentation of these cases, associated injuries and outcomes. Table 2 shows the ultrasound and, when performed, CT and MRI findings for each patient. A resume of each case is given below.

Table 1

Case	Age	Sex	Presentation	Injuries	Skull fracture	Neurology
1	14 weeks	F	Convulsions Drowsiness	Bruising: face, torso Fractures: ribs, limbs of differing ages Retinal haemorrhages: Yes Fontanelle: Tense	No	Drowsy Irritable
2	3 weeks	F	Drowsiness Failure to feed	Bruising: face, neck Fractures: right tibia Retinal haemorrhages: No Fontanelle: Full	Right parietal	Irritable Seizures Decorticate rigidity
3	4 months	M	Developmental regression	Bruising: No Fractures: ribs, limbs of differing ages Retinal haemorrhages: No Fontanelle: Tense	Left parietal	Drowsy Seizures
4	9 weeks	M	Found blue and stiff, malnourished Hypothermic	Bruising: No Fractures: ribs, limbs Retinal haemorrhages: Yes Fontanelle: Tense	Bilateral occipital Right parietal	Divergent squint Right gaze fixation
5	6 weeks	F	Unrousable Malnourished	Bruising: face, torso limbs Fractures: ribs, limbs of differing ages Retinal haemorrhages: Yes Fontanelle: Tense	Bilateral parietal	Unresponsive Ventilated Seizures
6	4 months	M	Unrousable Cyanosed ? near miss cot death	Bruising: no Fractures: NK Retinal haemorrhages: NK Fontanelle: Tense	NK	Unresponsive Ventilated Seizures

NK: Not Known

Table 2

Case	US	CT	MRI	Clinical outcome	Legal outcome
1	R. parietal SDH Bilat. frontoparietal tears Late cortical thickening	Severe atrophy (4 month scan)	ND	6 month assessment: hypertonic limbs, global p/m retardation Visual impairment	In local authority care Criminal proceedings pending
2	Bilat. posterior frontal tears Deep white matter echogenicities	Ill-defined white matter low densities	Deep white matter hyperintensities on T2WIs	8 month assessment: hypertonic legs Seizures Mild p/m retardation	Foster care Criminal proceedings pending
3	Large convexity SDHs Bilat. frontoparietal tears	1. Bilat. SDHs 2. Late atrophy	Bilat SDHs Hyperintensities on T1WIs	16 month assessment: Spastic quadriplegia Global motor retardation	Protected care Criminal proceedings
4	Thin R. SDH R. posterior frontal tear	R. convexity SDH	ND	8 month assessment: Early spastic diplegia	Foster care Criminal proceedings
5	Multiple tears in both frontal and parietal areas Deep white matter echogenicities Diffuse swelling	ND	ND	Died	Criminal proceedings
6	Multiple tears posterior frontal & deep white matter echogenicities	ND	ND	5 month assessment: Hypertonic limbs p/m retardation Brought in dead after 1 week	Criminal proceedings

p/m = psychomotor
T1WI = T1 weighted image
T2WI = T2 weighted image
ND = Note done
SFH = Subdural haematoma

Case 1

(JR). Spontaneous vaginal delivery (SVD) following a 28 weeks gestation. Several complications associated with prematurity occurred, however at 10 weeks of age she was well enough to be discharged home. Four weeks later she was readmitted with a history of convulsions. Examination showed her to be drowsy and irritable with widespread bruising, a tense fontanelle and a head circumference above the 97th percentile. Cranial sonography showed a right sided convexity subdural haematoma and several linear mixed but essentially transonic lesions located at the grey-white interface of both frontal and parietal regions (Fig. 1 a–e). Fresh blood was aspirated from the subdural collection immediately after the scan. Serial scanning over the ensuing three weeks demonstrated an evolving appearance of extensive cortical echogenicity (Fig. 1 f) and subsequently atrophy. A CT scan 4 months later demonstrated end stage atrophy and infarc-

