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CASE REPORT

Neonatal subcortical bruising

Gwenda Delanghe[1], Waney Squier[2], Michel Sonnaert[3], Jeroen Dudink[4], Maarten Lequin[5] & Paul Govaert[1]

[1]Department of Neonatology, Middelheim Hospital Antwerp, Antwerp, Belgium
[2]Formerly Consultant Neuropathologist at Oxford John Radcliffe University Hospital, Oxford, UK
[3]Department of Neonatology, University Hospital Brussels, Brussels, Belgium
[4]Department of Neonatology, Wilhelmina Children's Hospital Utrecht, Utrecht, The Netherlands
[5]Department of Radiology, University Medical Center Utrecht, Utrecht, The Netherlands

Correspondence
Gwenda Delanghe, Lindendreef 1, 2020 Antwerp, Belgium. Tel: +32 3 2803344
E-mail: Gwenda.Delanghe@zna.be

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Key Clinical Message

A specific type of acute brain injury can occur during birth, presenting on ultrasound examination with focal, unilateral, or asymmetrical change in the core of the superior frontal gyri. Ultrasound inspection of the superior gyri near the convexity of the frontal lobe is warranted following mechanically difficult delivery.

Keywords

Brain, neonate, pathology, subcortical white matter.

Introduction

The aim of this paper is to advance the definition of a specific variant of neonatal focal superficial brain damage associated with deliveries in which abnormal parturitional forces are suspected. We present ultrasound images of neonates with lesions in the white matter of superior frontal gyri without massive hemorrhage, in all cases with a patent superior sagittal sinus and all detected with ultrasound in the early neonatal period. Because of the focal aspect of damage to the subcortical white matter underlying an intact cortex, we propose the term "subcortical bruising." This type of white matter damage is one variant of subcortical leukomalacia, most often described in the context of global forebrain ischemia [1]. Recognition of this pattern may have medicolegal implications, especially in the absence of major intracranial bleeding, when the traumatic nature of injury is questioned. Au-Yong (2009) recently described five young infants with focal superficial brain damage, four following mechanically difficult deliveries [2]. Except for one neonatally confirmed diagnosis, the others were recognized in early infancy, mainly by CT and MRI. Some of these infants may have had the same lesion pattern as in our cohort.

Case Histories (Figs. 1–4)

The clinical cases were collected in three different units within a 7-year span. Parents at respective neonatal units agree (by waiver) to anonymous use of clinical and imaging data for scientific and educational purpose.

All lesions were recognized with early neonatal ultrasound scanning between the first and fifth day of life, in two infants only with high-frequency scanning (>10 MHz) in addition to routine scanning with a convex 8–10 MHz probe. These probes were connected to different ultrasound machines (Esaote MY Lab Twice, Philips HD11XE, and GE Vivid S5).

Five infants were admitted immediately after birth or on day one for poor condition or clinical seizures. Four were ventilated (Table 1). Focal epileptic EEG discharges were recorded in two on standard EEG recordings. All had parietal cephalhaematoma. Detailed ultrasound scanning was performed according to local protocol for

clinical seizures or poor neurological condition. In two infants, cranial fracture was ruled out with CT scan (Table 2). In all the brunt of injury was to the left hemisphere, always affecting at least the frontal lobe. Evolution into cystic destruction was seen in one infant within a time span of 10 days. In the first two cases, MRI characteristics were compatible with intracellular deoxyhemoglobin in the center of the lesion (isointense on T1-weighted and hypointense on T2-weighted images). In the periphery, the signal was compatible with intracellular methemoglobin (hyperintense on T1-weighted and hypointense on T2-weighted images). The lesions were positioned in the subcortical white matter, surrounded by unaffected white matter. In the first case, the FLAIR sequence demonstrated high signal in nearby subarachnoid spaces, compatible with limited subarachnoid or subpial hemorrhage. None of the survivors developed serious neurodevelopmental problems, but survival time is <2 years for four of them.

Case 5 is a term infant, surviving for only 5 days after secondary cesarean section for failure to progress. The baby's head was impacted necessitating forceps lift-out. The baby had early seizures and HIE grade 3 and was cooled for 72 h. Postmortem findings are shown in Figure 4 (as referred to in [1]).

Discussion

This retrospective cohort further defines an insufficiently reported pattern of neonatal brain injury, observed mainly in term infants. The hallmark is focal, unilateral, or asymmetrical hyperechoic change in the core of the superior frontal gyri. These lesions do not fit a global hypoxic–ischemic or macrovascular template of injury. In contrast to contusional lesions, the overlying cortex is intact and there is no major subarachnoid bleeding. Because of the elongated, slit-like appearance of the lesions along the frontal gyrus, the term clefting can be used in some infants.

