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ORIGINAL ARTICLE

Intracranial haemorrhage: an incidental finding at magnetic resonance imaging in a cohort of late preterm and term infants

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Abstract
Background Intracranial haemorrhage (ICH) in term newborns has been increasingly recognised but the occurrence in late preterm infants and the clinical presentation are still unclear.
Objective To investigate the appearance of intracranial haemorrhage at MRI in a cohort of infants born at 34 weeks' gestation or more and to correlate MRI findings with neonatal symptoms.
Materials and methods We retrospectively reviewed neonatal brain MRI scans performed during a 3-year period. We included neonates ≥34 weeks' gestation with intracranial haemorrhage and compared findings with those in babies without intracranial haemorrhage. Babies were classified into three groups according to haemorrhage location: (1) infratentorial, (2) infra- and supratentorial, (3) infra- and supratentorial + parenchymal involvement.
Results Intracranial haemorrhage was observed in 36/240 babies (15%). All of these 36 had subdural haemorrhage. Sixteen babies were included in group 1; 16 in group 2; 4 in group 3. All infants in groups 1 and 2 were asymptomatic except one who was affected by intraventricular haemorrhage grade 3. Among the infants in group 3, who had intracranial haemorrhage with parenchymal involvement, three of the four (75%) presented with acute neurological symptoms. Uncomplicated spontaneous vaginal delivery was reported in 20/36 neonates (56%), vacuum extraction in 4 (11%) and caesarean section in 12 (33%). Babies with intracranial haemorrhage had significantly higher gestational age (38± 2 weeks vs. 37±2 weeks) and birth weight (3,097±485 g vs. 2,803 ± 741 g) compared to babies without intracranial haemorrhage and were more likely to be delivered vaginally than by caesarian section.
Conclusion Mild intracranial haemorrhage (groups 1 and 2) is relatively common in late preterm and term infants, although it mostly represents an incidental finding in clinically asymptomatic babies; early neurological symptoms appear to be related to parenchymal involvement.

Keywords Full-term newborn · Late preterm infant · MRI · Intracranial haemorrhage · Subdural haemorrhage · Intraparenchymal haemorrhage

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Introduction

Intracranial haemorrhage in term newborns is considered to be relatively uncommon although the real incidence is unknown, mainly because most of the affected infants are asymptomatic. In the last 20 years intracranial haemorrhage in term newborns has been increasingly recognised because of advances in neuroimaging techniques, and recent studies report an incidence ranging from 19% [1] to 46% [2] (mostly subdural haemorrhages limited in size and location), depending on the sensitivity of the neuroimaging technique and the timing of the assessment in asymptomatic full-term infants after delivery. However no data have been published on late preterm infants, defined as babies born between 34+0 weeks'

and 36 + 6 weeks' gestation. Recently the interest of neonatologists has been focused on late preterm infants, who represent about 72% of preterm births in developed countries, 7–8% of total live births, and account for the striking increase of the number of babies categorized as premature in the last two decades [3, 4]. The higher vulnerability of late preterm infants to developing diseases in the early neonatal period, compared to term babies, has recently become recognised. Brain vulnerability has also been documented according to the concept of gestationally determined regional vulnerability of the developing brain, mainly related to the dramatic brain growth and developmental changes still occurring at these gestational ages: at 34 gestational weeks the brain weighs only 65% of the term brain, with a 35% increase in growth needed to reach term weight, and the cortical volume is only 53% of the term volume [5]. Therefore late preterm infants have a risk of developing brain lesions that is lower than more premature babies but higher than term newborns.

Intracranial haemorrhage can be classified according to either the site (supratentorial vs. infratentorial) or the compartment involved (epidural, subdural, subarachnoid, intraventricular or intraparenchymal haemorrhage) [6, 7].