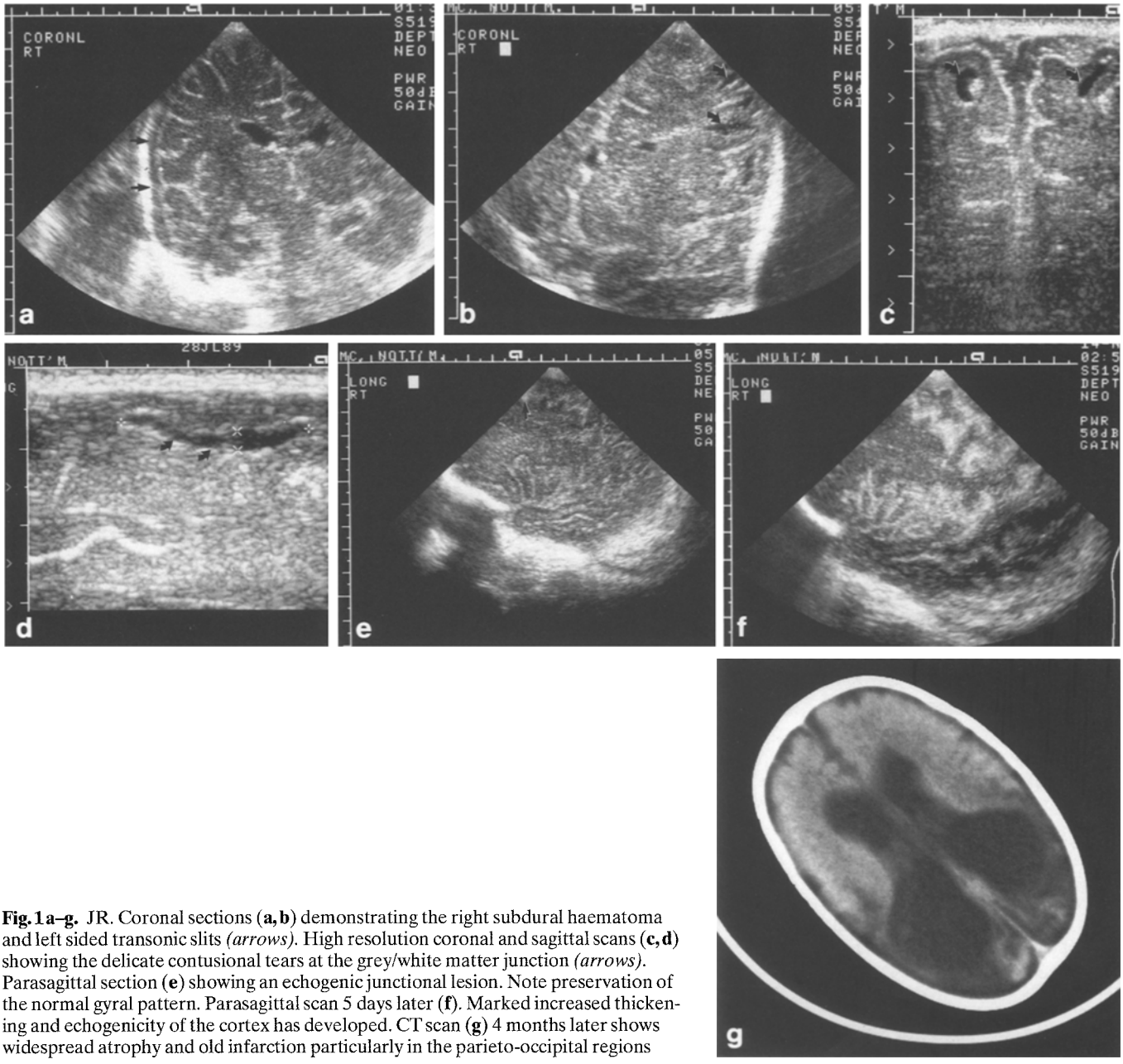

Fig. 1a–g. JR. Coronal sections (**a,b**) demonstrating the right subdural haematoma and left sided transonic slits *(arrows)*. High resolution coronal and sagittal scans (**c,d**) showing the delicate contusional tears at the grey/white matter junction *(arrows)*. Parasagittal section (**e**) showing an echogenic junctional lesion. Note preservation of the normal gyral pattern. Parasagittal scan 5 days later (**f**). Marked increased thickening and echogenicity of the cortex has developed. CT scan (**g**) 4 months later shows widespread atrophy and old infarction particularly in the parieto-occipital regions

tion (Fig. 1g). Clinical assessment six months after presentation revealed global developmental retardation, spastic quadraplegia and severe visual impairment. The child is currently in the care of the local authority.

Case 2

(KF). Term baby, SVD. At the age of three weeks this child was brought into the casuality department at the referring hospital with bruising of her face and head. The child was irritable but there were no other clinical features of note. No explanation of the bruising was offered by the parents. A skull radiograph showed a right parietal fracture. The initial CT scan showed ill-defined low attenuation change in the sub-cortical and deep white matter of both frontal and parietal lobes. The following day the baby showed evidence of decorticate rigidity and was transferred to the regional paediatric neurosurgical unit. High resolution sonography documented corresponding areas of ill-defined echogenicity in the deep white matter as well as several contusional tears in both frontoparietal regions (Fig. 2a–c). Repeat CT and MR imaging one week later demonstrated the evolution of large foci of white matter damage in the abnormal areas shown on sonography with evidence of extensive cerebral swelling (Fig. 2d–f) however both CT and MRI failed to demonstrate the delicate cortico-medullary junction tears. The child survived and is currently in protected care. Six months after presentation the child has a seizure disorder controlled with phenobarbitone and is showing signs of mild developmental retardation. There is sustained clonus in both lower limbs which are hypertonic.

Case 3

(NT). This child was born 4 weeks premature following an uneventful vaginal delivery. At the age of 4 months his general practitioner referred him to the local hospital with signs of developmental regression and lack of responsiveness. The anterior fontanelle was tense. Skeletal radiographs showed evidence of healing fractures of differ-