Various forms of neonatal brain damage have been described following mechanically difficult delivery. Epidural, subdural, parenchymal, and intraventricular

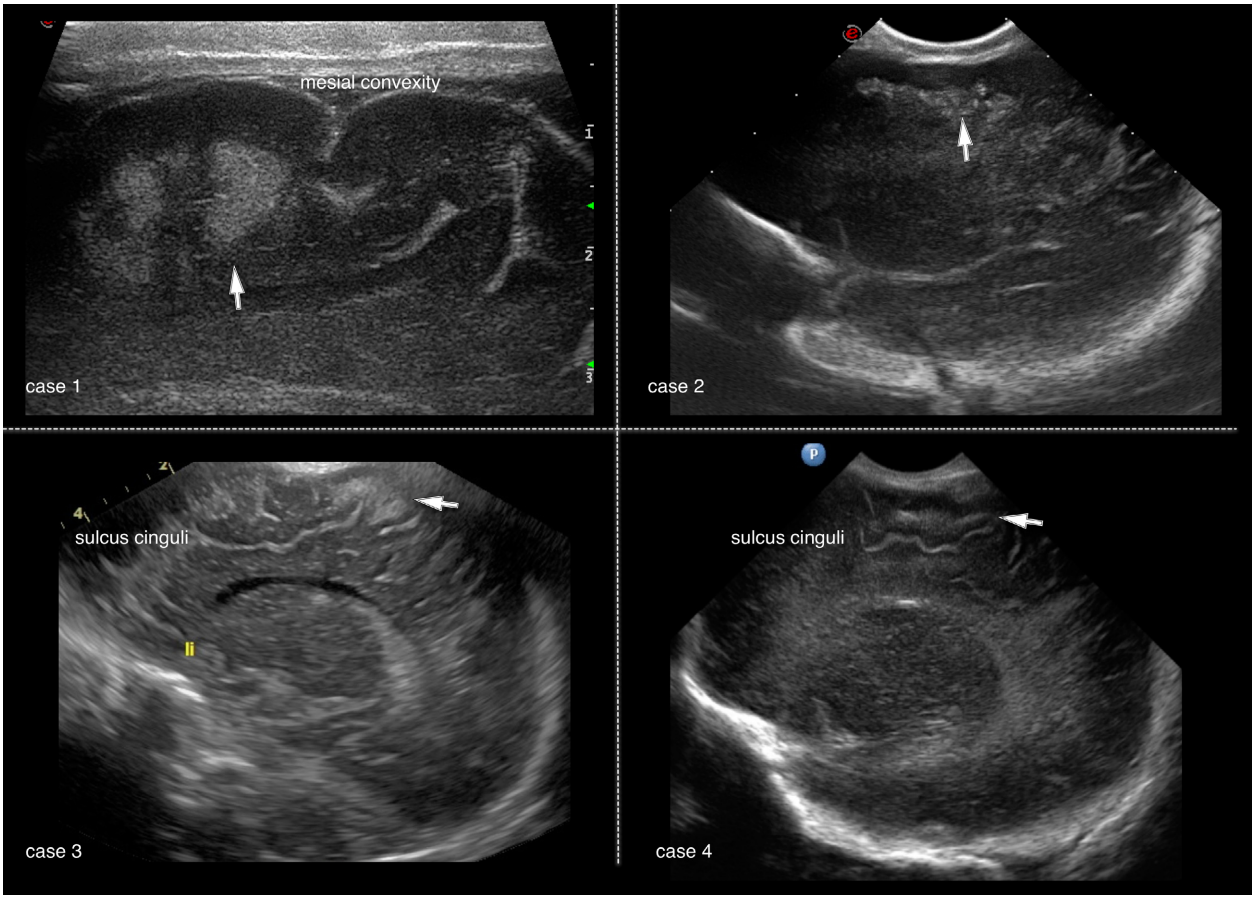

Figure 1. Panel of sagittal sonograms with arrows pointing to the frontal gyral core lesion in cases 1–4.