Intraventricular haemorrhage is known to be more common among very preterm babies, with an inverse relationship with gestational age [8]. It originates in the germinal matrix, which is highly represented at the lower gestational age, supporting the theory that the larger the germinal matrix, the more susceptible it is to bleed. In the term newborn intraventricular haemorrhage is rarely seen, and although it is supposed to originate from the choroid plexus it can represent a complication of sino-venous thrombosis [9]. The most frequent intracranial haemorrhage in full-term infants is subdural haemorrhage; it is the most common form of birth-related intracranial haemorrhage [2, 10] but in most cases it is undetectable after 4 weeks. Subdural haemorrhage is typically infratentorial, over the cerebellum, in the posterior fossa or located just above the tentorium or around the occipital lobes [11]. Tearing of the tentorium and the falx or rupture of bridging veins from stretching associated with deformation of the head during labor and delivery have been advocated as possible pathogenetic mechanisms [2, 7, 10].

Some reports suggest that the risk of intracranial haemorrhage is associated with maternal and perinatal factors such as maternal parity, process of labour, instrumental vaginal delivery (use of forceps or vacuum extraction), foetal macrosomia and perinatal asphyxia [2, 6, 10, 12, 13]. Clinical manifestations with neurological symptoms in the first days of life and adverse neurological sequelae have been described in intracranial haemorrhage complicated by parenchymal involvement [10]. Conversely most of the uncomplicated intracranial haemorrhages seem to remain clinically silent in the neonatal period, although it is not known whether they affect brain development.

The aim of this retrospective study was to investigate the appearance of intracranial haemorrhage at MRI in a cohort of infants born at 34 weeks' gestation or more and to correlate intracranial haemorrhage appearance to clinical symptoms in the neonatal period.

Materials and methods

No permission was required by the institutional ethics committee for this retrospective study. We obtained written informed consent from each child's parent to conduct brain MRI.

We retrospectively studied all late preterm (≥34 weeks' gestation) and term infants admitted to the neonatal tertiary intensive care unit (NICU) Fondazione IRCCS Ca'Granda Ospedale Maggiore Policlinico, Milan who underwent postnatal brain MRI for clinical purposes between 1 January 2009 and 31 December 2011. Brain MRI was performed according to the clinical routine protocol that included newborns with abnormal neurological signs (abnormal tone, encephalopathy), abnormal foetal or neonatal brain US findings, metabolic disorders, congenital infection, dysmorphic features, and perinatal asphyxia with hypoxic–ischaemic encephalopathy. Infants with accidental postnatal head trauma were excluded. Late preterm infants were brain-scanned at term-corrected age. Total number of infants included was 240.

MRI was performed on a 1.5-T Achieva 2.6 system (Philips Healthcare, Best, The Netherlands) using a paediatric-dedicated coil (Sense Ped, Philips Healthcare, Best, The Netherlands). Brain MR images were acquired using T1-W spin echo and T2-weighted turbo spin echo and fast field echo sequences and diffusion-weighted imaging (Table 1). Infants were scanned following a feed and were monitored by pulse oximetry and electro-cardiography (Invivo Precess monitoring; Invivo, Orlando, FL) during the MRI scan. Neonatal noise attenuators (MiniMuffs, Natus Medical Inc., San Carlos, CA) were used.

All MR scans were reviewed by two experienced neuroradiologists (SA, CC) who were blinded to neonatal

Table 1 Brain MRI protocol

Sequences	Slice orientation/thickness	FOV	TR/TE (ms)	Matrix
T1-W SE	Sagittal/3 mm	180	623/15	256×204
T1-W SE	Axial/3 mm	190	786/13	184×106
T2-W TSE	Axial/3 mm	190	6,000/200	272×133
T2-W TSE	Coronal/3 mm	170	6,000/200	256×152
T2-W FFE	Axial/3.5 mm	180	1,065/32	180×108
DWI	Axial/3 mm	160	4,316/72	108×104

DWI diffusion weighted imaging b value: 1000, *FFE* fast field echo, *FOV* field of view, *SE* spin echo, *TE* echo time, *TR* repetition time, *TSE* turbo spin echo