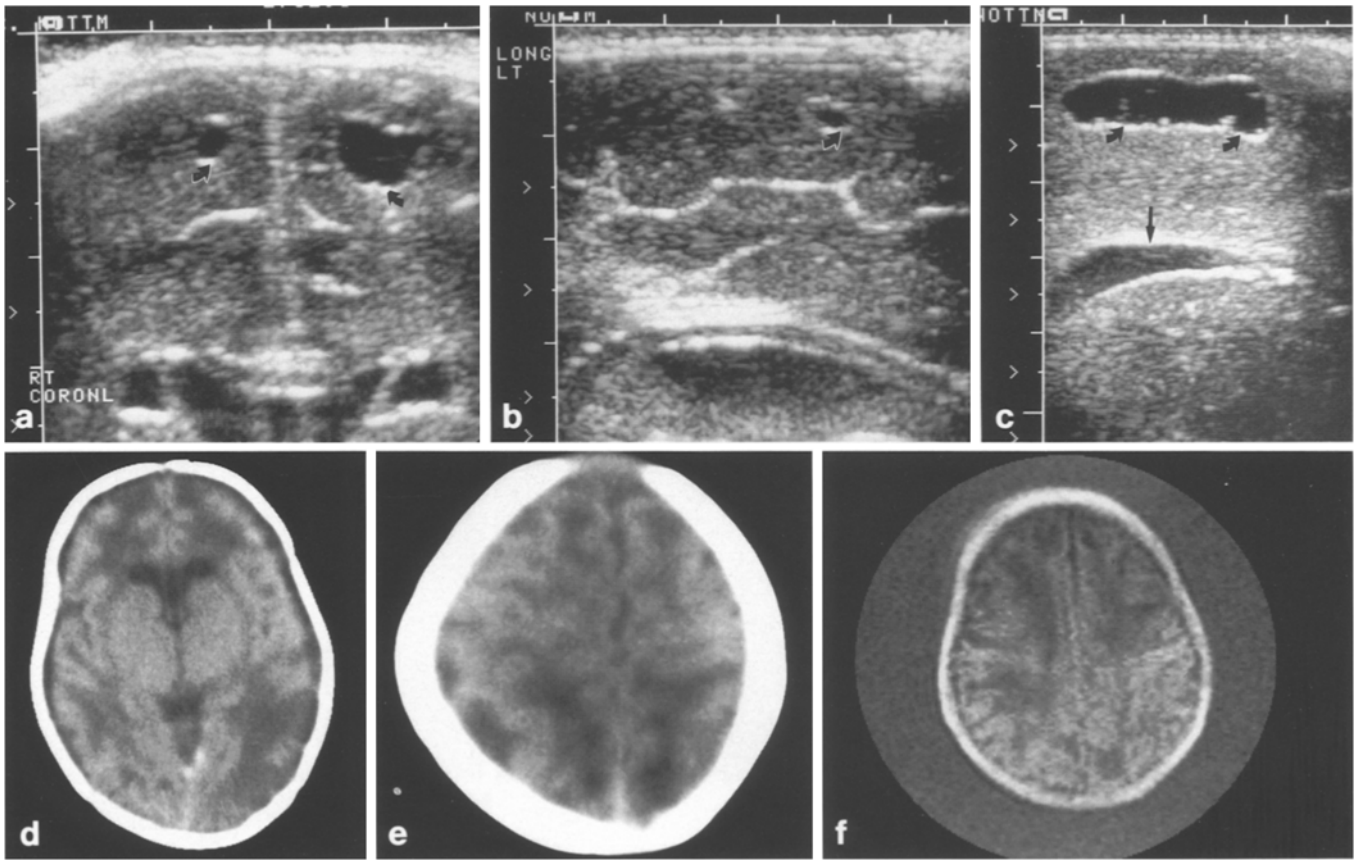

Fig. 2 a–f. KF. Coronal, left and right parasagittal sonograms (**a–c**) showing irregular clearly defined transonic fissures in both fronto-parietal regions *(arrows)*. Axial CT scans (**d, e**) one week later show diffuse low attenuation change in the cortical, subcortical and deep white matter regions. Axial T1 weighted inversion recovery MRI scan (**f**) showing ill defined low signal intensity lesions in the fronto-parietal regions

ing ages and sonography revealed large bilateral subdural haematomas. The parents denied all knowledge of the nature and causation of the injuries. Following transferral to the QMC, repeat cranial sonography confirmed the presence of large mixed echogenicity subdural haematomas (Fig. 3 a) and in addition demonstrated multiple small linear echogenic lesions situated at the corticomedullary junction in the frontal and parietal lobes of both cerebral hemispheres with predominant involvement of the left hemisphere (Fig. 3 b). Under ultrasound guidance, the subdural haematomas were aspirated at intervals over the ensuing 2 weeks. MR imaging performed 5 days after admission confirmed the presence of subdural haematomas (Fig. 3 c). Several high signal intensity lesions were seen on the T2 weighted spin echo images in the frontal and parietal regions, corresponding to the lesions demonstrated sonographically (Fig. 3 d). The child survived and was placed in protected care. A CT scan obtained 5 months after admission showed extensive mainly subcortical atrophy, particularly involving the parieto-occipital regions. Clinical assessment at 1 year revealed global motor developmental retardation with a spastic quadriplegia.

Case 4

(PF). Term baby, SVD. At the age of 9 weeks the child was admitted to the referring hospital having been found to be blue and stiff by his mother. There had been two previous admissions with apnoeic episodes and gastro-intestinal disorders. On admission the child was hypothermic, very thin, floppy and lethargic. The anterior fontanelle was bulging and a lumbar puncture demonstrated xanthochromia. Extensive haemorrhages were seen in both eyes. Plain radiographs demonstrated several skull and rib fractures and a healing fracture of the distal right femoral metaphysis. The child was transferred to the QMC with a clinical suspicion of a subdural haematoma. Sonography revealed a thin right sided echogenic subdural collection (Fig. 4 a). High resolution scanning demonstrated a transonic slit like lesion at the cortico-medullary junction of the right posterior frontal region (Fig. 4 b, c). A CT scan performed on the same day confirmed the presence of the subdural haematoma but failed to show the small slit like lesion (Fig. 4 d). The child survived and has been taken into protected custody. An assessment four months later showed a marked increase in the child's weight and well being however there is increased tone in the lower limbs with scissoring and hyperreflexia, features suggestive of early development of a spastic diplegia. Cranial sonography showed the right frontal lobe lesion to have evolved into a residual linear echogenic scar (Fig. 4 e).