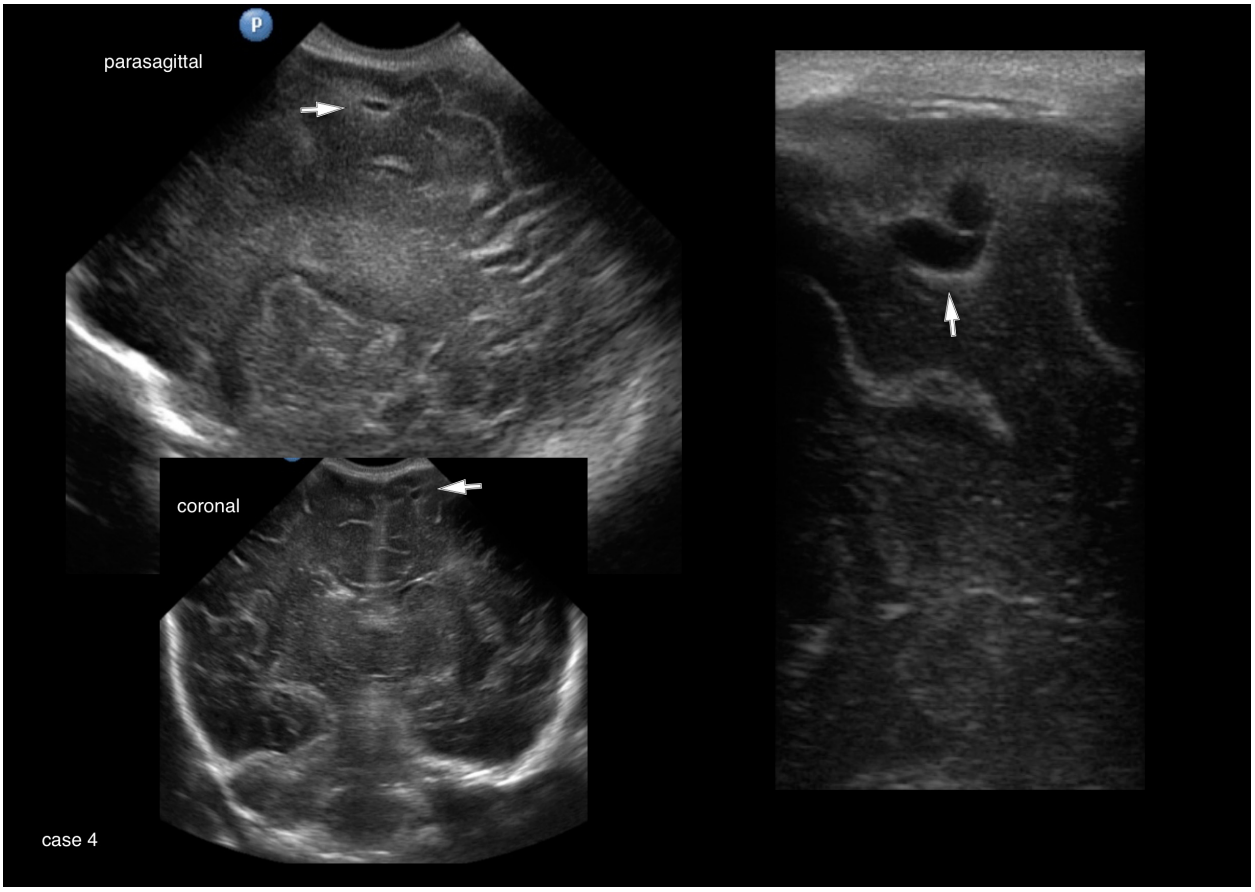

Figure 2. Evolution on day 9 to cavitation in case 4.

hemorrhage have all been linked to perinatal trauma, following mechanical disruption of bone or dural membrane [3, 4]. Subdural bleeding may follow disruption of the intradural venous plexus, often not associated with characteristic signs of trauma [5]. Displacement of bones may lead to excessive molding and subsequent tearing of dural membranes and/or sinovenous thrombosis as intermediary mechanisms. On occasion, extra-axial hemorrhage is associated with secondary arterial infarction [6]. Sinovenous thrombosis, starting from an impressed occipital squame tip, may lead to parenchymal bleeding in parasagittal tissue drained by the superior sagittal sinus itself, while propagation into deep veins leads to bleeding also in the thalamus (thalamo-ventricular hemorrhage) [7]. In infants, venous infarction may resemble contusion as it often has a hemorrhagic component [8]. Fetal trauma through the maternal abdominal wall may also lead to hemorrhagic lesion patterns [9]. Multifocal injury that is not hemorrhagic, but ischemic in nature, may follow cranial compression during delivery, when excessive in force or time. Reversible arterial compression by pressure is a hypothetical explanation for such rare instances, but this condition is not well described by neonatal imaging [10].