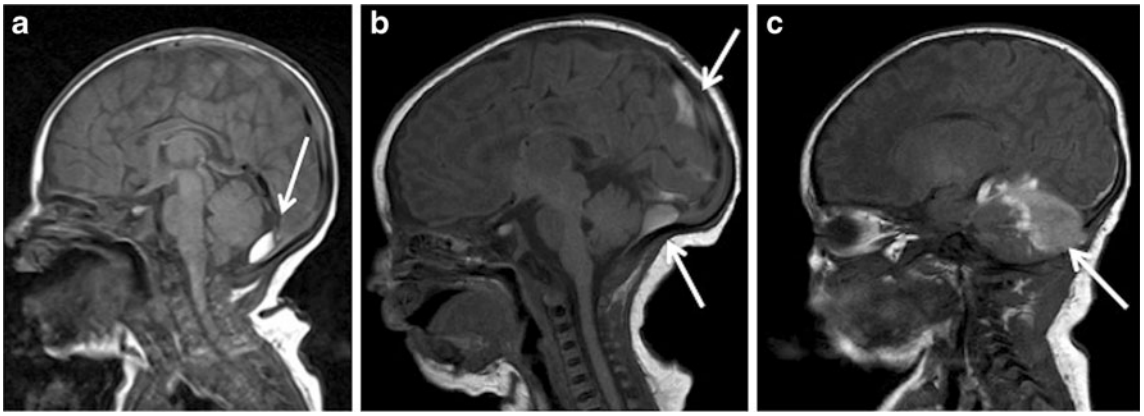

Fig. 1 Midsagittal T1-weighted spin-echo MR images in three different infants. **a** Infratentorial intracranial haemorrhage (*arrow*) in a newborn in group 1 (gestational age at birth 39 weeks, gestaional age at imaging 40 weeks + 4 days) (**b**) Infra- and supratentorial intracranial haemorrhage (*arrow*) in a newborn in group 2 (gestational age at birth 35 weeks + 4 days, gestaional age at imaging 39 + 6 days). **c** Intracranial haemorrhage with parenchymal involvement (*arrow*) in a newborn in group 3 (gestational age at birth 40 weeks, gestaional age at imaging 40 weeks + 1 day)

and obstetric data; cases of disagreement were resolved by consensus. All images were evaluated to identify intracranial haemorrhage, focusing on T1-W and T2-W fast field echo sequences.

As illustrated in Fig. 1, babies with intracranial haemorrhage were classified into three groups according to the site and the parenchymal involvement: group 1, infratentorial intracranial haemorrhage; group 2, infratentorial and supratentorial intracranial haemorrhage; and group 3, infra- and supratentorial haemorrhage with parenchymal involvement.

Subarachnoid and subdural haemorrhages were defined as presence of altered signal intensity, corresponding to blood products, respectively, into the sulci or exclusively in the subdural space appearing as a linear extracerebral alteration. Intraparenchymal haemorrhage was defined as presence of altered signal intensity corresponding to blood products in the cerebral parenchyma. We included only intracranial haemorrhage with acute, subacute (early and late) and early chronic signal intensity patterns (Table 2).

We collected obstetric data, including mode of delivery and complications during delivery, from maternal records. We collected meconium-stained amniotic fluid, gender, gestational age, birth weight, cord pH, Apgar score at 1 and 5 min, cooling treatment for perinatal asphyxia and neurological symptoms from the neonatal electronic records.

We compared the perinatal and neonatal characteristics of babies with and without intracranial haemorrhage.

Statistical analysis

Descriptive statistics are presented as mean ± SD for continuous variables and numbers and percentages for categorical variables. Categorical variables were compared between groups by using the Fisher exact test. Group differences in continuous variables were compared using the one-way Anova or Student's *t*-test. Statistical significance was set at $P = 0.05$.

Results

Intracranial haemorrhage was observed in 36 out of 240 (15%) infants (7/88, 8%, late preterm vs. 29/152, 19%, term infants). Median postnatal age at MR scan was 10 days (range 1–40). Clinical indication for MRI in babies with intracranial haemorrhage included: abnormal neurological signs ($n = 11$), perinatal asphyxia with hypoxic–ischaemic encephalopathy treated with cooling ($n = 8$), abnormal foetal or neonatal brain US findings ($n = 12$), metabolic disorders ($n = 1$), congenital infection ($n = 1$) and dysmorphic features ($n = 3$).

Babies with intracranial haemorrhage were slightly but significantly older and larger compared to babies without intracranial haemorrhage and were more likely to be born by vaginal delivery than caesarean section (Table 3). The difference in birth weight and gestational age persisted when considering only term babies.