Case 5

(JN). Seven weeks premature infant, SVD. The child was admitted directly from the casualty department at the age of six weeks. The parents initially claimed the child was not responding normally and had required "resuscitation" measures on several occasions to "make her breathe properly". On admission the child was found to be malnourished, the weight being below the third percentile. There were several bruises of differing ages on the head, torso and limbs. The child was unresponsive and required intubation for assisted ventilation as well as other resuscitative measures. The anterior fontanelle was bulging however there were no retinal haemorrhages. Cot side cranial sonography demonstrated widespread patchy increased

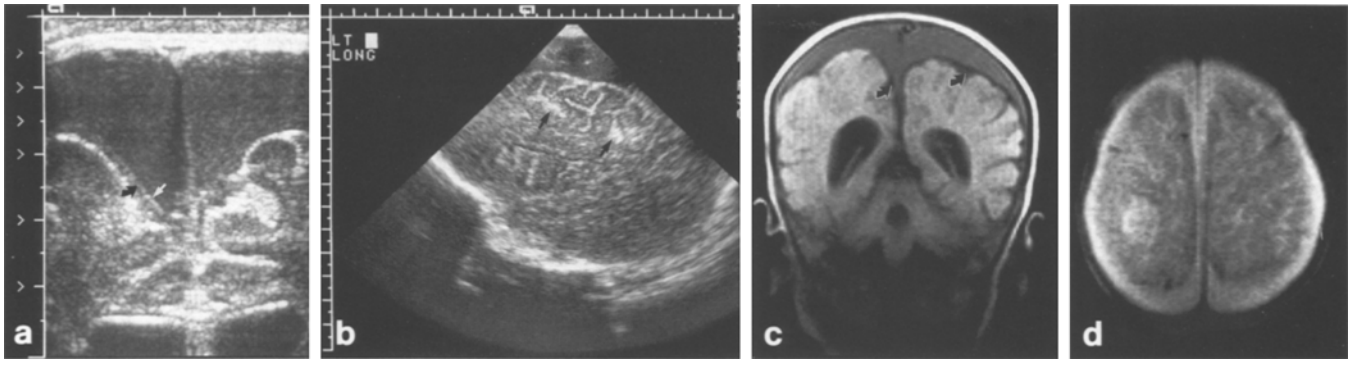

Fig. 3a–d. NT. Coronal section (**a**) demonstrating bilateral mildly echogenic subdural haematomas. The arachnoid mater is seen with a small underlying subarachnoid fluid collection *(arrows)*. Left parasagittal scan (**b**) demonstrating two echogenic lesions in the region of the gyral crests *(arrows)*. Note the subdural haematoma extending over the lateral surface of the brain. Coronal T1 weighted inversion recovery image (**c**) obtained five days after admission demonstrating the mildly hyperintense subdural haematomas and the thin low signal subarachnoid effusions. Axial T2 weighted spin echo sequence (**d**) showing a large hyperintense lesion in the subcortical white matter of the right parietal lobe. Several smaller similar findings are present in the left frontal and parietal regions. The subdural collections appear hyperintense

echogenicity in the white matter of both cerebral hemispheres, obliteration of normal landmarks, several deep focal echogenic lesions and effacement of the ventricular system suggestive of diffuse brain swelling and oedema (Fig. 5a). High resolution imaging showed multiple discrete transonic lesions in both fronto parietal corticomedullary junctional regions (Fig. 5b). In addition there were in addition several strongly echogenic lesions situated at the gyral crests (Fig. 5c). Some of the transonic tears also showed peripheral and/or central echogenicities probably representing small haematomas (Fig. 5d). The lesions varied in length from 5 mm to 24 mm and in width from 2 mm to 14 mm. Doppler analysis showed reversal of flow during diastole indicative of the very high intracranial pressure. A repeat examination 24 h later showed increasing echogenicity of the cerebral hemispheres indicating a probable combination of global cerebral swelling and ischaemic damage. The child failed to respond to further measures and was declared dead 36 h after admission. The father subsequently admitted to several episodes of shaking of the child.

Post mortem examination revealed right second to ninth and left second to eleventh rib fractures and a spiral fracture of the left femoral shaft of approximately three weeks age with further more recent fractures of the left 10th and 11th and right 11th ribs. Several bruises were found in the scalp over the cranial convexity as well as over the right forehead and the left eye. There was no skull fracture. A 2 mm