Focal superficial brain damage ("contusion") involving cortex and subcortical white matter near a displaced or fractured bone has been only incidentally reported following birth trauma. An often complex mixture of ischemia and bleeding may operate together in this context. Two types of superficial damage to the neonatal brain, related to mechanical trauma, have already been described in detail. The first, cerebellar contusion, is due to occipital squama impingement in the posterior fossa during delivery or intubation (occipital osteodiastasis), a condition sometimes recognized incidentally by finding cerebellar embolism in the lung at postmortem exam [11]. The second injury type, hypothetically associated with vigorous chest physiotherapy, has been described in a context of symmetrical parieto-temporal hemorrhagic brain destruction, of purely postnatal origin [12]. The lesions reported here are sufficiently different and merit detailed description.

Our cases were probably associated with excessive parturitional forces but similar clefts can be seen with asphyxia. Isolated gyral crest white matter injury is also

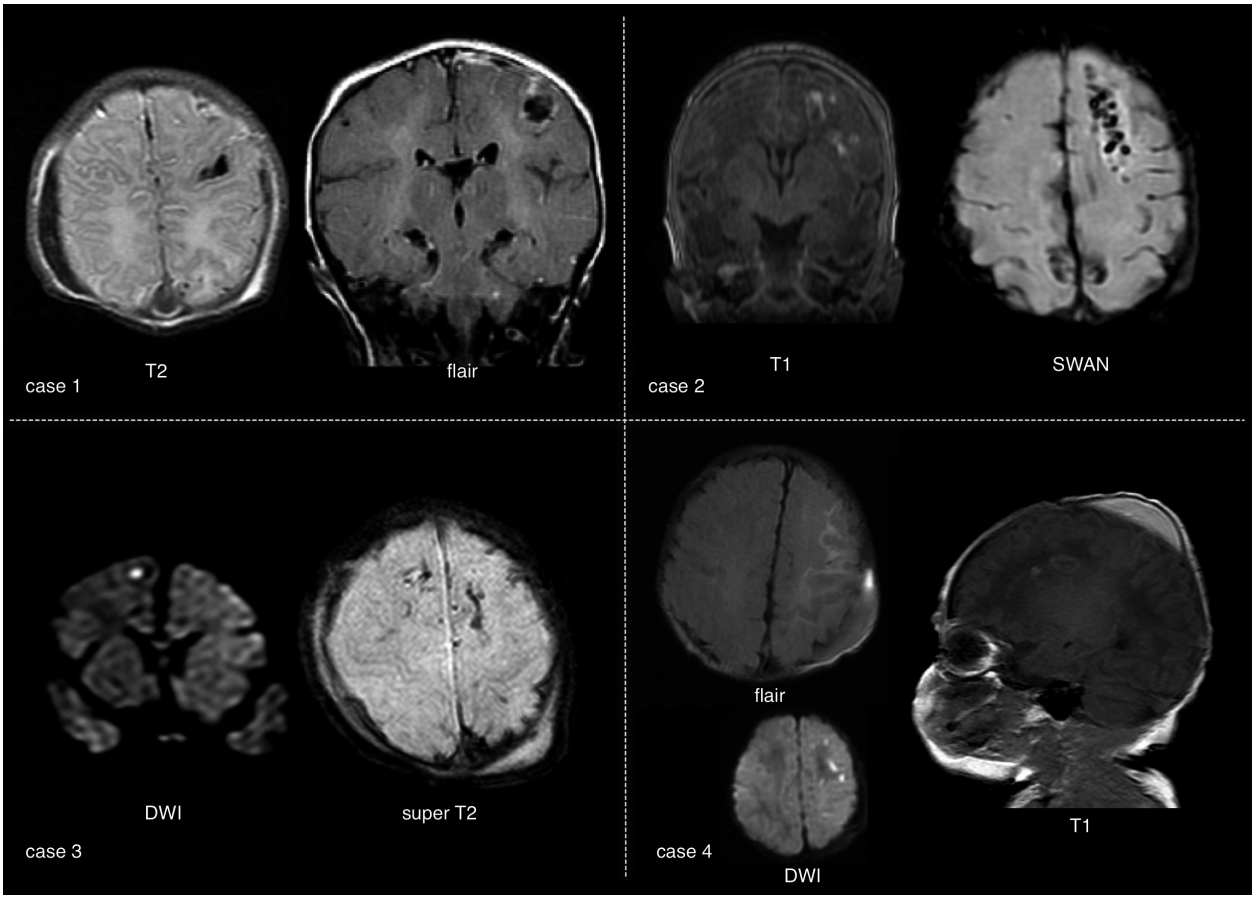

Figure 3. Panel of a mixture of MR findings in cases 1–4.