Table 2 Intracranial haemorrhage signal intensity pattern on MRI

	Acute	Early subacute	Late subacute	Early chronic
T1	Isointense	Hyperintense	Hyperintense	Hyperintense
T2	Hyperintense	Hypointense	Hyperintense	Hyperintense low signal rim
FFE T2	Hypointense	Hypointense	Hypointense	Hypointense

FFE fast field echo

Table 3 Clinical characteristics of the study population

	Newborns with intracranial haemorrhage $n=36$ (%)	Newborns without intracranial haemorrhage $n=204$ (%)	P-value
Birth weight (g) [mean ± SD]	3,097±485	2,803±741	<0.01
GA (weeks) [mean ± SD]	38±2	37±2	<0.01
Late preterm infants (%)	7 (20%)	81 (40%)	0.02
[a]Apgar score at 1 min [mean ± SD]	6.5±3.15	7.5±2.3	0.02
[a]Apgar score at 5 min [mean ± SD]	8.7±1.8	8.9±1.6	0.56
[b]Mode of delivery			
- Spontaneous vaginal delivery	20 (56%)	50 (25%)	0.0005
- Vacuum extraction	4 (11%)	6 (3%)	<0.001
- Elective caesarean section	4 (11%)	67 (33%)	0.005
- Emergency caesarean section	8 (22%)	79 (39%)	0.06

[a] Apgar score at 1 and 5 min was not known in five babies without intracranial haemorrhage

[b] Mode of delivery was not known in two babies without intracranial haemorrhage

All infants with intracranial haemorrhage had subdural haemorrhage. The site of subdural haemorrhage was only infratentorial in 16/36 (44%) infants (group 1) and both infratentorial and supratentorial in 16/36 (44%) (group 2). No additional MRI findings were observed in group 1 but in group 2 a combination of subdural and subarachnoid haemorrhage was present in two infants, intraventricular haemorrhage in two infants ($n=1$ grade 1 and $n=1$ grade 3, according to Papile et al. [14]) and mild white matter abnormalities (punctate lesions) in six babies. All infants of groups 1 and 2, except one infant with intraventricular haemorrhage grade 3, were asymptomatic. The distribution of different intracranial haemorrhage patterns in late preterm and term infants is shown in Table 4.

Subdural haemorrhage in both sites (infratentorial+ supratentorial) with parenchymal involvement was present in four infants (group 3). The parenchymal involvement consisted of cerebellar haemorrhage in three out of four infants and frontal–parietal haemorrhage in one infant (Fig. 2). The presence of regular flow-void signal of dural sinus and deep cerebral veins was assessed in all MR sequences (T1- and T2-weighted [turbo] spin-echo, and fast field echo); no direct sign (i.e. clots) of sino-venous thrombosis or resultant abnormalities were detected.

Three out of the four infants with parenchymal haemorrhage (group 3) presented with acute neurological symptoms such as seizures or apnoeic-equivalent seizures on the first day after birth. These babies required neurosurgical intervention for haematoma evacuation in one case and drainage of acute hydrocephalus in the other two. One baby, a late preterm infant with cerebellar involvement, was asymptomatic. None of the babies with parenchymal involvement had either thrombocytopenia or impaired coagulation; the presence of prothrombotic mutations (factor V Leiden, Arg506Gln, and G20210A prothrombin) was assessed and excluded.

The clinical characteristics of the three groups are compared in Table 5. Subdural haemorrhage was seen in 8 out of the 23 infants with hypoxic–ischaemic encephalopathy treated with cooling (group 1, $n=4$; group 2, $n=4$). The rate of cooled babies was higher in the group of infants with intracranial haemorrhage compared to the group without (8/36, 22%, vs. 15/204, 7%; $P<0.05$), although none of the cooled babies developed MR findings consistent with hypoxic–ischaemic injury.

Uncomplicated spontaneous vaginal delivery was reported in 20 out of 36 neonates (56%); vacuum extraction was used in 4 neonates (11%) while 12 babies (33%) were delivered via caesarean section (elective caesarian in 4 cases; emergency caesarian due to failed vaginal delivery or foetal distress in 8 cases). Uncomplicated vaginal delivery was reported in 3/4 neonates in group 3 (1 infant who developed cerebellar haemorrhage was delivered by vacuum extraction).