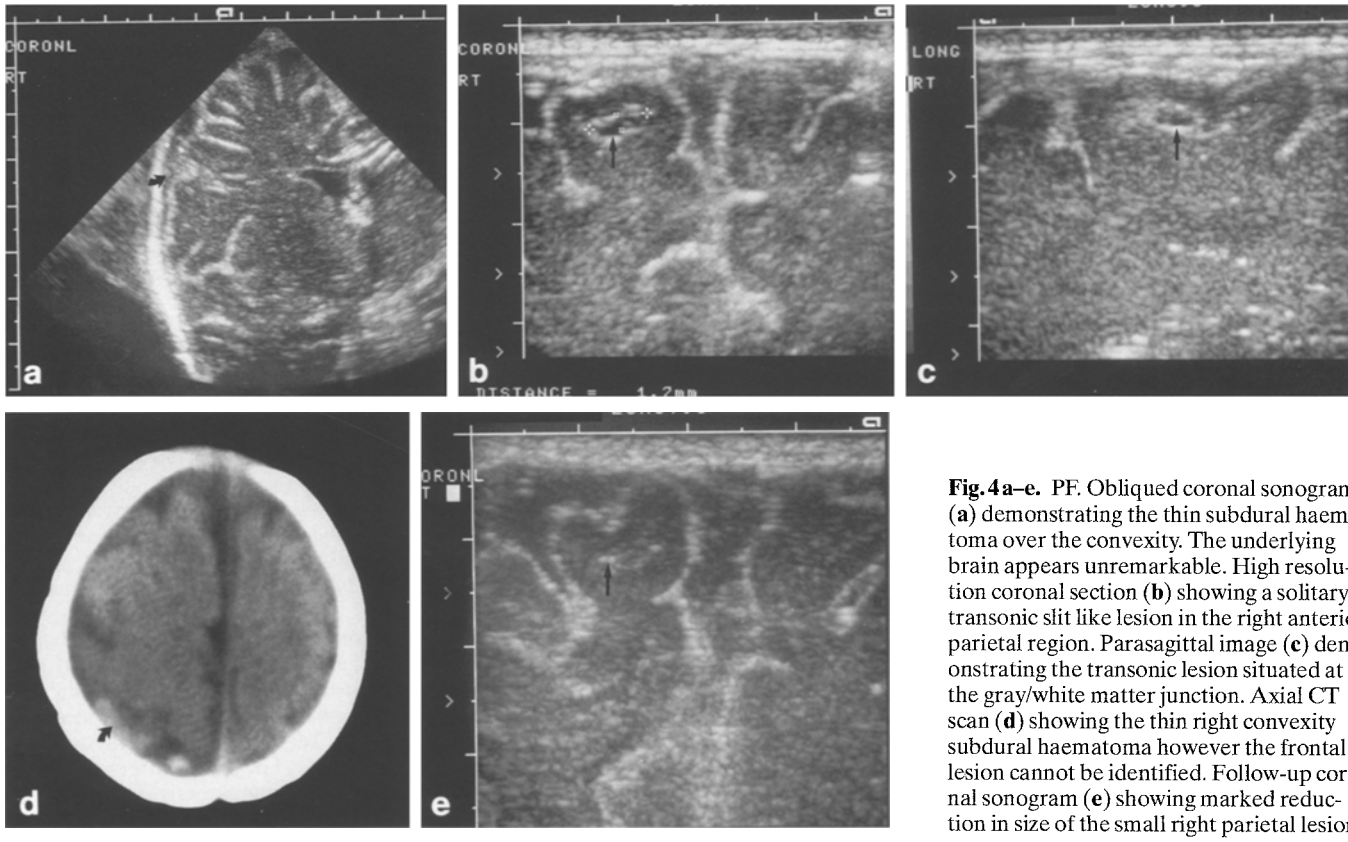

Fig. 4a–e. PF. Obliqued coronal sonogram (**a**) demonstrating the thin subdural haematoma over the convexity. The underlying brain appears unremarkable. High resolution coronal section (**b**) showing a solitary transonic slit like lesion in the right anterior parietal region. Parasagittal image (**c**) demonstrating the transonic lesion situated at the gray/white matter junction. Axial CT scan (**d**) showing the thin right convexity subdural haematoma however the frontal lesion cannot be identified. Follow-up coronal sonogram (**e**) showing marked reduction in size of the small right parietal lesion

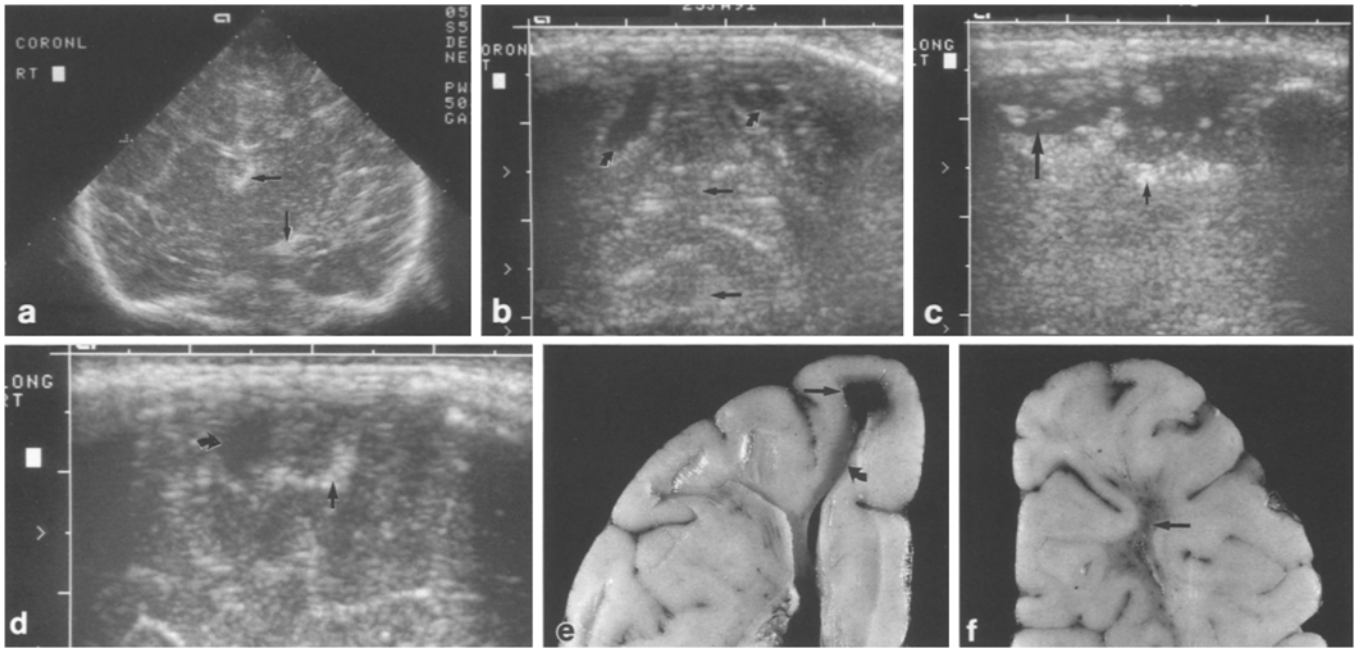

Fig. 5 a–f. JN. Mid plane coronal sonogram (**a**) at presentation showing diffuse hyperechogenicity of the brain. Deep echogenic contusions are demonstrated *(straight arrows)*. High resolution coronal scan (**b**) showing transonic contusional tears *(curved arrows)*. There is loss of visualization of the normal hypoechoic grey matter and ill definition of the sulci and falx *(straight arrow)* due to oedema and swelling. Left (**c**) and right (**d**) parasagittal high resolution scans showing transonic tears *(curved arrows)*, echogenic gyral crest contusions *(short arrows)* and mixed lesions with peripheral echogenicities *(long arrow)*. Coronal post mortem section (**e**) showing an area of sub-cortical haemorrhage *(straight arrow)* in continuity with a linear contusional tear in the white matter *(curved arrow)*. Coronal section (**f**) showing white matter discolouration *(arrow)* and petechial haemorrhages corresponding to deep white matter hyperechogenic areas demonstrated sonographically

deep subdural haematoma was present over both cerebral convexities.