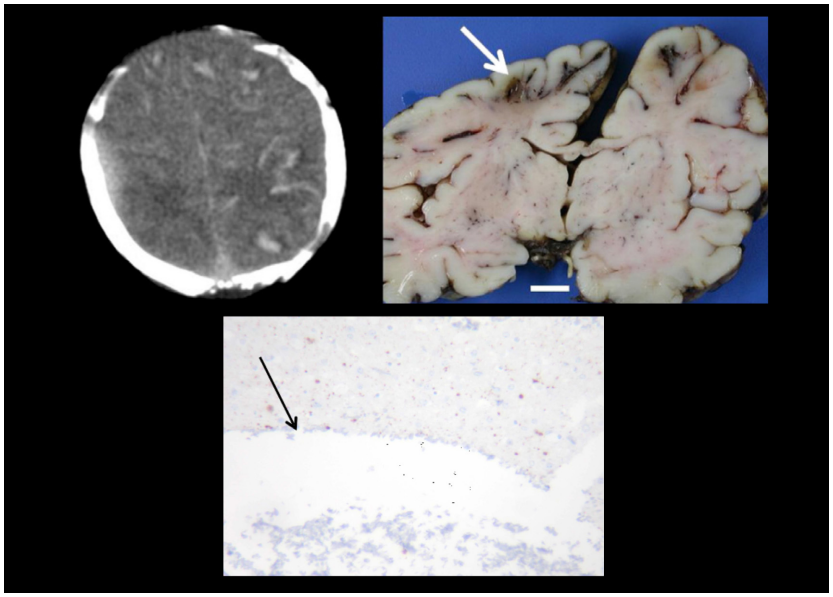

Figure 4. CT scan day 3: edema, multiple small subcortical white matter hemorrhagic changes underlying parietal bone fractures, originally reported as traumatic shearing. The fixed brain slice shows that these are small clefts and cysts containing blood (arrow) (scale bar = 1 cm). A subcortical cleft bordered by white matter (arrow). The section is stained with βAPP and shows small scattered axonal swellings consistent with hypoxic–ischemic injury. There are no axonal swellings bordering the cleft as would be expected in traumatic axonal disruption.

Table 1. Summary of clinical data in cases 1 to 4.

Name	1	2	3	4
Gender	M	F	F	M
Obstetric data				
Gestational age w/d	42 1/7	33 0/7	40	40 2/7
Induction	Not	Not	Not	Yes
PROM	Not	Not	Not	Not
Maternal fever	Not	Not	Yes	Not
Meconium staining	Yes	Not	Not	Yes
CTG	Tachycardia	Fetal distress?	Decelerations	Decelerations
Presentation	Occiput	Occiput	Occiput anterior	Occiput anterior
Second stage	Arrest of descent			
Instrumental traction	Not	Not	Failed vacuum	Not
Cesarean section	Secondary to dystocia	Secondary to fetal distress	Secondary to dystocia	Secondary to dystocia
Delivery of the head	Difficult	Difficult	Tight nuchal cord	Difficult retrieval of the head
Birth				
Cord pHart	6.86	—	—	—
Apgar 1′	2	3	1	4
Apgar 5′	4	7	3	9
Apgar 10′	8	—	5	10
Resuscitation	Bag and mask	Bag and mask	Bag and mask, adrenalin, thoracic compression	Bag and mask
Intubation	Not	Not	Yes	Not
Seizures	Day 1	One episode of focal left arm twitching on day 1	Smacking on day 1	Focal right arm and leg convulsions day 2
Anticonvulsants	Phenobarbitone	None	Phenobarbitone	None
Ventilation	7 days	7 days	5 days	Not
EEG	Left epileptic activity		Occipital epileptic activity	No epileptic activity
aEEG		No epileptic activity		
Clinical evolution				
Birthweight grams	3700	1690	3375	3850
Head circumference cm	36.7	27.6	34	35.5
Bruising				Left upper arm
Cephalhematoma	Right parietal		Left posterior parietal	Left parietal
Skull fracture	Not	Not	Not	Not
Fontanel	Normal	Normal	Normal	Normal
Encephalopathy	Quiet but reactive on day 1		Thompson score 7 at 1 h, sarnat stage 2	Painful and hyper-reactive on day 1
Cooling	Not	Not	Yes	Not