Table 4 Distribution of different intracranial haemorrhage patterns in late preterm and term infants

	Late preterm infants $n=7$ (%)	Term infants $n=29$ (%)
Group 1 Infratentorial ICH	2 (28.6%)	14 (48.3%)
Group 2 Infratentorial + Supratentorial ICH	4 (57.1%)	12 (41.4%)
Group 3 Infratentorial + Supratentorial ICH + parenchymal involvement	1 (14.3%)	3 (10.3%)

ICH intracranial haemorrhage

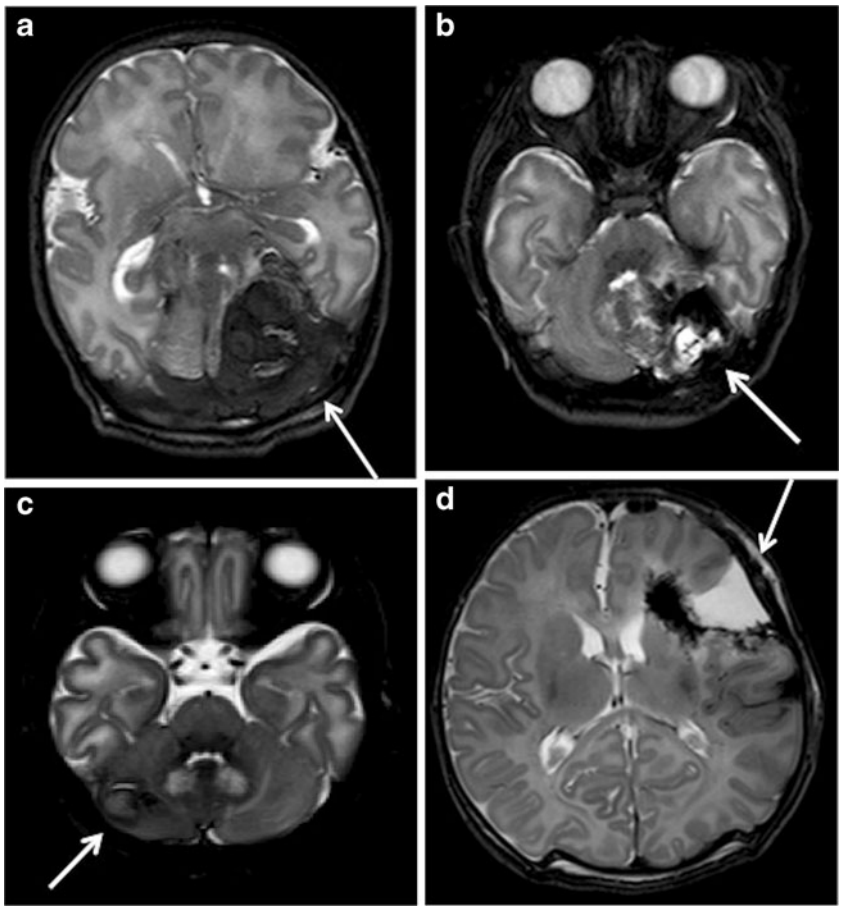

Fig. 2 AxialT2-weightedMR images of haemorrhages (*arrows*) with parenchymal involvement in four different infants. **a–c** Cerebellar haemorrhages. (Fig. 2a gestational age at birth 40 weeks, gestaional age at imaging 40 weeks + 1 day; Fig. 2b gestational age at birth 38 weeks + 5 days, gestaional age at imaging 39 weeks + 5 days; Fig. 2c 1 (gestational age at birth 35 weeks + 3 days, gestaional age at imaging 40) (**d**) Fronto-parietal haemorrhage that required surgical evacuation (gestational age at birth 41 weeks, gestaional age at imaging 43 weeks + 1 day)

Discussion

Our data support the evidence that mild intracranial haemorrhage is relatively common in full-term babies, although it mostly represents an incidental finding in clinically asymptomatic infants.