The brain was subsequently examined following fixation in formalin. There was generalised cerebral swelling with diencephalic descent and assymetric herniation of the parahippocampal gyri through the tentorial hiatus. Yellow/brown discolouration of the white matter was seen in the occipital and frontal lobes (Fig. 5e). Small slit-like cavities were present in the hemispheric white matter. The largest, measuring 5 mm in size in the right parietal area, contained blood in a small cavity sited just below the cortex and was in continuity with a slit like tear in the white matter extending radially down to the lateral ventricle (Fig. 5f). Histology confirmed the presence of contusional tears with the visible slit-like cavities showing infiltration by phagocytic microglial cells and adjacent reactive astrocytic gliosis. In addition areas of reactive gliosis and phagocytic macrophages were seen in the sub-cortical white matter indictive of contusional tears which had not been macroscopically evident. Widespread neuronal death was present in the cortex consistent with the effects of cerebral ischaemia consequent upon the raised intracranial pressure.

Case 6

(CS). Term baby of Afro-caribbean ethnic origin, born by spontaneous vaginal delivery. At the age of 4 months the child was admitted to the paediatric intensive care unit in a collapsed state. No relevant antecedent history could be obtained from the mother, a single parent, apart from some recent gastrointestinal upset. There were no features to alert the attending physicians to the possibility of non accidental injury and a skeletal survey was not obtained. The child was cyanotic and required intubation and ventilation. A presumptive diagnosis of a near miss cot death was made. Cranial sonography at the cot side showed widespread but patchy increased echogenicity of the deep cerebral white matter (Fig. 6a). High resolution near field scanning demonstrated focal small echogenic areas at the cortico-medullary junction of both fronto-parietal regions as well as several small linear transonic slit like lesions (Fig. 6b, c). The significance of these findings was not appreciated at the time and the child was assumed to have suffered a global hypoxic/ischaemic episode of unknown origin. Serial ultrasound examinations over the following three weeks demonstrated the development of extensive swelling of the cortex in the fronto-parieto-occipital regions, the cortex appearing abnormally echogenic (Fig. 6d). In addition the numerous focal echogenic lesions in the deep white matter and at the cortico-medullary regions transformed into transonic slit like lesions. The ventricular system became mildly enlarged and associated with this there was an expansion of the subarachnoid spaces (Fig. 6e). In the presence of an increase in the head circumference this was felt to indicate a mild communicating hydrocephalus. After 3 weeks of assisted ventilation the child was weaned off the ventilator and after 4 weeks the child was discharged from hospital. Neurological examination 4 months after discharge indicated severe visual impairment, generalized hypertonicity and gross psychomotor developmental retardation.

Having subsequently scanned infants with similar appearances, the child's sonograms were reviewed six months later and felt to be compatible with a diagnosis of severe non accidental injury. The relevant authorities were contacted immediately however unfortunately the child had been admitted to the casualty department one week previously and pronounced dead on arrival. The child had been battered by the mother's consort who also admitted to previous episodes of abuse of the child. The autopsy findings demonstrated extensive haemosiderin staining of the cerebral convexities and gross cerebral atrophy. The contusional tears could not be identified macroscopically. Histological examination of the formalin fixed brain revealed extensive cortical lamellar necrosis with diffuse replacement of the cortex by astrocytic gliosis. There was widespread astrocytic gliosis in the hemispheric white matter, however a pattern consistent with contusional tearing was masked by the extensive cortical lamellar necrosis.