one of the possible corollaries of nonaccidental blunt head trauma or shaking in young infants [13–22]. This slit-like subcortical white matter injury in young infants has been named contusional tearing, inferring a mechanism whereby the different physical properties of cortical and subjacent white matter predispose to shearing stress, leading to white matter tears, according to some of these authors in a context of shaking injury. Such clefts are delineated by limited hemorrhage and astroglial reaction and are characterized by the absence of axonal swellings, reflecting the uncertainty of a traumatic mechanism. Their location is mainly orbitofrontal and temporal. In the infantile variant of this type of injury, shearing forces have been suggested to occur in acute acceleration or deceleration following shaking or blunt head trauma, some with the aspect of "coup and contrecoup" injury. Such forces are unlikely to occur during birth; hence, this pattern of neonatal brain pathology raises the question of the underlying intermediary mechanism. Unequally distributed pressure could explain the lesions. It has been suggested that the smoothness of the inner skull table and the pliability of neonatal skull bones protect against typical contusion as observed in adults [15]. During vaginal delivery, skull compression leads to increased intracranial pressure, up to four times higher than intra-amniotic pressure at specific sites like the equator or the area of pelvic contact [23–25]. When molding is not excessive, this pressure distribution remains almost equally spread throughout the cranium. Excessive focal bone displacement could provoke an unequal pressure distribution [26]. The combination of general high intracranial pressure and local excessive pressure on the brain surface is

Table 2. Summary of imaging findings in cases 1 to 4.

Ultrasound	Hyperechoic aspect of posterior left frontal cortex on day 1; minimal residual hyperechoic change at the end of the first week; patent superior sagittal sinus at the anterior fontanel	Irregular hyperechoic change in the entire left frontal lobe area near the convexity on day 2; patent superior sagittal sinus at the anterior fontanel	Limited hyperechoic focus in left superior frontal gyrus white matter core on day 4; no cavitation at the end of the first week; patent superior sagittal sinus at the anterior and posterior fontanel	Hyperechoic change in mesial frontal gyral cores on day 2, more pronounced on the left; diffuse hyperechoic change in left frontal lobe white matter with some left hemisphere swelling displacing midline and tentorium; slit ventricles; on day 9: cavitation in left mesial frontal hyperechoic lesion, aligning along the gyral core; patent superior sagittal sinus at anterior and posterior fontanel
CT			No obvious intracranial bleeding or ischemia on day 11; no fracture on bone window setting	Minimal bleeding in left frontal lobe on day 1, no areas of arterial attenuation, no fracture on bone window setting
MRI	Right parietal cephalohematoma; T1 hyper- and T2 hyposignal compatible with bleeding in and along the left (post)central area on day 4; discrete similar hemorrhagic punctate and linear lesions in the left parieto-occipital area overlaid by subarachnoid and limited subdural bleeding	Subarachnoid bleeding overlying both cerebellar hemispheres; several regions with lower ADC values in the left hemisphere corresponding partially with the hemorrhagic areas on day 5, additional small right prefrontal subcortical focus	Bilateral (largest on the right) parietal cephalohematoma on day 5; extensive hemorrhagic change in the left parasagittal area of the premotor and prefrontal (sub)cortex (T1 hyper- and T2 hyposignal); in addition bilateral similar changes in the mesial parietal area; all areas had low ADC values and an additional low ADC area was present along the posterior part of the right superior frontal gyrus	Limited focal high signal on DWI in right superior frontal gyrus on day 2; low signal in same area on super T2W-GRE- EPI and in an additional area on the left compatible with the hyperechoic focus left parietal cephalohematoma; extensive left frontal hypersignal on PD in subcortical white matter of the premotor area; focal even higher signal in five small subcortical areas on the left and one on the right (diffusion positive but not dark on T2); focal lesion in left thalamus (best seen on DWI)