Differently from previous studies, we also included the population of late preterm infants whose brain is known to be more vulnerable, and theoretically more exposed, to intracranial haemorrhage than more mature infants. This hypothesis was not supported by our findings. One late preterm infant developed intracranial haemorrhage with parenchymal involvement, but unlike term babies he did not present any neurological symptoms. This finding is consistent with the observation that cerebral lesions occurring in preterm babies are often clinically subtle or silent and can be

Table 5 Clinical characteristics of infants with intracranial haemorrhage according to site of bleeding

	Group 1 Infratentorial ICH $n=16$	Group 2 Infratentorial + supratentorial ICH $n=16$	Group 3 Infratentorial + supratentorial ICH + parenchymal involvement $n=4$	*P*
Female/male	6/10	8/8	1/3	0.59
Birth weight (g)	3,169 ±420	2,995±579	3,213±280	0.49
Gestational age (weeks)	39±1.52	38±1.92	39±2.65	0.27
Head circumference [mean ± SD]	34.8±1.14	33.6±1.86	33.8±0.5	0.08
[a]Arterial cord pH	7.09±0.2	7.05±0.17	7.17±0.01	0.62
[b]Venous cord pH	7.23±0.15	7.22±0.14	7.20±0.18	0.88
Meconium-stained amniotic fluid	0	1	1	0.87

[a] Arterial cord blood pH was not performed in 9 newborns

[b] Venous cord blood pH was not performed in 4 newborns

misdiagnosed. However, this result needs to be confirmed by larger observations in late preterm infants.

Neuroimaging studies report a wide range of incidence of intracranial haemorrhage in term newborns. However caution is needed when interpreting those findings because of several confounding factors: study population, field strength of magnet and timing of assessment. Moreover, few prospective studies have been published. Because of its subtle or silent clinical presentation, intracranial haemorrhage is often undiagnosed and the real incidence is not known.

Rooks et al. [2] prospectively investigated a cohort of healthy term infants and reported an incidence of intracranial haemorrhage in 46%, while Looney et al. [1] included infants who underwent brain MRI as part of an ongoing prospective study that included both healthy newborns and babies at high risk for psychiatric or neurodevelopmental disorders (babies with prenatal diagnosis of foetal isolated mild ventriculomegaly or infants born to mothers with schizophrenia) and observed an incidence of 19%.

The wide difference in the reported incidence of intracranial haemorrhage is also related to the sensitivity of the MR field: the stronger the field, the higher the sensitivity (19% using a 3-T scanner versus 8% with a 0.2-T scanner) [1, 15]. Timing of MR scanning appears to be relevant because intracranial haemorrhage detection seems to be inversely related to postnatal age at MRI. The prospective study by Rooks et al. [2] suggests that most subdural haemorrhages present at birth are resolved by 1 month.

Therefore the highest incidence is reported by Rooks et al. [2] (46%), who scanned the babies very early (within 3 days of life) using a 1.5-T magnet, while the lower incidence quoted by Looney [1] (19%) may be related to a later scanning (1–5 weeks) even though a stronger MR field (3 T) was used. Although Whitby et al. [15] imaged babies within the first 48 h after birth, they observed a very low incidence of intracranial haemorrhage (8%), probably because of the low field strength of the 0.2-T magnet they used.

Our study's objective was not to estimate the incidence of intracranial haemorrhage, because of the retrospective selection of the study population. However, we studied a large population (240 newborns) and we used a strong magnet (1.5 T) but we scanned our babies at a median postnatal age of 10 days (range 1–40 days) and we probably missed some babies in whom intracranial haemorrhage had resolved. Late preterm newborns were scanned at term-corrected age, therefore intracranial haemorrhage might have been further underdiagnosed in these babies compared to the term infant cohort.

The observation about the timing of the appearance of intracranial haemorrhage supports the hypothesis that it is birth-related, being that birth itself is potentially and inherently traumatic to the foetal and neonatal brain. Local mechanical trauma, head squeezing, and overlapping sutures during labour and delivery leading to venous compression and rupturing of bridging veins and capillaries have been advocated as possible pathogenetic mechanisms involved in intracranial bleeding, even when an uneventful delivery occurs according to obstetric parameters [2, 10, 16, 17]. This finding was confirmed by our study, in which three out of four babies with parenchymal haemorrhagic involvement had an uneventful vaginal delivery. Altogether babies with intracranial haemorrhage were more likely to be born by vaginal delivery than caesarean section, although 12 infants (33%) with intracranial haemorrhage were delivered by caesarean section (4 of them elective). This finding is in agreement with data by Rooks et al. [2] and Brouwer et al. [10], who reported 9% and 9.4%, respectively, of infants with intracranial haemorrhages in their populations were delivered by caesarian; one infant in each study was delivered after elective caesarian section. The pathogenetic factors leading to intracranial haemorrhage after elective caesarian section have not been studied, and traumatic mechanisms during the extraction procedure can only be hypothesised and need to be investigated.