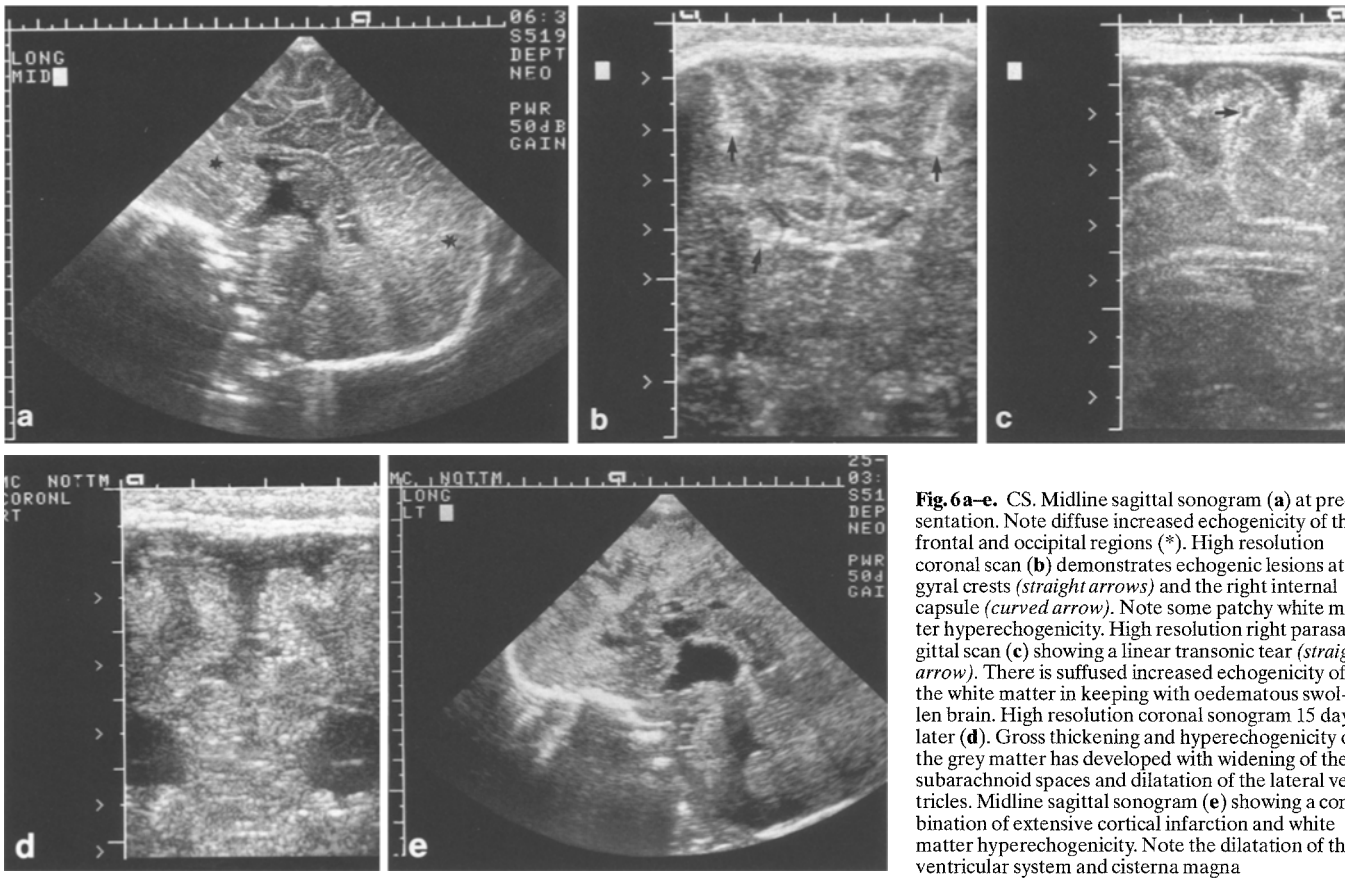

Fig. 6a–e. CS. Midline sagittal sonogram (**a**) at presentation. Note diffuse increased echogenicity of the frontal and occipital regions (*). High resolution coronal scan (**b**) demonstrates echogenic lesions at gyral crests *(straight arrows)* and the right internal capsule *(curved arrow)*. Note some patchy white matter hyperechogenicity. High resolution right parasagittal scan (**c**) showing a linear transonic tear *(straight arrow)*. There is suffused increased echogenicity of the white matter in keeping with oedematous swollen brain. High resolution coronal sonogram 15 days later (**d**). Gross thickening and hyperechogenicity of the grey matter has developed with widening of the subarachnoid spaces and dilatation of the lateral ventricles. Midline sagittal sonogram (**e**) showing a combination of extensive cortical infarction and white matter hyperechogenicity. Note the dilatation of the ventricular system and cisterna magna

Discussion

Post mortem reports of white matter contusional tears have been documented for many years [5, 6]. These tears occur most frequently in the orbital, temporal and first and second frontal convolutional areas [6] and are characteristic of blunt head trauma in infants under the age of five months [5, 6]. Injuries in older children and adults subjected to blunt injuries appear as typical haemorrhagic contusions or diffuse axonal injury. The macroscopic appearance of contusional tears is a sharply outlined slit like or irregular cleft in the white matter which may be so clean as to suggest an artefact produced by removal or sectioning of the brain. Fresh tears may contain small amounts of blood. Older tears appear as smooth walled defects which may coexist with newer lesions if, as is often the case, the child has been subjected to more than one episode of physical abuse [3]. Those which occur in the subcortical white matter may be virtually invisible macroscopically but are evident on histological examination. Lindenberg and Freytag first described these injuries in 16 autopsied infants [5]. The brains were all swollen and the lesions could be difficult to differentiate from artefact. Microscopical examination revealed two types of lesions; white matter and horizontal cortical tears. White matter tears showed early cellular activity at the edges of the lesions with microglial and astrocytic proliferation in the marginal zone, reaching a peak in the third week after injury. The late appearances showed astrocytic gliosis with no evidence of glial or pial elements lining the clefts. The minority of their cases showed cortical tears occuring at the crests of convolutions and orientated parallel to the convolutional surfaces. These lesions were found within the first cortical layer and separated this layer from the rest of the cortex. Except for some residual blood and the presence of increased cellularity and phagocytes associated with the tears they could be easily overlooked.