probably the cause of the extensive injury pattern seen in some of the infants reported here. This appears to result in selective white matter damage underneath the mesial margin of the frontal or parietal bone. Circulatory stasis (venous congestion) and swelling may concur to selectively damage the most vulnerable brain part, that is, the immediate subcortical white matter [1]. Alternatively, a sudden increase in amniotic pressure, a sudden forceful grip on the skull by the obstetrician's hand or a forcep blade, or acute pressure relief during delivery of the head may generate forces focused on selective superficial gyri. The buildup of pressure during delivery occurs in a matter of many seconds to minutes, different from instantaneous events during nonaccidental injury. Therefore, the similarity of white matter damage in two apparently different mechanistic contexts, that is, compromised birth and nonaccidental injury, remains intriguing. The infants we describe all had signs suggestive of traumatic delivery such as long second stage, cesarean secondary to dystocia or failed instrumental traction, cephalohematomas, poor Apgar scores, and encephalopathy. None had impressive extra-axial or parenchymal bleeding, occipital osteodiastasis, or superior sagittal sinus thrombosis [3, 27]. Excessive pressure may, as in some cases presented here, lead to relatively subtle and specific injury patterns that are dissimilar to frank trauma. A vascular mechanism is a logical link between increased pressure on the brain surface and focal injury to the white matter core in a frontal gyrus. Compression of arteries from the surface would lead to ischemia in both cortex and subcortex, also further down into deep white matter at some distance from the ventricle [10]. On the other hand, compression of veins from the surface might lead to selective subcortical vulnerability in the venous watershed area (the zone between superficial cortical drainage and deep ventricular drainage): Veins present in the cortical drainage system might not be totally

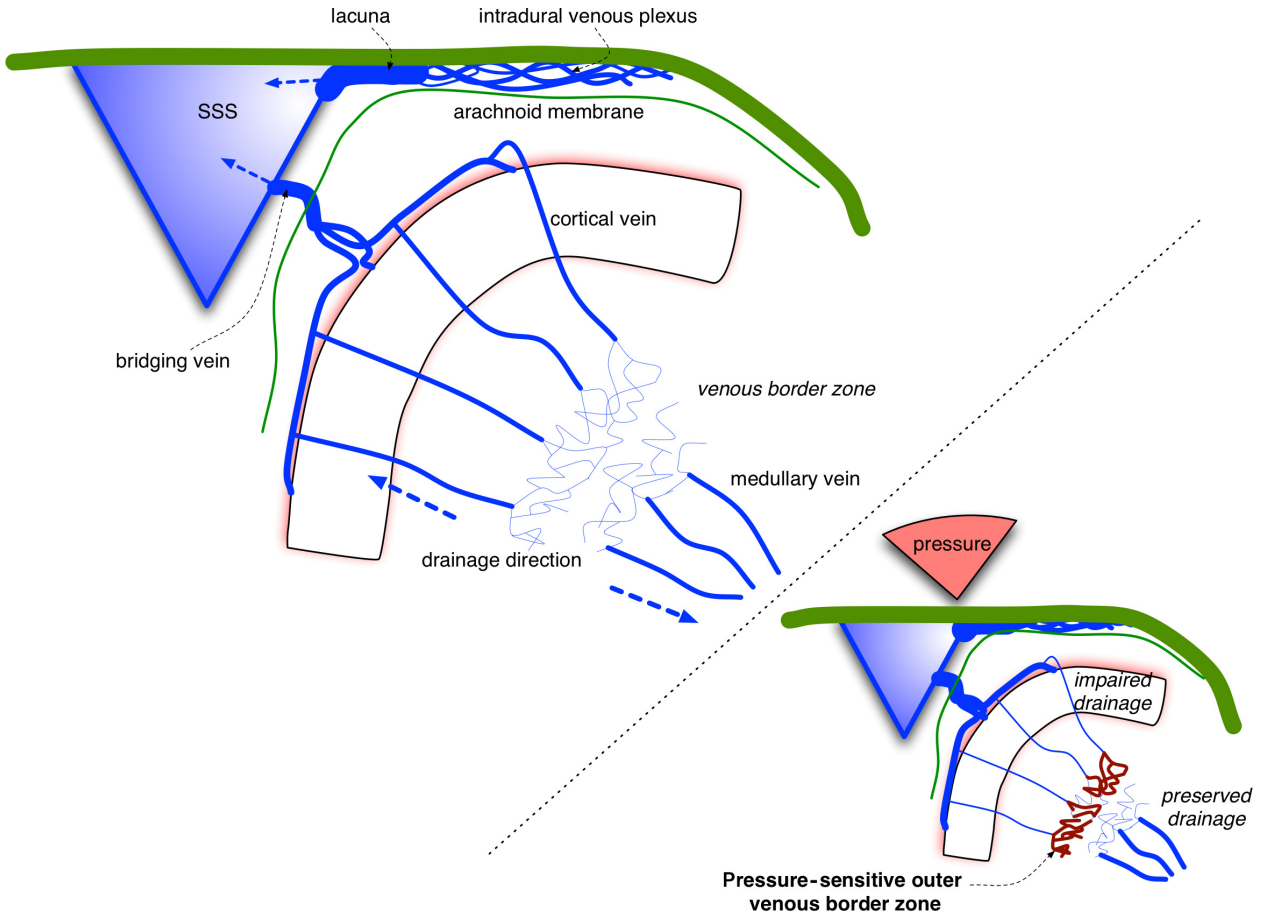

Figure 5. Scheme depicting the possibility of a venous mechanism set in order by mechanical compression near the SSS.