Infratentorial subdural haemorrhage is the most frequent form of intracranial haemorrhage in the neonatal period, as confirmed by our results. A recent theory based on neuropathological studies supports the dural origin of subdural haemorrhage [18]. During foetal life the posterior fossa is characterised by the presence of an extensive venous plexus in the dura which, in the neonate, forms sinuses in the tentorium, posterior falx and the dura of the floor of the posterior cranial fossa [19]. The dural anatomy is immature and vulnerable to the mechanical forces applied on the unfused infant skull during labour, predisposing it to subdural bleeding. The observation that neonatal subdural haemorrhage is particularly common over the dural folds bearing the venous sinuses further supports the key role of venous plexuses in subdural haemorrhage [18].

Several perinatal risk factors, such as high foetal weight, meconium-stained amniotic fluid and foetal distress, have been identified [1, 2, 6, 20]. In our study babies with intracranial haemorrhage were significantly larger than babies without but their birth weight was adequate for gestational age.

Primiparity maternal hypertension, placenta previa, abruptio placenta and autoimmune disorders have also been proposed as prenatal risk factors for intracranial haemorrhage in neonates, although the underlying pathogenetic mechanisms have not been fully investigated [11, 12, 21]; these clinical data were not analysed in the present study.

We found that babies with intracranial haemorrhage were born with a slightly lower Apgar score at 1 min but they recovered at 5 min. The rate of perinatal asphyxia causing hypoxic–ischaemic encephalopathy in the first hours of life, treated with whole-body cooling, was higher in infants with intracranial haemorrhage, although none of these babies showed MRI features consistent with hypoxic–ischaemic injury, suggesting a mild and reversible hypoxic–ischaemic

insult. Previous studies reported the association between hypoxia and subdural haemorrhage in neonates and children younger than 5 months of age [20, 21]. Traditionally perinatal asphyxia has been related to a traumatic birth and subsequent intracranial bleeding, but more recently alterations in the permeability of the cerebral vessels induced by hypoxia have been hypothesised as possible mechanisms of hypoxia-related intracranial haemorrhage, and the contribution of vascular leakage secondary to reperfusion injury has been emphasized [16]. Therefore subdural haemorrhage associated with hypoxia is likely to be related to a phenomenon of tissue immaturity rather than to a traumatic mechanism of rupture of bridging veins, as suggested by Geddes et al. [22] based on histological studies of the dura mater.

Early neurological symptoms only presented in babies with parenchymal involvement, supporting the theory that limited subdural haemorrhages mostly represent an incidental finding at MRI and do not affect neonatal clinical presentation or long-term neurological outcome. However some authors speculate that traumatic forces occurring during labour can cause, beyond intracranial bleeding, more subtle injury to the foetal/neonatal brain that is undetectable at conventional MRI but later involved in long-term neurocognitive impairment and idiopathic epilepsy, but this hypothesis has not been confirmed [1].

Our study has several limitations, mainly related to the retrospective selection of the study population and timing of MR scanning. Nevertheless the aim of this study was not to define the incidence of intracranial haemorrhage but to explore MR appearance and clinical presentation in the neonatal period in both term and late preterm babies, who were not studied before.

Conclusion

Mild intracranial haemorrhage, and in particular subdural haemorrhage, is confirmed to be relatively common in both late preterm and term infants, although it mostly represents an incidental finding in clinically asymptomatic babies. Early neurological symptoms appear to be related to parenchymal involvement. Further prospective and long-term studies are needed to explore the correlation between intracranial haemorrhage and perinatal risk factors and to investigate potential effects of intracranial haemorrhage on brain development.

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Conflicts of interest None.

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