The pathophysiology of these lesions has been variously ascribed to severe repetitive shaking or blunt impact injuries. It is probable that in most cases there is a combination of these two mechanisms. A variety of factors render the child particularly vulnerable to severe intracranial injury in its first five months of life. Young infants have a softer, pliable calvarium, smoother surface to the cranial fossae floors and widely patent sutures and fontanelles. There is therefore considerable potential for overriding of the skull bones. In addition infants have a higher brain to body size ratio, accounting for 10% of the infant's total weight in comparison to 2% in the adult. The infant's neck muscles are weak and less able to absorb impact. Infants who have been shaken are probably subjected to rapid and repetetive acceleration-decceleration forces in the sagittal plane which are known to be particularly involved in the evolution of subdural haematomas in the experimental sub human primate model [16]. This mechanism most likely accounts for subdural haematomas, the commonest intracranial injury seen in the abused infant at autopsy [5]. Other mechanisms include direct impact of the child's head against a firm object, which may only be a sofa or even a mattress on a bed, or shaking in the lateral plane

which tends to generate torsional forces in the brain [17]. The special conditions pertaining to the sub five month old infant described above are compounded by the immature state of myelination of the brain at this age. Under the age of five months myelination has only reached the level of the corona radiata and has barely commenced in the centra semiovale [18]. Due to the different physical properties of the grey matter and the semi-liquid underlying white matter, gliding of the relatively fixed grey matter over the more gelatinous white matter may occur. Additionally there is, compared to older children and adults, relatively little subarachnoid fluid affording less protection for the surface of the brain. Cerebral contusions at this age occur predominantly at the grey/white matter interface and occur in preference to intracerebral haemorrhage, which is rare in young infants [5]. Gyral crests are particularly vulnerable and there may be a variable degree of extension into the subjacent subcortical white matter [19, 20]. Both whiplash and blunt impact injuries can cause these appearances [2, 3] and in one series of 15 such cases, 5 were thought to be caused by the child accidentally falling on it's head [2].

Although at variance with the earlier studies of Calder [5] and Lindenberg and Freytag [6], Vowles et al. found evidence of axonal swelling and retraction balls indicative of diffuse axonal injury in all 10 babies examined at autopsy under the age of 5 months [7] who exhibited cerebral white matter lacerations. They suggested that contusional tears may therefore be associated with more diffuse axonal injury.

These previous studies were all based upon autopsy findings as imaging modalities during life were too insensitive to detect the relevant lesions. Even with the advent of fourth and fifth generation CT scanners, small (< 3 mm) contusional tears will still, probably, be missed. Larger lacerations can be detected by CT scanning [21], however MR imaging has been shown to be considerably more sensitive in the detection of the characteristic smaller lacerations [14]. In a recent editorial in Radiology, CT has been advocated as the imaging modality of choice in the acute phase of presentation of the head injured infant, with MR imaging being superior in the subacute and chronic phases [15]. Cranial sonography was dismissed due to it's dependence upon patency of the anterior fontanelle, poor depiction of the extraaxial spaces and peripheral cortex and of the posterior fossa. High resolution sonography has, however, been shown to demonstrate a large haemorrhagic laceration in a six week old infant [22]. The authors comment that, in their experience, sonography is more sensitive for the detection of smaller cystic spaces than CT. It is the current authors' experience that high resolution near field imaging of the infant brain, particularly under the age of six months, is considerably more sensitive than CT scanning in the assessment of the extraaxial spaces and subjacent brain. At our institution, employing a second generation low field strength resistive magnet, sonography has also proved to be superior to MR imaging however at higher field strengths with current "state of the art" systems MRI would undoubtedly be more rewarding. No significant lesion was missed by sonography in the supratentorial compartment, however MR imaging did reveal a thin posterior fossa subdural collection not detected by sonography.

We have found that sonography is able to clearly demonstrate both grey and white matter and is able to detect lesions of less than 1 mm in size. The six cases presented in this paper demonstrate the highly characteristic white matter tears associated with blunt impact and shaking injuries associated with child abuse in the first six months of life. Sonography revealed such lesions more clearly than CT and MR scanning in all our cases. Subdural and subarachnoid collections in the supratentorial compartment could be clearly demonstrated however their precise characterization and the ageing of subdural haematomas could not be accurately assessed. The ageing and precise anatomical delineation of intra- and extra-cerebral haematomas is best assessed by MR imaging. A major advantage of sonography is that it permits a cot side examination of the more severely injured cases thus avoiding the need for a disruptive transfer to the CT or MR suite and enabling minimal handling of the child. Frequent follow-up scans may be made by the cot side without the burden of repeated radiation exposure.

In two of the cases in our series post mortem examinations were available for comparitive analysis. The striking finding was a lack of direct macroscopical correlation of all the contusional tears demonstrated by sonography. In case 5 the child died within 24 h of the sonographical examination. The autopsy was performed, following routine fixation, three weeks later. Several of the larger lesions demonstrated in life were found along with severe brain swelling and multiple foci of deeper haemorrhagic contusions however the smaller lesions in the subcortical white matter could not be identified macroscopically. In case 6 the autopsy was obtained six months after the initial episode of child abuse. Sonography had demonstrated the evolution of diffuse cortical swelling and hyperechogenicity suggestive of infarction. The latter was confirmed at autopsy however the slit like lesions could not be clearly identified, the predominant feature being the extensive severe cystic gliosis affecting the whole of the cerebral cortices in a lamellar necrosis pattern. The extent and severity of these changes probably masked the presence of the delicate slit like lesions. In case 4 the slit like lesion demonstrated at presentation was monitored by repeat sonographical examinations. These showed the lesion undergoing a slow evolution, the central transonic component disappearing leaving a slit like residue. This lesion measured 6 mm in length and less than 1 mm in width. Such a small lesion could be easily missed if only a delayed examination had been performed. These lesions could, therefore, also be missed at post mortem unless extensive histology was performed to specifically search for them.

Conclusion

Cerebral contusional tears suffered by severely abused infants can now be detected whilst they are still alive. Their presence should alert clinicians to the very strong possibility of child abuse in a young infant. High resolution cranial sonography can provide exquisite detail of the critical

grey-white matter interface and is recommended as the primary imaging modality in the investigation of suspected child abuse in the first six months of life. Further research is required to compare high resolution sonography with MR imaging. At present we regard the two techniques as complimentary to each other and advocate their combined use when the initial sonographical examination has demonstrated an abnormality.

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