occluded and some efflux could persist, while stasis of extracellular fluid in the venous watershed area facing the cortex could be important (Fig. 5). Higher pressures are probably also needed to compress arteries compared to veins. Hypothetically therefore, this focal white matter destruction might have a venous signature. Swelling of tissue and venous congestion have also been suggested to play a role in subcortical cystic leukomalacia associated with intrapartum asphyxia [1]. The pressure exerted on the fetal brain and body by forces of labor leads to complex reactions, difficult to understand in clinical practice [28].

Because superior frontal gyri are readily accessible to (high-resolution) ultrasound, this is the optimal method of screening for this lesion promptly after admission following delivery complicated by dystocia or excessive uterine contractions. It is very likely that some of the injuries described by Au-Yong et al. [2] in 2009 follow a pattern as described in this report, but their case 1 is suggestive of focal arterial infarction in a branch of the ACA and their case 2 resembles watershed injury. We agree with these authors that both such diagnoses should be kept in the differential diagnosis. The major difference in our observations is that in our series the cortex appeared to remain intact. This may be a reflection of the period of survival; our cases were all examined on days 1–7 after birth. In Au-Yong's series, cortical destruction was not observed before 23 days of life and may be the result of atrophy following loss of subcortical white matter. Their case 4 best resembles our cases.

On MRI the lesions are positioned in subcortical white matter surrounded by unaffected white matter and without extensive subarachnoid bleeding. High signal in the subarachnoid space on the FLAIR sequence could in part be limited subarachnoid bleeding, especially in the high signal areas on the corresponding T1-weighted images. But areas affected by high signal on the FLAIR sequence are more extensive than changes on the other image sequences (T1-, T2-, and diffusion-weighted imaging), suggesting only part of it is subarachnoid blood. Goericke et al. [29] suggest that part of the subarachnoid high

FLAIR signal is due to a temporary increase in blood volume in superficial brain vessels. Therefore, in the absence of histopathological correlation, some findings merely suggest a venous contribution. In the one case where we have histopathology, there was subpial bleeding associated with the subcortical damage. Lack of severe low signal from surrounding tissue on the ADC map, including overlying cortex, does not fulfill the criteria of venous infarction. This corresponds to the pathology, and subpial bleeding in this context was described in Squier et al. [1]. In all cases, the superficial and deeper cortical veins and arterial vessels near to the lesions seem patent, but this interpretation is fraught with uncertainty as flow techniques for small vessels are imprecise in the newborn. Also, distant larger superficial and deep venous collectors are not occluded.

The importance of further research into the imaging behavior of subcortical linear lesions is not only relevant to neonatal care but also relevant to trauma care in infancy and early childhood, where the distinction between shearing injury and laceration remains problematic yet clinically and medicolegally relevant [30].

Conclusion

In conclusion, we define a variant of neonatal focal subcortical white matter damage associated with deliveries in which abnormal parturitional forces are suspected. Although the precise mechanism is unknown, we suggest the term subcortical bruising. Different from contusion, although not really well defined in the current literature, is the normal aspect of cortex overlying typical subcortical lesions. Because the ultrasound and MR findings are yet incompletely understood, we propose that in essence, mesial frontal gyral core intensity change (hyperechoic on neonatal ultrasound, partly hyperintense on T1-weighted MRI) and in some evolving to cavitation ("clefting"), be used as the hallmark of this lesion pattern. Additional damage includes asymmetrical ischemic and hemorrhagic lesions, mainly to the frontal lobe. Further research should focus on the details of forces during delivery that precede this pathology (head presentation and pressure load), on outcome and prevention. Based on the findings in this cohort, we propose that detailed high-frequency scanning of the frontal lobe and superior sagittal sinus should be part of an admission sonogram following mechanically difficult delivery.

Authorship

All coauthors contributed equally to the input of clinical and imaging data and were involved in revising the manuscript. GD, WS, and PG: writing of the manuscript.

Conflict of Interest

There are no competing interests.

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